VOLUME XXXIV JANUARY 1936 NUMBER 1

United States Naval Medical Bulletin

PUBLISHED *for the* INFORMATION OF MEDICAL DEPARTMENT *of the* NAVY

THE MISSION OF THE MEDICAL CORPS OF THE NAVY:

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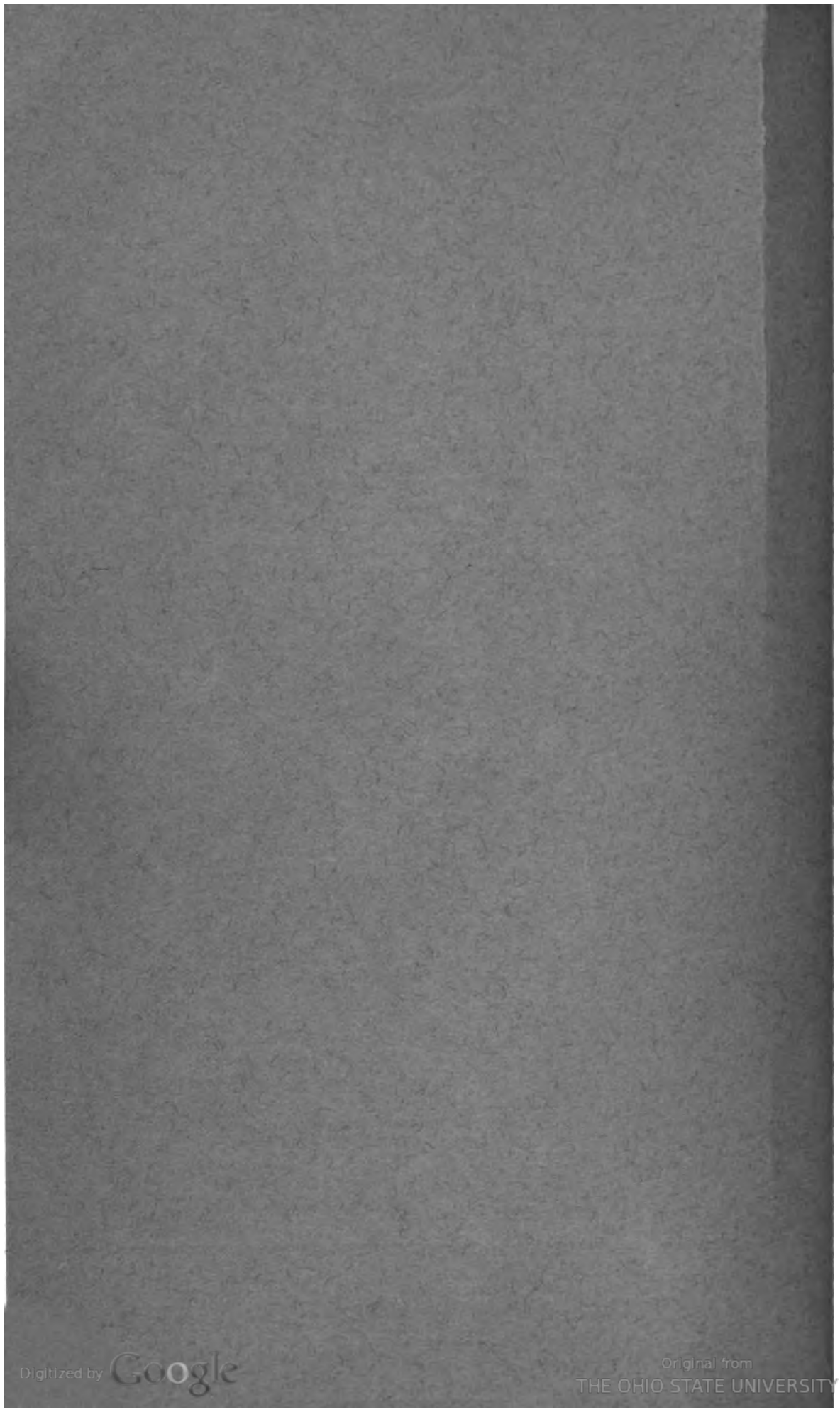
TO KEEP AS MANY MEN AT AS MANY GUNS AS MANY DAYS AS POSSIBLE

Issued Quarterly by the Bureau of Medicine and Surgery
Washington, D. C.



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UNITED STATES NAVAL MEDICAL BULLETIN

PUBLISHED QUARTERLY FOR THE INFORMATION OF THE MEDICAL DEPARTMENT OF THE NAVY

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NAVY DEPARTMENT,
Washington, March 20, 1907.

This UNITED STATES NAVAL MEDICAL BULLETIN is published by direction of the Department for the timely information of the Medical and Hospital Corps of the Navy.

TRUMAN H. NEWBERRY,
Acting Secretary.

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Volume X, no. 2, April 1916.
Volume XI, no. 3, July 1917.
Volume XII, no. 1, January 1918.
Volume XII, no. 3, July 1918.

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PREFACE

THE UNITED STATES NAVAL MEDICAL BULLETIN was first issued in April 1907 as a means for supplying medical officers of the United States Navy with information regarding the advances which are continually being made in the medical sciences, and as a medium for the publication of accounts of special researches, observations, or experiences of individual medical officers.

It is the aim of the Bureau of Medicine and Surgery to furnish in each issue special articles relating to naval medicine, descriptions of suggested devices, clinical notes on interesting cases, editorial comment on current medical literature of special professional interest to the naval medical officer, and reports from various sources, notes, and comments on topics of medical interest.

The Bureau extends an invitation to all medical and dental officers to prepare and forward, with a view to publication, contributions on subjects of interest to naval medical officers.

In order that each service contributor may receive due credit for his efforts in preparing matter for the BULLETIN of distinct originality and special merit, the Surgeon General of the Navy will send a letter of commendation to authors of papers of outstanding merit.

The Bureau does not necessarily undertake to endorse views or opinions which may be expressed in the pages of this publication.

P. S. ROSSITER,
Surgeon General, United States Navy.

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U. S. NAVAL MEDICAL BULLETIN

VOL. XXXIV JANUARY 1936 No. 1

SPECIAL ARTICLES

THE IDENTITY OF LYMPHOGRANULOMA INGUINALE AND CLIMATIC BUBO[1]

By L. E. GILJE, Lieutenant, Medical Corps, United States Navy

A specific disease which produces an indolent inguinal adenitis has been known to tropical residents for many years under the name of "climatic bubo."

In 1896 Ruge (1) reported an epidemic of this disease on board a German man-of-war off the coast of Zanzibar. Several medical officers (2) (3) (4) of the United States Navy reported observations on the disease as early as 1912. Durand, Nicolas, and Favre (5) reported a series of cases in France in 1913 and called the disease "lymphogranuloma inguinale."

The recent impetus of descriptions in the American literature was begun in 1924 by Hansmann (6) who described four cases which occurred in Boston under the term "nontuberculous granulomatous lymphadenitis." Following the introduction of a specific intradermal diagnostic procedure by Frei (7) in 1925, the American observers De Wolf and Van Cleve (8) collected a large series in the United States. Several other Americans (9) (10) (11) have since reported the disease in various sections of the United States.

The identity of climatic bubo and lymphogranuloma inguinale is now quite well established, although the disease is still known in the tropics as "climatic bubo" and in the United States as "lymphogranuloma inguinale."

In order to emphasize this identity, certain cases observed at the San Diego Naval Hospital are reported here together with some observations in connection with a new concept of treatment which should be of considerable practical interest to Navy medical officers because of the frequency with which they will encounter the disease among service personnel.

A brief discussion of the disease in its salient features is given here for the information of those whose contact with it may have been only casual.

[1] From the Department of Urology, U. S. Naval Hospital, San Diego, Calif. Read before the weekly staff conference at the U. S. Naval Hospital, San Diego, Calif., on Mar. 21, 1935.

Synonyms.—Climatic bubo; Durand, Nicolas, and Favre disease; tropical bubo; bubo, inguinal, nonvenereal; fourth venereal disease; nontuberculous lymphadenitis; strumous bubo; lymphopathia venereum are the most common.

Definition.—A disease caused by an unknown filtrable virus which may and usually does invade the genital organs primarily and produces a subsequent involvement of the regional lymphatics which are especially prone to suppurate and produce sinuses into the surrounding tissues.

Etiology.—A filtrable virus is considered to be the causative organism. The virus is killed by heating to 60° C. for 30 minutes. The organism is quite resistant to most antiseptics but is killed by 1:1000 formaldehyde, by drying, and also dies in glycerin. The disease can be transmitted to certain species of monkeys by intracerebral injection. The virus has been grown as described by Tamura (12) on a special media composed of Tyrodes solution and rabbits tissue (kidney, liver, or spleen) or guinea pig tissue under aerobic conditions at 37.5° C. Tamura was unable to stain the virus except for granules by the use of Giemsa and eosin-Giemsa methods.

Pathology.—The lymph node is chiefly involved in the center, but the whole node is involved to a less degree and there is also some periadenitis. Multiple small abscesses may be seen microscopically.

The histopathological picture of the primary lesion shows necrosis, granulation tissue, growth of the epithelium in normal manner with moderate keratinization and infiltration of the underlying corium and subcutanea with lymphoid, plasma, and endothelial cells in large numbers and with an occasional foreign body giant cell.

The microscopic section of the lymphnode shows areas of central necrosis with collections of lymphoid, endothelial and giant cells about the necrotic areas which closely resemble tubercles except that, as pointed out by Hansmann (6), there is no caseation.

The pathology of this condition does not give any characteristic which renders a specific diagnosis.

Incubation period.—The stage of invasion is variable but is usually from 8 to 20 days following sexual exposure.

Primary lesion.—This may or may not be present but from our observation it is much more frequently present than the literature would lead one to believe. The lesion may be evanescent and closely resemble a herpes or it may be papular or ulcerative. The most frequent site for the lesion is on the prepuce, but cases have been described in which an intraurethral lesion was present.

Regional lymphadenopathy.—This is the most uniform finding in the disease and may be the only tangible evidence at hand. In genital lesions the inguinal and iliac groups are involved in the

male, while in the female the deep pelvic nodes and at times the inguinal and iliac groups are invaded.

About 2 or 3 weeks following exposure the glands begin to enlarge and become firm and somewhat tender. The nodes may recede and then enlarge again before they fuse to the overlying skin. When the skin is involved there is a violaceous discoloration which was called the "adenite violette" by Phylactos. The glands tend to multiple rather than to solitary suppuration so that when they break through the skin multiple sinuses are formed from which thin yellow pus exudes.

General symptoms.—The signs of a general reaction are usually present with fever, malaise, general aches, and pains, headache, and at times splenomegaly, polyarthritis, and cutaneous eruptions may be present.

Late syndromes.—These chronic symptoms evolve about the chronic sinuses in the groins in the male. Elephantiasis may occur as the result of extensive lymphatic damage.

Rectal-anal syndromes.—In the female the deep pelvic nodes frequently ulcerate into the recto-anal canal producing sinuses and stricture as pointed out by Cole (13).

Diagnosis.—This may be inferred by the late clinical picture but early as well as late diagnosis may be made quite definitely by cutaneous response to the intradermal injection of an antigen prepared from the suppurating gland of a known case of lymphogranuloma as advocated by Frei in 1925.

The test is specific for lymphogranuloma and will not give a positive response in granuloma inguinale, chancroid, gonorrhea, or syphilis.

The antigen is prepared by aspirating under aseptic technique the purulent contents of a suppurating lymphogranulomatous gland. This material is diluted 1:5 or 1:10 with normal saline depending upon the viscosity of the aspirated material. The diluted antigen is then placed in a water bath for 2 hours at 60° C. The antigen is again placed in the water bath at 60° for 1 hour the following day. The antigen should be cultured for sterility and by guinea-pig inoculation for acid-fast organisms before it is used. After preparation the antigen is placed in rubber stoppered ampules and kept in a refrigerator. The antigen has been kept active by DeWolf and Van Cleve (8) for 10 months, but Hoffman (11) recommends testing on a known case every 3 months to insure its activity.

The Frei test.—An area on the flexor surface of the forearm is selected for the test and is thoroughly prepared by soap and water followed by alcohol which is allowed to dry. With a tuberculin syringe and a 23-gage needle one-tenth of a cubic centimeter of the

antigen is injected intradermally. The intradermal injection should be so shallow that the lumen of the needle can be seen through the overlying skin. A control of one-tenth cubic centimeter of normal saline is injected about an inch from the antigen injection in the same manner.

At 24 hours a positive test will show a small papule at the site of the antigen injection about 2 to 5 millimeters in diameter. About 48 to 72 hours after injection the reaction reaches its height and will show a papule 5 to 10 millimeters in diameter. A negative reaction at 48 to 72 hours will only show the needle hole with perhaps a small area of erythema. A positive may be seen as a dull red or bluish discoloration for as long as 7 to 10 days. A markedly positive will produce a small area of necrosis in the center of the papule.

False positives rarely occur unless the antigen has been contaminated by other organisms. One negative reaction does not necessarily rule out the presence of the disease, and several tests may be required before a positive results.

The reaction usually becomes positive in from 10 to 20 days after the onset of the adenitis and probably remains positive for life. Hellerstrom (10) has reported a case in which the test was positive 23 years after the disease was contracted.

Syphilis may occur concurrently with lymphogranuloma and some observers have expressed the opinion that a positive Frei test may not occur until antisyphilitic treatment has been instituted, but we have seen cases in which this was not true.

During the stage of general reaction with fever the Kahn has been reported at times to be positive without the presence of syphilis.

Differential diagnosis.—Granuloma inguinale: This condition produces an ulcerating lesion of the pudenda together with at times an inguinal adenitis which does not heal after incision but does not produce a positive Frei test. There is no uniformity in the isolation of the causative organism, so one must rely on the clinical signs and a negative Frei reaction to differentiate this disease from lymphogranuloma inguinale.

Chancroid: This produces a more acute bubo which suppurates, forming one cavity. A culture and a Frei test may be, and usually is, necessary to differentiate this disease from lymphogranuloma.

Syphilis is usually quite easy to rule out by dark field and serological studies.

Bubonic plague: The causative organism can be identified by morphology and culture of the aspirated contents of a questionable gland.

Lymphoblastoma: Biopsy and histo-pathological examination may be necessary together with hematologic studies to rule out this

group of conditions if the Frei is not positive in a suspected case of lymphogranuloma.

Inflammatory lymphadenitis: There is usually an area of infection in the area drained by the involved nodes, but a Frei test may be necessary to establish the identity of the lesion because extragenital infection with lymphogranuloma has been reported.

Tuberculosis: Biopsy studies and guinea-pig inoculation should establish this condition.

Neoplasms: Tumors, of course, can usually be differentiated by the clinical picture, but at times only biopsy will prove or disprove the diagnosis.

Gonorrhea, tularemia, glanders, actinomycosis, blastomycosis, and coccidiodes infections may be ruled out by proper laboratory procedures if there is any question about the clinical diagnosis.

A study of the Frei reaction.—Two antigens were obtained from two different sources: Antigen A was prepared from a case of climatic bubo and antigen B was obtained from a case of lymphogranuloma inguinale.

A control series was made for each antigen as recorded in tables 1 and 2. The patients selected for the control series were suffering from gonorrhea, syphilis, or granuloma inguinale, and one control had no venereal infection.

As shown in the first two tables, negative results were obtained in every case in the control series.

TABLE 1.—*Antigen A*

Case	Diagnosis	Time in hours after injection					
		24		48		72	
		A	Con.	A	Con.	A	Con.
A	Epididymitis, acute (nonvenereal)	−	−	−	−	−	−
B	Gonococcus infection, urethra	−	−	−	−	−	−
C	Gonococcus infection, urethra	−	−	−	−	−	−
D	Syphilis, primary	−	−	−	−	−	−
E	Chancroid, penis	−	+	−	−	−	−
F	Granuloma inguinale	+	−	−	−	−	−

TABLE 2.—*Antigen B*

Case	Diagnosis	Time in hours after injectoni					
		24		48		72	
		B	Con.	B	Con.	B	Con.
B	Gonococcus infection, urethra	±	−	±	−	+	−
D	Syphilis, primary	±	−	+	−	+	−
E	Chancroid, penis	−	−	−	−	−	−
F	Granuloma inguinale	±	−	−	−	−	−
G	Chancroid, penis	±	−	−	−	−	−

TABLE 3.—*Antigens A and B, and saline control*

		Time in hours after injection								
		24			48			72		
		A	B	Con.	A	B	Con.	A	B	Con.
I	Climatic bubo	+	+	−	++	++	−	+++	+++	−
II	Lymphogranuloma	+	+	−	++	++	−	+++	+++	−
III	Climatic bubo	+	+	−	+++	+++	−	+++	+++	−
IV	Lymphogranuloma	+	+	−	++	++	−	+++	+++	−
V	Lymphogranuloma	++	++	−	+++	+++	−	+++	+++	−
VI	Climatic bubo	++	++	−	+++	+++	−	+++	+++	

Legend:
A = Antigen A.
B = Antigen B.
Con. = Saline control.
− = Negative.
± = Slight area oferythema.
+ = Small area of erythema and a minute papule.
++ = Erythema with a papule 2 to 4 millimeters in diameter.
+++ = Erythema with a papule 5 to 10 millimeters in diameter.
++++ = Same as +++ except that an area of necrosis is present in the center of the papule.

Interpretation: −, ±, and + were considered to be negative results; ++, +++, and ++++ were considered to be positive results.

Discussion of the antigen study.—The control series for both antigen A and B which were performed on cases of gonorrhea, chancroid, granuloma inguinale, and syphilis, gave entirely negative reactions.

The climatic bubo and lymphogranuloma cases all gave positive reactions to both antigens and in approximately the same degree of reaction.

A control series of this type is valuable in that it illustrates the specific nature of the intradermal response when considering the venereal diseases.

A double antigen series of a group of cases with these diseases as shown in table III illustrates that the climatic bubo of the tropics and the lymphogranuloma of the United States give the same response to both antigens.

It has been shown by Tamura (12) that a positive Frei reaction will occur in an individual with lymphogranuloma when the subcultures of the virus are used as an antigen. The reaction is also positive when the virus is used after passage through a Berkfeld filter. This would indicate that the antigen of the Frei test is the virus or its toxins.

The above two statements are considered to be specific evidence that climatic bubo and lymphogranuloma are caused by the same virus.

CASE REPORTS

Case no. 1.—W. J. C., Sea1c, February 1, 1935.

Admission diagnosis: Chancroidal Lymphadenitis.

C. C.: Ulcer in coronal sulcus; bubo, right groin.

Past history: G. C. infection, urethra, 1931 and 1932. Lymphadenitis inguinal, left, 2 years earlier, secondary to wound, left foot. Denies syphilis.

Present illness: Exposure in Panama, October 30, 1934. On January 8, 1935, he noticed two small ulcers in the coronal sulcus. Dark fields were then negative for treponema pallida. Kahn, January 28, 1935, was negative. On January 20 he had malaise, fever, and general aches. On January 22 he developed a right inguinal adenitis about the size of a hen's egg. On February 1 the penile ulcers were unimproved and the bubo had softened and was opened and packed. Transferred to hospital the following day.

Physical examination: Showed a recent incision of the area overlying the right inguinal glands. There was a yellowish pus exuding from the wound. The edges were discolored a bluish tinge and were undermined. There was a scar over the left inguinal glands at site of a previous lymphadenitis in 1933. No other glands involved. No urethral discharge. Scrotal contents were normal. There were two ulcers in the coronal sulcus, one on the frenum and one the right side of the frenum which were small and punched out in appearance.

Progress notes: Three dark fields done in the first week after admission were all negative. On February 9 the Frei test was positive and on Febraury 10, 2 plus, and on February 11, 3 plus. Weekly Kahns throughout hospitalization were negative. Tartar emetic solution given intravenously in increasing doses on alternate days for 2 weeks, at end of which time the ulcers and inguinal wound were healing nicely; but were incompletely healed by March 1. Frei antigen 0.2 cc given intradermally on March 2 and 5. March 8 all lesions had healed completely.

Case no. 2.—F. H. P., Sea1c, January 18, 1935.

Admission diagnosis: G. C. Infection urethra.

C. C.:(1) Purulent urethral discharge. (2) Ulcer in coronal sulcus.

Past history: Gonorrhea 3 years ago, chancroid 3 years ago with no follow-up Kahns.

Present illness: Exposure on January 11, 1935, in San Diego, Calif. On January 17, he developed a purulent urethral discharge and a day or two later he noted an ulcer about the size of a small pea in the coronal sulcus. The left inguinal glands began to swell about January 18 and were not very tender.

Physical examination: Showed slight general adenopathy. The inguinals very large, slightly tender; but not necrotic. There was a purulent, urethral discharge, and a small punched out ulcer in the coronal sulcus.

Progress notes: The urethral smear did not reveal G. C. The Kahn was 4 plus on the day of admission. As the primary sore was too young to account for the 4-plus Kahn, the incubation period rather too short for syphilis and the dark fields negative, it was concluded that the syphilitic infection was acquired 3 years earlier at the time of the chancroid, and that the present ulcer was nonsyphilitic. Antisyphilitic treatment was begun on the day of admission. Three days later the left inguinal glands became swollen and tender and by the twelfth day showed suppuration. They were incised with the drainage of thick yellow pus. By the seventh week the inguinal wound was still unhealed. A Frei test was then done with result of 3 plus. Frei antigen was given intradermally on alternate days with complete healing of the penile ulcer and inguinal wound by the ninth week. The failure of the ulcer to heal by antisyphilitic treatment was further proof that it was nonsyphilitic and that the syphilitic infection had occurred 3 years earlier.

Case no. 3.—E. L. B., Pfc, U. S. M. C., February 8, 1935.

Admission diagnosis: Chancroidal lymphadenitis, right.

C. C.: Swollen glands in the right groin.

Past history: Chancroid 2 years earlier; no bubo; healed rapidly. Gonorrhea 1 year earlier.

Present illness: Exposure about December 17, 1934, in Shanghai, China. On January 2, 1935, he developed a small ulcer on the ventral surface of prepuce. Dark fields were taken daily with negative results. On January 17, the right inguinal glands began to swell, but were not tender and did not suppurate. The penile lesion healed by January 28. There had been no clinical or laboratory evidence of syphilis. During the middle of January the patient had malaise, felt flush, and under par.

Physical examination: Showed the right inguinal glands to be enlarged about the size of a lemon. They were firm, not fluctuant, and not tender. The glands were matted together but not adherant to the skin or deep fascia. There were two small scars on the penis, one on the prepuce at the site of the chancroid of two years earlier, and one on the other more recent site on the frenum.

Progress notes:

February 8, 1935. Kahn negative.

February 12, 1935. Frei test done. Kahn negative.

February 13, 1935. Frei test positive, 1 plus.

February 14, 1935. Frei test positive, 3 plus.

February 15, 1935. Frei test positive. Kahn negative.

February 18, 1935. Bubo subsiding and showed no sign of suppuration. Treatment has been bed rest and sedatives. No other medication has been used. Frei antigen has possibly exerted a favorable influence in this case to date.

February 21, 1935. Kahn negative, glands subsiding rapidly. No signs of suppuration.

March 1, 1935. To duty, bubo almost completely subsided. Weekly Kahns were negative throughout hospitalization. Frei test done on fifth day after admission was 1 plus; on the sixth day, 3 plus. The glands had entirely subsided within 3 weeks after admission and patient returned to duty. He returned 2 weeks later for another Frei test which gave a 3-plus result. Has remained entirely well with no return of symptoms.

Case no. 4.—A. E., Seaic, March 2, 1935.

Admission diagnosis: Bubo, inguinal, nonvenereal.

C. C.: Bilateral inguinal buboes.

Past history: Gonorrhea 4 years ago, 3 years, and 2 years ago. No complications. Syphilis contracted 18 months ago. All Kahns have been strongly positive. Received 16 injections of arsphenamine and 46 of bismuth during the first year of infection and 3 of neoarsphenamine to date during second year.

Present illness: Exposure in San Francisco 8 weeks ago, and in San Diego 4 weeks ago. About 3 weeks ago he noted a swelling in each inguinal region. These gradually became larger. During the past week he has had a fever ranging from 99 to 103. Redness and fluctuation appeared in the glands the last few days. More marked on the right side. Patient denies any genital lesion during the past year.

Physical examination: Genitalia, negative. Glands: the iliac and inguinal glands are indurated and markedly enlarged; the overlying skin is a purplish hue. There are large areas of fluctuation about the size of a hen's egg in the right iliac glands on the right and about the size of a walnut in the left iliac glands. Not very tender.

March 3, 1935. Right gland aspirated, thick, yellowish-green pus. Three puctures, unable to drain completely because it is multilocular. Frei test. Icebag and pressure bandage.

March 4, 1935. Frei, 2 plus. Icebag and pressure bandage.

March 5, 1935. Frei 3 plus. The left inguinal glands have softened.

March 6, 1935. Frei 2 plus. Both glands incised.

March 7, 1935. 0.2 cc antigen intradermally.

March 9, 1935. 0.25 cc antigen intradermally. There is no further suppuration of the glands. The incisions are clean and granulations appear quite healthy. The drainage is scant, thin, yellowish pus. The other glands apparently are not going to break down. Patient's general symptoms are now absent.

March 11, 1935. 0.30 cc antigen subcutaneously.

March 13, 1935. 0.35 cc antigen subcutaneously. Very slight serous discharge; convalescence is rapid. Daily irrigation with 1 percent chlorazene.

March 15, 1935. 0.40 cc antigen subcutaneously.

March 17, 1935. 0.45 cc antigen subcutaneously.

March 20, 1935. 0.50 cc antigen subcutaneously. Patient's incisions granulating nicely.

April 12, 1935. Healed; to duty.

Case no. 5.—W. A. S., Matt3c, March 4, 1935.

Admission diagnosis: Bubo, inguinal, nonvenereal.

C. C.: Bubo, left inguinal.

Past history: No illness of importance before entering the Navy 7 months ago. No history of gonorrhea or genital sore.

Present illness: Exposure in Little Rock, Ark., on November 9, 1934. About December 22, 1934, he noticed bilateral inguinal adenopathy. He had some headache and general malaise. Five days later the glands became as large as a walnut and then became smaller. He was exposed again in Panama, December 21, 1934. About March 1, 1935, the left inguinal glands suppurated and drained spontaneously. Admitted 3 days later.

Physical examination: Showed a well developed and well nourished colored male about 18 years of age. Shotty posterior cervical glands. Genitalia—negative. Extremities—shotty epitrochlear adenopathy. Skin—clear. The left inguinal glands are about the size of a small egg and are firmly attached to the overlying skin. There is some softening and over the glands there is a small draining sinus from which a small amount of thin pus exudes. The right inguinal glands are about the size of a hickory nut and are not fluctuant or attached to the skin.

Progress notes:

March 5, 1935. Kahn, negative.

March 5, 1935. Frei 2 plus.

March 6, 1935. Frei 3 plus.

March 8, 1935. Frei antigen 0.20 cc intradermally.

March 10, 1935. Frei antigen 0.25 cc subcutaneously.

March 12, 1935. Frei antigen 0.30 cc subcutaneously. The bubo has ceased draining and is much smaller in size. No further evidence of suppuration of the glands.

March 15, 1935. To duty, lesions healed.

Case no. 6.—C. L. A., SC2c, March 19, 1935.

Admission diagnosis: Diagnosis undetermined (bubo, inguinal, left nonvenereal.)

C. C.: Left inguinal bubo.

Past history: No illness of importance before entering the Navy 6 years ago. Had a chancroid in May 1934: the Kahn was negative in August 1934. No Kahns have been made since August 1934. No history of gonorrhea. Has been quite well during past year until onset of the present illness.

Present illness: Exposure in Guadaloupe, West Indies about February 1, 1935. About March 1, 1935 he noticed some swelling of the left inguinal glands. The bubo has since gradually increased in size. During the past 10 days there has been some afternoon temperature and moderate malaise. There is no history of a penile lesion since May 1934.

Physical examination: Patient appears to be ill and anemic. The left inguinal glands are firm, not tender, and about as large as a lemon. No areas of fluctuation. No other lymphnodes are involved. Genitalia—there is a small pale scar on the prepuce at the site of a lesion in 1934. No lesions are present.

Progress notes:

March 19, 1935. Frei test; 0.2 cc antigen.

March 20, 1935. Frei test 1 plus.

March 21, 1935. Frei test 2 plus.

March 22, 1935. Frei test 3 plus. Kahn 4 plus.

March 23, 1935. Frei antigen 0.25 cc intradermally.

March 25, 1935. Frei antigen 0.3 cc subcutaneously.

March 26, 1935. Kahn 2 plus.

March 28, 1935. Frei antigen 0.4 cc subcutaneously.

March 29, 1935. Kahn 1 plus.

March 30, 1935. Temperature normal for 2 days. Bubo subsiding. Out of bed. General systemic symptoms have disappeared.

April 2, 1935. Frei antigen 0.5 cc subcutaneously. Kahn negative.

April 10, 1935. Bubo well subsided.

April 12, 1935. Well; duty. Note the rise and fall of the Kahn, without antisyphilitic treatment, conciding with the rise and fall of the febrile stage of the lymphogranuloma inguinale.

Treatment.—The following measures have been used in the treatment of this disease: Antiseptics, radical excision of the lymphnodes, antimony compounds, incision and drainage, closed aspiration alone and in conjunction with instillation of solutions, roentgen therapy and injections of the antigen intradermally (13) and subcutaneously (14). An antiserum prepared from a goat has been used by Tamura (12).

The treatment we have used on this series of cases consisted in:

(1) Encouraging resolution rather than suppuration in the lymph glands by conservative measures.

(2) Tartar emetic 10 cc of 1 percent solution intravenously every 48 hours if tolerated by the patient.

(3) Multiple incisions of the glands only when they suppurate and daily irrigations of the incised glands with 1 percent chlorazene solution.

(4) The Frei antigen intradermally every other day beginning with 0.10 cc and increased 0.05 cc a dose until 0.25 cc is reached and then the antigen is given subcutaneously in doses beginning with 0.30 cc and increased 0.05 cc every other day until 0.50 cc is reached.

(5) In some cases we have used mercurochrome crystals alternated each day with argyrol crystals on the primary lesions.

SUMMARY

A discussion of lymphogranuloma inguinale is presented.

The Frei reaction has been used in two control series. A double antigen series using two antigens is presented for consideration as evidence that the climatic bubo of the tropics and the lymphogranuloma inguinale of the United States are produced by the same virus.

Six cases of the disease are recorded, 3 of which originated outside the United States and 3 of which were contracted within the United States.

Observations on the therapeutic effects of the Frei antigen are reported.

BIBLIOGRAPHY

(1) Hoffman, Eugene (quoted from): Lymphopathia Venereum. The Urologic and Cutaneous Review, vol. XXXVII, 2011.
(2) Phillips, E. W.: Two Cases of Climatic Bubo. U. S. Naval Medical Bulletin, 6: 402–404, 1912.
(3) Heiner, R. G.: Eighteen Cases Resembling Climatic Bubo. U. S. Naval Medical Bulletin, 7: 126–1913.
(4) Whitmore, W. H.: Climatic Bubo. U. S. Naval Medical Bulletin, 25–89–102, January 1927.
(5) Durand, M., Nicolas, J., and Favre, M.: Bull. et Mem. Soc. d. hop de Paris, 35: 274–288, February 6, 1913.
(6) Hansmann, G. H.: Nontuberculous Granulomatous Lymphadenitis, Surgery Gynecology and Obstetrics 39: 72–82, July 1924.
(7) Frei, Wilhelm: Kline, Wchnschr, 4: 2148, November 5, 1925.
(8) De Wolf, H. F., and Van Cleve, J. V.: Lymphogranuloma Inguinale, Journal American Medical Association, 99: 1065.
(9) Tomlinson, C. C., and Cameron, O. J.: Lymphogranuloma Inguinale, Archives Dermatology and Syphilology, May 27, 1933.
(10) Burney, Leroy E.: Lymphogranuloma Inguinale—Venereal Diseases Information, vol. XV, no. 7.
(11) Hoffman, Eugene F.: Lymphopathia Venereum—The Urologic and Cutaneous Review, vol. XXXVII, no. 11.
(12) Tamura, Joseph T.: The Virus of Lymphogranuloma Inguinale—Journal of Lab. and Clin. Med. vol. 20, no. 4, pp. 393–401.
(13) Wein, Max, and Perlstein, Minnie O.: Intradermal Treatment of Lymphogranuloma Inguinale, Arch. Dermat. and Syph., 28: 42, 1933.
(14) Hermans, E.: Klimatische Bubonen und Lymphogranuloma Inguinale, Kline, Wchnschr, 7: 2436, 1928.

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THE FREI TEST IN LYMPHOGRANULOMA INGUINALE AND OTHER TYPES OF INGUINAL ADENITIS

By C. B. Galloway, Lieutenant, junior grade, Medical Corps, United States Navy

The announcement in 1925 by Wilhelm Frei (1) of a specific intradermal test for lymphogranuloma inguinale marked a new era in the diagnosis and clinical appreciation of the importance of this disease. Prior to 1925 the etiology of lymphogranuloma inguinale was unknown, the clinical picture illdefined, and the relationship of the virus to esthiomène and rectal stricture unsuspected.

Today lymphogranuloma inguinale is a clear cut clinical entity (2), (3). A specific virus has been isolated which fulfills the postulates of Koch (4), the mode of infection has been established (5), the climatic relationship defined (6), a rational form of therapy established and a characteristic histopathological picture demonstrated (7), (8).

For the past 3 years the author has carried out the Frei test in three groups of patients. 1. Active subjects with a health record history of bubo inguinal nonveneral. 2. Those with a health record history of syphilis, chancroid, chancroidal lymphadenitis, gonorrhea or nonspecific inguinal adenitis. 3. Patients with active clinical cases of lymphogranuloma inguinale. This has been done in an attempt to determine; first, the specificity of the reaction in uncomplicated lymphogranuloma inguinale; second, the duration of the reaction following the acute phase and; third, its diagnostic value in the presence of other diseases causing an inguinal adenitis.

Preparation of the Frei antigen (9).—Pus from a fluctuant intact lymphogranuloma inguinale bubo was aspirated with an ordinary 18-gage needle; 1 part of the aspirated material was diluted with 9 parts of normal saline and heated at 60° C. for 2 hours the first day, and for 1 hour on the second day. When proven sterile by aerobic and anaerobic cultures the antigen is ready for use.

Technique of the Frei test (9).—One-tenth of a cubic centimeter of Frei antigen is injected intradermally into the volar surface of the left forearm. This injection mechanically produces a small colorless papule some 3 millimeters in diameter which disappears in a few hours, leaving no gross evidence of the antigen's presence.

In patients suffering with lymphogranuloma inguinale an inflammatory papule appears in 24 hours and reaches the height of its reaction in 72 hours. The size of the inflammatory papule depends upon the extent and activity of the disease, and the strength of the antigen used. The average case with a single bubo will show a dull red papule from 5 to 10 millimeters in diameter on the third day. Cases with bilateral bubo formation frequently show a pustular reaction from 1 to 1.5 centimeters in diameter. An area of dis-

coloration remains in the positive cases from 1 to 3 weeks. The pustular reactions may leave a small permanent scar.

Table 1 gives the results of the Frei test in 15 healthy individuals with a health record diagnosis of bubo inguinal nonvenereal.

TABLE 1.—*Results of Frei test on patients with a health-record history of bubo inguinal nonvenereal*

Case	Health-record diagnosis	Date of health-record diagnosis	Date of Frei test	Result of Frei test
1, Y. J., U. S. M. C.	Bubo inguinal nonvenereal.	July 1932	November 23, 1933	Positive.
2, S. S., U. S. M. C.	do	March 1932	do	Do.
3, B. O., U. S. N.	do	June 1932	do	Do.
4, C. H., U. S. N.	do	November 1933	do	Do.
5, G. A., U. S. N.	do	October 1933	do	Do.
6, G. R., U. S. N.	do	July 1926	do	Do.
7, H. O., U. S. N.	do	August 1933	do	Do.
8, M. I., U. S. N.	do	July 1930	do	Do.
9, P. O., U. S. N.	do	July 1932	do	Do.
10, E. C., U. S. M. C.	do	May 1927	November 27, 1934	Do.
11, O. G., U. S. M. C.	do	August 1929	do	Do.
12, A. R., U. S. M. C.	do	June 1932	December 1933	Do.
13, E. D. B., U. S. N.	do	June 1931	June 1933	Do.
14, R. K., U. S. N.	do	1923	do	Do.
15, L. T., U. S. M. C.	do	August 1933	December 1933	Do.

It will be seen from table 1 that each case of bubo inguinal nonvenereal reacted in a positive manner to the Frei test, thus identifying the disease as lymphogranuloma inguinale. Evidence of a permanent cutaneous sensitivity to the Frei test is shown by case 14, in which the original bubo existed in 1923 (10 years previous), also case no. 6, with the original bubo in 1926 (7 years previous). These results are in accord with those of Hellerstrom (10) who reports a positive Frei reaction in a surgeon 23 years after accidental infection while operating upon a case of inguinal adenitis (clinically lymphogranuloma inguilale).

Case no. 15 acquired syphilis between August 1933 (date of acute lymphogranuloma inguinale) and October 25, 1933; on the latter date his Kahn reaction was 4+ and the usual signs and symptoms of secondary syphilis were present. Although the patient was under antileutic treatment for an active syphilis his Frei test was definitely positive on December 14, 1933. This is contrary to the observations of Gregorio (11) that in mixed infections the Frei reaction gives way to an anergy brought about by syphilis, and returns only after antileutic treatment has been given. The severe reaction to the Frei antigen 48 days after the start of antileutic treatment indicates the theoretical existence of a positive reaction throughout the course of the syphilitic infection.

Table 2 shows the results of the Frei test in seven patients, each with a health record history of multiple venereal diseases, any one of which may produce an inguinal adenitis clinically difficult to differentiate from lymphogranuloma inguinale.

TABLE 2.—*Control cases with a history of venereal diseases or inflammatory inguinal adenitis*

Case	Venereal history	Date of Frei test	Result
1, H. L. P., S. 1/C, U. S. N.	Gonorrheal ureteritis, 1918, 1920, 1921; syphilis, 1922; chancroid, 1924; gonorrhea, 1924; chancroid, 1925; gonorrhea, 1925.	June 1933	Negative.
2, B. F. S., F. 2/C, U. S. N.	Gonorrhea, March 1931 and August 1931; chancroid penis, Jan. 1933, followed by suppurating inguinal adenitis resistant to treatment (32 sick days).	do	Do.
3, A. E. N., F. 2/C, U. S. N.	Severe epidermophytic infection of right toe followed by rightinguinal adenitis with subsequent suppuration and healing in 60 days.	do	Do.
4, W. R. M., Pvt., U. S. N.	Chancroid penis, 1932, followed by chancroid lymphadenitis.	December 14, 1933	Do.
5, F. R., Pvt., U. S. M. C.	History of syphilis, chancroid, and chancroid adenitis.	November 22, 1933	Do.
6, J. A. H., Corp., U. S. M. C.	Chancroid followed by chancroid lymphadenitis, 1930.	July 1933	Do.
7, K. V. F., Pvt., U. S. M. C.	Health record history, chancroid penis, Mar. 30, 1930, to Apr. 29, 1930; bubo inguinal nonvenereal, May 26, 1930, to July 21, 1930.	December 10, 1933	Obviously clinical error in diagnosis. Negative.

Case no. 7 represents an obvious error in the diagnosis of bubo inguinal, nonvirulent, both from the standpoint of the Frei test and the clinical history.

A résumé of six case histories of typical lymphogranuloma inguinale, in which the Frei test was uniformly positive, follows:

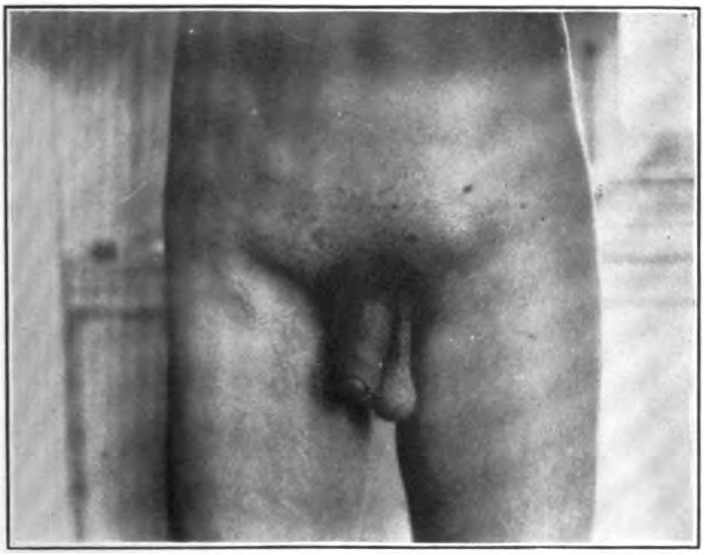
The Frei test in active cases of lymphogranuloma inguinale

Case 1.—G. H. L., Sgt. U. S. M. C.: History of unprotected coitus on May 28 with a Filipino prostitute. On June 8 a single painful node was noticed in the left groin, which enlarged rapidly, and on June 13 measured 3 centimeters in its greatest diameter. On June 20 the patient was admitted to the hospital with a smooth nonfluctuant ovoid bubo measuring 6 centimeters in its greatest diameter and lying parallel to Poupart's ligament. There was no pain when at rest and only moderate tenderness to pressure. The tumor was fixed in the subcutaneous tissues but not to the skin. There was no discoloration of the overlying skin and no objective evidence of a local inflammatory process. Frei test positive on July 1.

Case 2.—B. K. C., Pvt. U. S. M. C.: History of unprotected coitus on several occasions with a Japanese prostitute. On February 20 a single palpable, nonpainful nodule was noticed in the right groin. This node remained quiescent until March 5 when, following prolonged exercise, it became painful and rapidly increased in size until on March 20 the patient presented a firm, moderately painful tumor 5 centimeters in its greatest diameter and lying just above but parallel to Poupart's ligament. Frei test positive on March 20.

Case 3.—F. P. E., Pfc. U. S. M. C.: History of frequent unprotected coitus with various Chinese prostitutes. Admitted to the hospital on December 6 with a left inguinal tumor measuring 5 centimeters in its greatest diameter and lying parallel to and over Poupart's ligament. The tumor was firm but rather tender to pressure. Frei test positive December 20.

TWO POSITIVE FREI TESTS IN ACTIVE CASES OF LYMPHOGRANULOMA INGUINALE. (72 HOURS.)



LYMPHOGRANULOMA INGUINALE. ACUTE PHASE WITH NONFLUCTUANT BILATERAL BUBO. FREI TEST POSITIVE.

Case 4.—E. J. E., Pvt. U. S. M. C.: History of unprotected coitus with Chinese, Japanese, and Russian prostitutes. On October 10 a single small painless node was noticed in the left inguinal region. This increased rapidly in size, and on October 31 measured 4.5 centimeters in its greatest diameter. This tumor was firm, fixed to the subcutaneous tissue, nonfluctuant, and definitely painful to pressure. The overlying skin was not adherent. Frei test positive on November 22.

Case 5.—M. H. A., Sgt. U. S. M. C.: History of unprotected coitus with Chinese and Filipino prostitutes. On June 8 the patient reported with a left inguinal tumor 3 centimeters in diameter; duration 2 weeks. On June 20 the tumor measured 6.5 centimeters in its greatest diameter, was painful to pressure and presented at its center an area of fluctuation 1.5 centimeters in diameter. Tumor incised June 27 and 20 cubic centimeters of pus evacuated. Completely healed on July 3. Frei test positive on November 11.

Case 6.—K. A., Phm. 2/c, U. S. N.: History of unprotected coitus with Chinese and Japanese prostitutes. The patient was first seen on June 1 and at that time presented a firm, smooth, painless tumor 4.5 centimeters lying superior and parallel to Poupart's ligament on the right. Duration approximately 14 days. Frei test positive on June 5.

Table 3 lists eight control cases with no history of venereal disease or inguinal adenitis.

Frei tests were done in individuals free of lymphogranuloma inguinale as a control for each positive reaction.

TABLE 3

Case	Venereal history	Date of Frei test	Result of Frei test
1. F. J. J., Lt. U. S. N.	None	June 1933	Negative.
2. W. L. B., Lt. (M. C.) U. S. N.	do	do	Do.
3. R. M. S., S. 2/C., U. S. N.	do	do	Do.
4. B. C. B., F. 1/C, U. S. N.	do	do	Do.
5. C. B. A., Lt. (M. C.) U. S. N.	do	Nov. 22, 1933	Do.
6. D. H. D., Lt. (M. C.) U. S. N.	do	do	Do.
7. G. E., Phm. 2/C, U. S. N.	do	do	Do.
8. W. F., Phm. 2/C, U. S. N.	do	do	Do.

SUMMARY

1. The Frei test becomes positive during the acute phase of lymphogranuloma inguinale and remains positive for many years.

2. All of the cases in this series carried under a health record diagnosis of bubo inguinal nonvenereal were found to be lymphogranuloma inguinale.

3. The Frei test is negative in cases of inguinal adenitis due to syphilis, gonorrhea, chancroid, or nonspecific infections.

4. Venereal infections subsequent to lymphogranuloma inguinale has no effect on the Frei test.

5. The Frei test becomes positive at the time of bubo formation.

BIBLIOGRAPHY

(1) Frei, W. (Berlin). New Skin Reaction in Lymphogranuloma Inguinale. Klin. Woch., 1925. 4, 45, p. 2148 (quoted by Stannus).
(2) De Wolf and Van Cleve. J. A. M. A. 1932. 99, 13, p. 1065.
(3) Stannus, H. S. A Sixth Venereal Disease. 1933. p. 1.
(4) Findlay, G. M. Lancet. 1932. July 2. pp. 11–13.
(5) Stannus, H. S. A Sixth Venereal Disease. 1933. pp. 30–34.
(6) Gibson, P. L. Brit. Jour. Ven. Dis. Oct. 1931, vol. VII. no. 4.
(7) Lillie, R. D.: Inguinal Lymphadenitis. Arch. Path. 8–19, July 1929. Quoted by Amtman and Pilot, Arch., Derm., and Syph., no. 5, vol. 26. Nov. 1932. pp. 868–878.
(8) Stannus, H. S. A Sixth Venereal Disease. 1933. pp. 57–58.
(9) De Wolf and Van Cleve. J. A. M. A. 1932–99–13. p. 1067.
(10) Hellerstrom. Quoted by Stannus. A Sixth Venereal Disease. p. 53.
(11) Gregorio Edourd de Ann. d. Mal. Ven., Paris, July 1934. 24, 491. (Abstract in Venereal Disease Information, vol. 16, Jan. 1935, no. 1, p. 28.)

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AN EPIDEMIC OF BACILLARY DYSENTERY

By A. A. Shadday, Lieutenant Commander, Medical Corps, United States Navy

Bacillary dysentery is an infectious disease which is usually encountered in the acute stage. It is caused by a family of bacilli of which there are two important species and some related varieties. "The organisms credited with the etiology of this disease are characterized by the following characters: They are short rods, have no flagella, nonmotile, form no spores, do not liquefy gelatin, are Gram negative, ferment glucose and sometimes other sugars and alcohols without the formation of gas" (Andrewes and Inman). The bacillus discovered by Shiga in 1898 and again isolated by Kruse in 1900 is now regarded as a species distinct from that isolated by Flexner and the types related to it, classified by Hiss in 1904 as the Flexner, Strong, and Hiss-Russell or Y bacilli. The latter are distinguished from Shiga's bacillus by the fact that they ferment mannite. The species are serologically distinct. Shiga strains are all agglutinable by Shiga serum, while the mannite, fermenting bacilli are not. The latter form a group of five strains, called V, W, X, Y, Z, which are related by the production of group agglutinins, but differentiated from each other by the higher agglutination titer of their immune sera toward homologous strains than toward the others. This serological classification has superseded that made by Hiss on the basis of sugar fermentation. This disease has been clinically classified by Manson-Bahr into the following groups: (*a*) Mild, (*b*) acute, (*c*)

fulminating or toxic, (*d*) relapsing, and (*e*) chronic. The fulminating group is divided into two subgroups the choleraic and gangrenous.

An outbreak of bacillary dysentery occurred on the U. S. S. *Salt Lake City* on October 22, and continued until October 31, 1934. There were 91 cases admitted to the sick list. The average complement during the period concerned was 53 officers and 594 men. Fourteen percent of the personnel contracted the disease. As is usual in epidemics, it is suspected that some mild cases did not report for treatment and were therefore not admitted to the sick list, although precautions were taken to avoid this contingency, and it is doubted if more than a very limited number of cases failed to report.

The ship left Guantanamo Bay on the afternoon of October 20 and proceeded to Gonaives, Haiti, where she was anchored until the morning of the 22d, when we proceeded for Colon, Panama. No liberty was granted in Gonaives. The stay in Guantanamo Bay had been for a period of 3 weeks, the last 2 of which the ship had been at anchor just off Caracoles Point and Hospital Cay. During the late afternoon and night of the 22d, 12 men reported to the sick bay with a severe abdominal complaint and dysentery. Thirty-one men were admitted to the sick list on the second day of the epidemic, 29 on the third day, 6 on the fourth day, 3 on the fifth day, 2 on the sixth day, 1 on the seventh day, 2 on the eighth day, 2 on the ninth day, and 3 on the tenth day. All messes were involved except that of the commanding officer and warrant officers. All divisions had men afflicted except the H division (medical department). All food is prepared in one common galley. No food handler was afflicted on the first day except the wardroom cook, a Filipino, who had a severe case. Six mess cooks were affected later on during the outbreak.

The men reported on the first day of the outbreak as on subsequent days, complaining of severe griping pain in the abdomen; rectal tnesmus and almost a constant desire to defecate. Each man stated that the attack had come on abruptly from 1 to 6 hours previous. No one of these men gave a history of vomiting or complained of any nausea. Palpation of the abdomen revealed considerable tenderness over the transverse and descending colon. They were found to have a rapid pulse and an elevation of body temperature to 100°. The temperature after a few hours usually rose to 102°, was higher in some cases. These patients complained of headache and a few were delirious. The stools at first were clear with transparent mucus, but soon appeared to be of almost pure blood.

The examination of specimens of the stools of the first few patients revealed about as follows: Macroscopically, the specimen appeared to consist chiefly of blood and mucus; microscopically, innumerable red blood cells were seen and many polymorphoneculear leucocytes were noted. Numerous macrophages were seen in the field. Some

of the large phagocytic cells appeared to be ingesting a gram negative bacillus. Examination of the blood gave the following picture: W. b. c. 10,200. Neutrophiles: Band forms, 11 percent; segmented, 53 percent; lymphocytes, 32 percent; monocytes, 4 percent. From the above clinical and laboratory findings, the epidemic was tentatively diagnosed and treated as a bacillary dysentery. The diagnosis was later confirmed by the laboratory on the U. S. S. *Relief*. The report of a typical case reads as follows: "S. C. A. Sea. 1c. A nonmotile, gram negative bacillus isolated. Carbohydrate fermentation studies show reaction for Eberthella paradysenteriae (Hiss-Russell, type Y) and the organism is agglutinated by polyvalent dysentery serum." Specimens from all cases were not studied, as this was impossible, due to being at sea, but it is thought that a sufficient number, about 30 in all, collected at different times, were examined during the course of the epidemic together with the similarity in symptomatology and clinical course, to warrant the diagnosis of bacillary dysentery in all of the 91 cases.

By early morning on the second day, 43 of these patients were admitted to the sick list. They were isolated in the chief petty officers' living quarters. This compartment is on the second deck in the extreme after part of the ship. It is provided with separate bath and toilet facilities. The space is considered ideal for such an emergency. Typhoid precautions were instituted and apparently were effective as no attendant contracted the disease. The medication consisted of an initial therapeutic dose of oleum ricini and 20 cc of polyvalent antidysenteric serum, given intramuscularly. This was followed by bismuth subnitrate and salol by mouth. The diet consisted of barley water until the diarrhea stopped, which was usually about 3 days. The serum injection was not repeated except in a few severe cases; usually the severe symptoms subsided within 12 hours after the administration of serum. After the diarrhea stopped, a soft diet was given until the 10-day period had elapsed.

Six of these patients, average cases, were selected for massive doses of bismuth subnitrate by mouth. It was found that bismuth is not a specific for bacillary dysentery. No serum was given intravenously. One serum accident of severe proportions occurred, which the writer considered as anaphylactic. This patient gave no history which seemed to indicate desensitization. As is usual, about 10 percent of these patients developed serum sickness after an average interval of 8 days. They exhibited the usual symptomatology. The amount of serum given or the severity of the infection appeared to have no bearing on this condition. There were no serious complications; a few men developed a mild arthritis, usually of the knee joint; no sequelae except serum sickness; and no deaths. Additional serum was secured from the U. S. S. *Relief* at sea; also six members of

the Hospital Corps were temporarily transferred for duty during the emergency.

Bacillary dysentery occurs endemically in certain parts of the world, especially in the Tropics. It is said that during the Great War it prevailed on every front. The incubation period is considered from 2 to 7 days. Contamination of food and water is an important source of infection.

The ship left Port Arthur, Tex., on September 27, after a stay of 1 week where liberty was granted freely. It was necessary to take potable water from the beach in that port for the reason that it was impracticable to operate the ship's evaporators because of the heavy crude-oil pollution in the harbor. The health officer of that city reported no dysentery at the time of our visit. The ship anchored in Guantanamo Bay on September 30 opposite Caracoles Point, where she remained for the most part, until October 20. The water of the harbor was heavily polluted with fecal matter after the first few days of the fleet's stay there. It must be noted that there is very little current in Guantanamo Bay and that the discharges of some 42,000 men were being dumped into the harbor. The evaporating plant of the *Salt Lake City* is of the so-called "low-pressure type." The temperature of the water in the plant during this period ranged from 158° in a vacuum of from 19 to 24 inches. Samples of the drinking water on the ship were examined bacteriologically on the U. S. S. *Relief* as often as it was thought necessary and were in each instance found to be negative for gas formers and dysentery bacilli. The salt content of this water was reported to have not been greater during this period than 0.8 gr. per gallon.

A signal was received on October 13, 9 days prior to the outbreak on board, to the effect that bacillary dysentery had appeared in the battle force. Upon receipt of this information, the medical officer recommended additional sanitary precautions, the chief of which was that all harbor water be prohibited on board except in the flushing systems. This order was strictly adhered to. Fresh water was used to "clamp down" the decks, in the shower baths, and at all other places where sea water is ordinarily accessible. The running boats were in the water all of the time and harbor water was being used for scrubbing them down. The crews of the boats, liberty and recreation parties were subjected to the spray from the polluted water in the harbor. The percentage of infections among the men assigned to these boats was no higher than in any other group. Swimming was not permitted over the side, but was permitted near the officers' club. A check up on these officers and men did not reveal the source of infection, as the percentage does not run any higher in this group. This recreation also was stopped 9 days before the beginning of the epidemic.

No soft drinks of local manufacture were brought aboard in Guantanamo. The ice cream sold in the soda fountain was made with the powdered mix. The water used was that from the potable system as previously discussed. Ice cream which was manufactured at Guantanamo City was served to the general mess on October 17. On October 18 a signal was received to the effect that samples of potable water from some ships of the Scouting Force showed a high bacterial count and ordered that all potable water be chlorinated until 2 days after departing Guantanamo Bay area. That procedure was continued on this ship until after the epidemic had subsided.

No fresh milk was purchased by any of the messes in Guantanamo. Ice and fresh provisions such as lettuce, radishes, cabbage, and sweetpotatoes were purchased in Caimanera by all of the messes until October 18, when all such food from the beach was prohibited. This was done because of the signal referred to above stated that ice manufactured at Guantanamo City and sold at Caimanera had been found to have a high bacterial count. Fresh provisions and ice from the same source were used regularly by the naval station at Guantanamo and other ships of the fleet with seeming impunity, as no dysentery was reported.

Liberty to Guantanamo City was granted only on October 2, 20 days before the disease appeared. It was noted that very few men were in that liberty party. Liberty was later granted to chief petty officers. The only chief petty officer affected in this epidemic did not leave the ship while in Guantanamo Bay. Recreational parties were permitted on the naval station ashore. No liberty was granted the men to the "Red Barn" or Caimanera.

Men were sent to the rifle range at the naval station. No cases of bacillary dysentery were reported on the naval station at this period. The last available health report of Guantanamo City, which is for the month of August, does not list any cases of bacillary dysentery.

The ship was particularly free from flies at Guantanamo. According to the complaint record in the sick bay, no one had complained of a diarrhea for several weeks previous to the outbreak. There were no cases of chronic dysentery on the ship. All food handlers are inspected at regular intervals and a close watch is kept for complaints of a dysenteric nature. The following additional sanitary measures were invoked to check the spread of the infection.

(*a*) All men were instructed as to the nature of the disease and the usual modes of infection. They were told of the importance of reporting any complaint to the sick bay. This was repeated frequently by division officers.

(*b*) Basins containing 0.5 percent liquid cresol compound were placed in each head, wash room, and pantry. A sentry was stationed

in each of the crews' heads, whose duty it was to see that each man washed his hands in this solution before leaving the "head." This procedure was carried out strictly in the galley, butcher shop, bake shop, and all pantries.

(*c*) All cases of dysentery were promptly isolated and typhoid precautions maintained. No attendant contracted the disease. No patient was permitted to be out of the compartment where they were isolated until he had been symptom free for a period of 10 days.

(*d*) The regulations which govern the scullery were more exacting during this period.

(*e*) The drinking fountains were cleaned and the outlets flamed with a blow torch daily.

(*f*) All laundry was soaked in a mercurial solution before being sent to the laundry.

(*g*) The soda fountain was closed.

(*h*) The potable water was chlorinated.

(*i*) Sanitary regulations were tightened all along the line. It will be noted that while this epidemic hit with an explosive effect (72 cases within 3 days) it subsided promptly; the last case was admitted on the night of the tenth day.

The writer believes that the source of infection was the polluted and contaminated harbor water in Guantanamo Bay, the source of contamination being the case reported in the Battle Force on October 13. It is easy to imagine that the source of infection may have been one of the small rivers or streams, and there are several, which empty into Granadillo Bay. These streams drain a large area of the adjoining Province as well as the villages of Caimanera, Boqueron and the "Red Barn" districts. Granadillo Bay empties into Guantanamo Bay at Caracoles Point, near where the *Salt Lake City* was anchored. The velocity of the tidal current in this area is said to be but 0.1 knot. It is not impossible that a particular purchase of fresh provisions was contaminated by a gardner or handler before being delivered to market in Caimanera. This does not appear likely inasmuch as other ships, in general, escaped the infection. It is assumed that some of the fresh provisions brought to the ship by the market boat were sprayed with the harbor water or possibly contaminated by swabbing of gunwales and thwarts while the food was in the boat. It is the practice to wash down these boats in the early morning. It is believed that the infected-food theory is more tenable in view of the explosive nature of the outbreak than to assume that it was a "harbor-water-direct-to-mouth method." It is believed that all of these cases were infected on the same day and possibly by the same contaminated food. The variable resistance of different men to the organism and the different amounts of the infected food consumed would account for the elas-

ticity in the periods of incubation, it is believed. The best source of information available at the time of the outbreak indicated that the commanding officers' steward and the warrant officers' steward, the only messes not involved, did not have provisions in the boat on the morning of the 18th, the last day such provisions were permitted on board.

The writer believes that no ship which has a suspicious intestinal disease on board, particularly if a water-borne infection is suspected, should discharge feces into a harbor where the fleet is at anchor, especially Guantanamo Bay, which has very little current movement. It does not seem possible that so many men, who are gathered from every port in the Nation, could dump their effluence into such a small and relatively stagnated bay as that of Guantanamo for a period of 3 weeks without there being an outbreak of some water-borne disease, unless unusual precautions were exercised. A review of the literature reveals that this disease has been epidemic on ships of the United States Fleet on previous occasions while anchored in Guantanamo Bay. This epidemic was short-lived and comparatively mild in virulence as compared with some of the cases seen in the Orient; yet 14 percent of the complement was on the sick list while the ship was at sea. There is no reason to assume that a more virulent type of organism, Shigra bacillus for instance, would not be encountered in another epidemic of bacillary dysentery with disastrous results.

A THOUSAND APPLICANTS

By H. D. Templeton, Lieutenant, Medical Corps, United States Navy

A medical officer assigned to recruiting duty may expect his share of inquiries by both laymen and civilian physicians, and he may find himself in the embarrassing position of being unable to state specifically what percent of all applicants pass his physical examination, the percentage of major defects, and how his results compare with those of other districts. Such questions as these and many others of a similar character prompted us to compile various data on 1,000 applicants as they passed through the main recruiting station at Indianapolis, Ind.

This group of applicants is representative of the young adults of this district. It includes the occasional drifter, the country, small-town, and city boy, from the average family whose circumstances have been so altered by prevailing economic conditions that he is denied continuance of education or employment.

Every applicant, with the exception of 119 who were rejected for various causes other than physical defects, was given a thorough

physical examination. Regardless of how apparent a major defect might be, a complete physical examination was made, (1) thus assuring the applicant he had received every consideration, (2) his physical disqualifications were explained to him, (3) complete data for form 54 was thus obtained, (4) a more comprehensive appreciation of the various physical defects existing in applicants of this district, and (5) a relative knowledge of how much man-power might be reclaimed and made acceptable in the event of a national emergency.

Our survey yielded the following data:

TABLE 1

Applicants	Applicants examined	Rejected, other causes	Rejected on physical	Applicants accepted
1,000	881	119	623	258
Percentage of all applicants	88.1	11.9	62.3	25.8
Percentage of those examined	----	----	71.0	29.0

Physical rating of accepted applicants: A, 124—12.4 percent; B, 94—9.4 percent; C, 40—4.0 percent.

On the assumption that our physical examinations were fairly reliable, it is reasonable to conclude that 25 percent of the young men of this district should qualify physically for enlistment. Approximately one-half of those qualifying will be excellent material, one-third may be classed as average, and the remainder will be acceptable.

The 119 previously referred to constitute a group composed of drifters, married, and over-age applicants, and those with police records of major offenses. This group was included in our survey since we were more concerned with the information obtained from 1,000 applicants, rather than a compilation of data on 1,000 physical examinations.

II

The following table, no. 2, is a classification of all defects noted in the 623 physically disqualified applicants from a total of 881 examined.

TABLE No. 2

	All dental defects	Flat feet	Defective vision	Defective color perception	Defective hearing	Hernia	Infected ears	Deformities	Underweight	Underheight	Unclassified—other causes	Total
All defects noted	401	279	79	54	12	56	12	63	177	15	394	1,539
Major disqualifying defects	196	131	73	52	8	17	5	15	9	9	108	623

In the above table we have classified under various headings, a total of 1,539 physical defects present in the 623 disqualified applicants. Each applicant was credited with one major defect, which represented, in practically all cases, a physical handicap not amenable to correction that would render the applicant acceptable for enlistment under present physical standards. For example, an applicant may be underweight, have dental defects, insufficient physical development, and defective color perception; in such a case we regarded defective color perception as the outstanding disqualifying defect.

Four hundred and one, practically two-thirds of the 623, had marked dental defects, of which 196 were classed as major or irreparable.

Moderately depressed arches and marked eversion of the feet were included under the heading "flat feet", however, only 131 cases of defective feet were regarded as major and representing the condition pes planus.

The 56 cases appearing under the heading of hernia represent varying degrees of abnormality, from the large and relaxed external ring to those constituting the 17 major cases with herniae that had descended to, or beyond the external inguinal ring.

It has not been our practice to regard underweight as a major disqualifying defect except in nine cases; we have found in practically every instance other body disproportions present to such a degree that these cases are more appropriately classified as "insufficient physical development." A goodly number of such cases are included in that group under the heading of "unclassified other causes," the remainder is made up of miscellaneous diseases and conditions such as large varicocele, cardiac conditions, history of enuresis, epilepsy, suspected mental diseases, and hypospadias.

III

In the following table no. 3, we have taken our 623 physically rejected applicants and redistributed them under headings that more clearly identify the causes for rejection. Under each such heading the percentages for the disqualifying defect is noted as it relates to both the total number of rejections and the total examinations.

TABLE No. 3

	All dental defects			Flat feet	Defective vision	Defective color perception	Defective hearing	Hernia	Infected ears	Deformities	Underweight	Underheight	Overheight	Insufficient physical development	Unclassified—other causes	Total
	Caries	Mal occlusion	No opposing molars													
Rejected	73	23	100	131	73	52	8	17	5	15	9	9	3	54	51	623
Percent of total 623 rejections	11.7	3.7	16.0	21.0	11.7	8.3	1.2	2.7	0.8	2.4	1.4	1.4	0.4	8.6	8.1	....
Percent of all physical examinations (881)	8.2	2.5	11.2	14.8	8.2	6.0	0.9	2.0	0.5	1.7	1.0	1.0	0.3	6.1	5.7	....

Dental defects account for the greatest number of rejections—31.9 percent, and 21.9 percent of all applicants examined; either way one applies these percentages they are high and disclosed the fact that 1 of every 5 young adults has dental caries or has lost teeth to the extent that he is not acceptable for enlistment.

One hundred and thirty-one, 21 percent of all rejections, and 14 percent of all examined, were definitely flat footed. Just at this point it might be well to note that very few admitted symptoms of foot strain and also that very few have worn arch supports. We have every reason to believe these boys gave truthful answers, which led us to question whether or not depressed arches may have developed in childhood.

Defective vision which accounts for 11.7 percent of those rejected, or 8.2 percent of all applicants examined, stands third in importance of the major disqualifying defects. The vast majority claimed their vision had never been tested. On the other hand, not infrequently an applicant stated he had worn glasses a year or more in childhood; in a large percent of such cases our examination revealed no defect of vision. This led us to conclude that possibly early treatment of eye strain or a minor refractive error might have reduced this relatively high percentage of those with defective vision.

The 54 applicants grouped under the heading "insufficient physical development", represents a class definitely undernourished. We have limited this group to those poorly developed and malnourished boys whose general health might be further impaired by the strenuous life and duties of a sailor.

These tables with their various totals and percentages were not prepared with the idea of proving any contentions, but rather for the purpose of interpreting results. The importance of such interpretations lies with the individual; his deductions may not coincide with ours, however it is our belief that those applicants rejected because of dental defects, underweight, and insufficient physical development and constituting 41.4 percent of all rejections or 29 percent of all applicants examined, represent the undernourished adults

of the ages acceptable for enlistment of this district. More conclusive and substantiating evidence of this statement is borne out by the fact that over 55 percent of all cases of insufficient physical development showed major dental defects, and conversely, 34 percent of all rejections revealed major developmental defects.

On analysis of all cases of pes planus, 86 had nutritional and developmental defects; of these, dental caries existed in 54. Underweight, poor physique, and dental defects existed in the remaining 34. We are not contending that malnutrition plays a major role in our cases of pes planus, but it is quite evident in two-thirds of all such cases.

Whether or not these percentages and our conclusions regarding evidence of malnutrition are correct, we believe few will maintain that it has not increased among young adults throughout the past 5 years. It appears quite evident that this period of economic depression has denied many of our growing youths adequate nourishment essential for development.

Recruiting has not been adversely affected by such conditions. Small monthly quotas have worked to our advantage and selection of only the very best material is possible at this time; however, should economic conditions improve within the next 6 or 12 months, we believe we shall lose all or a majority of our class A applicants. Obviously, they are the best material for industry as well as the naval service.

IV

A review of all tabulated results was made in an attempt to obtain some appreciation of how much material might be reclaimed in the event of a national emergency, demanding quick expansion of naval personnel. Only approximate figures are quoted, since such results depend on what exceptions may be made to our present fairly rigid physical requirements. The following table no. 4, represents an estimated number of those that might be reclaimed for limited duties.

TABLE No. 4

Dental defects	Defective vision	Defective color perception	Hernia	Underweight	Underheight	Unclassified—other causes	Total
25	32	32	10	2	1	43	145

The above-tabulated results indicate that approximately 20 to 25 percent of the 623 rejected physically might be made acceptable for the period of an emergency. These figures are only relative and those tabulated represent the best material from our files of rejections.

CONCLUSIONS

This survey has been helpful to us inasmuch as fairly reliable percentages have been obtained by which we may estimate the recruiting possibilities in this district. This group of 1,000 includes applicants from every social and economic strata.

We have learned that approximately 80 percent of all disqualifying defects are quite obvious and should be recognized by a properly trained recruiter at a substation.

The vast majority of our best material is recruited from small communities and agricultural areas; thus it appears that substations in small cities are more favorably located to supply the best applicants.

We have found parents most cooperative in furthering their son's enlistment by correcting minor physical defects.

Thus, it is reasonable to assume that, of 1,000 applicants in this district, 260 will qualify physically for general duty, and in the event of a national emergency an additional 160 might be made available for duty after certain disqualifying defects are corrected or waived.

Of primary interest to this community is the large number of dental defects, flat feet, defective vision, and malnutrition. Much of this appears to be a public-health problem made more difficult by the depression. This survey suggests that periodic physical examinations of school children might have resulted in the correction of many nutritional and developmental defects.

The statement is frequently made that the Navy's physical standards are far too rigid. Those making such an assertion are not familiar with the many and arduous duties performed by our personnel. Their impressions are formed from news-reel films of the Navy "on parade", and it is quite obvious that they know little of life "below deck." A detailed account of such a life is far less convincing than a comparison of values received in service rendered the taxpayer by men who have satisfied the highest physical requirements. When the taxpayer realizes that his interests are best served by maintaining rigid physical qualifications, he appreciates the efforts exerted by the naval service in its selection of the best material.

NEUROPSYCHIATRIC SERVICE, U. S. S. RELIEF—AN ANALYSIS OF 1 YEAR'S WORK

By F. L. McDANIEL, Commander, Medical Corps, United States Navy

It may be of some interest to give an analysis of nervous and mental cases handled on board the hospital ship of the fleet during a certain specified period of time. In order to do this the year

1934 has been taken. During this year the hospital ship functioned as a base hospital, medical center, and clearing house for the entire United States Fleet. A considerable portion of the time was spent away from the continental United States during the cruise of the fleet from the west to the east coast and return. During this period, 1934, practically all cases of mental and nervous disorder originating in the fleet were transferred to the U. S. S. *Relief* for treatment and disposition. This was accomplished by the close cooperation of the force surgeons with the senior medical officer of the *Relief*.

In the following analysis attention is called to the fact that the figures given represent individual patients, diagnosed and treated, and their disposition.

During the calendar year ending December 31, 1934, there were admitted to the neuropsychiatric service of this vessel the following:

DISEASES OF THE NERVOUS SYSTEM (CLASS XVII)

Total number of patients, 30. Following diagnoses were established:

Effort syndrome	2	Neuritis, deltoid	2
Encephalitis, acute	1	Neuritis, sciatic	5
Epilepsy	5	Neurosis, traumatic	2
Narcolepsy	1	Paralysis agitans	1
Neuralgia	2	Paralysis, nerve	9

In all the above, the history indicated that condition was acquired incident to service conditions. Of these class XVII patients, disposition was as follows:

Transferred to naval hospital ashore	15
Invalided from service (medical survey)	1
Returned to duty, recovered	13
Still under treatment on board	1

Percentage of patients with definite diagnosis of disease of the nervous system returned to duty after treatment on board, 43 percent.

The condition diagnosed *narcolepsy*, although carried as an italicized diagnosis in the Navy Nomenclature, is recognized as a clinical entity. This patient presented typical history and clinical syndrome. He was retained on the sick list for a period of 77 days. Treatment of ephedrine sulphate three-eighths gr. t-i-d as recommended by Dr. Jacobsen (Jour. Ped. St. Louis 1933) and others. He showed marked improvement and for some time prior to his discharge to duty all symptoms had cleared up.

A patient dignosed as paralysis, nerve, pupillary, parasympathetic, is worthy of note. He was admitted with pupils widely dilated and fixed to light and accommodation. No other neurological signs or symptoms. Complete examination and serological findings nega-

tive for possible etiological factor. Paralysis disappeared completely in 48 hours. Patient admitted drinking "some kind of wine" while ashore on liberty the day prior to onset.

Admissions for diseases of nervous system by occupational groups

	Effort syndrome	Encephalitis, acute	Epilepsy	Narcolepsy	Neuralgia	Neuritis		Neurosis, traumatic	Paralysis	
						Deltoid	Sciatic		Agitans	Nerve
Officers, Navy and marine						1	1			
Electricians		1			1			1		
Engine-room force				1						
Fire-room force	1								1	1
Culinary force										2
Clerical force										1
Marines							1			
Deck force	1		5			1	3	1		5
Ordnance rate						1				

SYPHILIS (OF CENTRAL NERVOUS SYSTEM) CLASS XII

The diagnosis of syphilis (of central nervous system) was established in eight cases admitted to medical service.

Six of the above class XII patients were admitted to the medical service with admission diagnoses as follows:

Arterial hypertension	1
Diagnosis undetermined (brain abscess)	1
Tonsillitis, chronic	1
Diagnosis undetermined (psychoneurosis neurasthenia)	1
Paralysis of nerve (right tibial)	1
Vomiting	1

The patient admitted with diagnosis of paralysis (right tibial), nerve showed paralysis of upper motor neurone type of entire right lower extremity. History of sudden onset while marching in parade. Proved to be central lesion with meningo-vascular syphilis.

Patient admitted with diagnosis of tonsillitis, chronic, on admission complained of severe headache and exhibited transitory ocular palsy involving left external rectus. Proved to be meningo-vascular type of neurosyphilis.

Patient admitted with diagnosis of vomiting, gave history of persistent vomiting for past 24 hours. No history of dietetic indescretion. No pain in abdomen. Proved to have syphilis of central nervous system, tabetic type.

Patient admitted with diagnosis undetermined (brain abscess) was found to have typical chancre on penis and dark-field examination positive for treponema. Condition proved to be primary syphilis with early involvement of meninges. He responded to treatment and was returned to duty.

Of the above class XII cases, seven were transferred to naval hospitals ashore for further treatment. One patient returned to duty under treatment.

DISEASES OF THE MIND (CLASS XV)

Year ending December 31, 1934, the following patients coming under classification XV were admitted and diagnoses were established as follows:

Total number (diagnoses established)	45
Constitutional psychopathic inferiority, without psychosis	2
Constitutional psychopathic state, emotional instability	7
Constitutional psychopathic state, inadequate personality	1
Constitutional psychopathic state, sexual psychopathy	1
Dementia praecox	9
Psychoneurosis, hysteria	4
Psychoneurosis, neurasthenia	4
Psychoneurosis, psychasthenia	2
Psychoneurosis, unclassified	5
Psychosis intoxication, alcoholic	1
Psychosis, manic depressive	2
Psychosis unclassified	4
Somnambulism	3

In addition to the above, there were 11 patients admitted with diagnosis undetermined for mental observation. These were disposed of as follows:

No disease (mental observation), to duty	7
C. and R. A. syphilis (central nervous system)	1
C. and R. A. gonococcus infection prostate	1
C. and R. A. oxyuriasis	1
C. and R. A. hypothyroidism	1

As has often been noted in the Annual Report of the Surgeon General, dementia praecox heads the list of admissions for mental diseases. The reason for this is, of course, because the greater proportion of the men in the Navy are in the schizophrenic age group. However, in the admissions for this vessel one will note that the psychoneuroses and the constitutional psychopathic states are responsible for a high percentage of admissions. The reason for this is not far to seek. This vessel obtains its patients almost entirely from the active ships of the fleet. A great many of the dementia praecox patients are weeded out either during or shortly after leaving the training stations. The constitutional psychopathics and the psychoneurotics are able to weather the storm for a time, but eventually break down under the stress and strain of the artificial environment such as is met with aboard seagoing vessels of the fleet.

Disposition of the above class XV cases

Transferred to hospital	39
To duty	6

The diagnosis of hypothyroidism noted above was the case of an officer patient admitted with diagnosis of psychoneurosis, neurasthenia (undetermined). He presented a symptom complex of malaise, fatigability, loss of kinetic drive, and emotional depression. Basal metabolic rate was minus 16 percent and after 1 week minus 14 percent. Under thyroid administration basal metabolic rate was plus 1 percent. Patient immediately improved physically and mentally, and was returned to duty.

Disposition of patients admitted under class XV

Total number of patients admitted for mental observation, diagnosis being either established or undetermined	56
Diagnosis established under class XV	45
Transferred to hospital	39
Returned to duty after period of observation and treatment	6
Returned to duty, diagnosis no disease (mental observation)	7
Diagnosis established in other classifications	4

***Admissions class XV by occupational groups** (diagnosis established)*

	Psychoneurosis (all)	Constitutional psychopathic inferiority without psychosis	Constitutional psychopathic states (all)	Dementia praecox	Psychosis			Somnambulism
					Alcohol intoxication	Unclassified	Manic depressive	
Officers, Navy and Marine	3						1	
Electricians	1							
Engine-room force						1		
Fire-room force	1			2		1		1
Culinary	1			1				
Clerical	1							
Marines			3					2
Deck force	8	2	6	5	1	2	1	
Ordnance rating				1				

COMMENT.—It will be noted that the largest number of admissions are from the deck force. This group includes a majority of the lower ratings, as apprentice seaman and seaman first and second class. It is to be expected that most of the dementia praecox cases and psychoneuroses will be furnished by this group.

Admissions class XV according to age groups

	16–19	20–24	25–29	30–34	35–39	40–44	45–49	50–54
Psychoneuroses (all)	4	4	3	1	2			1
Constitutional psychopathic inferiority without psychosis		2						
Constitutional psychopathic state (all)	2	5	2					
Dementia praecox	2	6	1					
Psychosis intoxication alcohol					1			
Psychosis unclassified		2	2					
Psychosis manic depressive				1				1
Somnambulism	1	1			1			

COMMENT.—It is interesting to note that 80 percent of all patients diagnosed in class XV were under 29 years of age, and about 65 percent were under 24 years of age.

The problems involved in treating mental patients on a seagoing vessel serving as a hospital ship might well be discussed in a separate paper. Mechanical restraint has been found necessary in but very few instances. Only patients who were actively psychotic and disturbed were confined to the locked ward. During the year no psychotic patient while on board the hospital ship harmed either himself or any attendant. Patients suffering from organic disorders of the nervous system were treated as indicated. Those suffering from the so-called "functional conditions", psychoses and psychoneuroses, were given the advantage of heliotherapy, exercise and amusement (movies, etc.), and the physiotherapy and hydrotherapy department was utilized to the fullest extent. Psychotherapy was employed in selected cases.

Although the total number of neurological and psychiatric patients handled is relatively small, attention is called to the variety of conditions studied. One is afforded excellent opportunity for a well-rounded study of neuropsychiatric conditions during a tour of duty in this hospital ship.

THE PROPHYLAXIS OF VENEREAL DISEASE

By J. A. MILLSPAUGH, Lieutenant, junior grade, Medical Corps, United States Navy

An Annapolis graduate assured me that while at the academy he was given an infallible formula for venereal prophylaxis, namely, to remain chaste. Since so eminent a syphilographer as J. H. Stokes states "the physician fails in both duty and privilege if he does not swing what influence he can in behalf of intellectual and spiritual rather than merely mechanical and chemical preventives of syphilis and gonorrhea", I invariably exhort, particularly young recruits, to rely upon the above advice.

Nevertheless, centuries of experience denote that the majority of men are not voluntarily and consistently continent. Since year after year the Surgeon General's report reveals venereal disease a leading cause of morbidity, despite punitive measures directed against the infected, and in the face of known nearly certain preventive measures conceded by authorities, there is definitely misunderstanding of or disregard for prophylaxis.

Soap and water is not only a good spirocheticide but it is also a good gonococcocide; furthermore, chancroids and venereal warts are outstandingly associated with unclean habits.

If, following exposure, the subject would take a long, hot shower using plenty of soap, not only would many venereal infections be prevented but there would be a marked decrease in incidence of parasitic infestations, namely crabs and scabies.

Alcoholism is responsible for many venereal infections. In small quantities alcohol is an aphrodisiac, thus stimulus is furnished. Alcohol releases inhibitions, restraint is cast aside, and resistance diminished. Judgment and discrimination are impaired, visual acuity lessened. Immoderate quantities retard culmination of orgasm, thereby affording longer exposure. Coordination is lost if not consciousness, and if prophylaxis is taken it is likely delayed and incomplete.

It has been related that, following illicit intercourse, men have used liquor as a penile-scrotal-perineal wash. For obvious reasons this custom is not without value in the absence of better measures.

All writers concerned with venereal prophylaxis advise the exposed to urinate immediately as a preliminary to chemical prophylaxis. Some of the initiated drink beer purposefully before exposure to insure ability to urinate later. Urination serves as a mechanical ablution of the urethra.

It is necessary to assume that all prostitutes are infected. Display of a certificate asserting the bearer is free from venereal infection is of value, if valid, only to show that she does visit a doctor and that she was presumably noninfectious at the time of examination. In all probability the prostitute has been exposed, and therefore possibly infected many times since the last examination, even though recent. It is often difficult to demonstrate latent or chronic venereal infection of the female in repeated painstaking examinations.

At present, though not ideal, the condom is at once the most widely used and safest prophylactic. It is the nearest approach to a universal venereal prophylaxis. It is easily obtainable, carried, and applied. Though sold ostensibly for protection from venereal disease only, innumerable people use it solely as a contraceptive device. Inspection and moderate stretching assure the condom is intact and elastic enough to withstand usual usage. The condom should be carefully removed as a glove is stripped from the hand to avoid contact with the contaminated external surface. Needless to state, the condom should be applied before penile intromission. This is mentioned because many people practice a modified coitus interruptus, applying the condom just previous to ejaculation.

Strikingly, the three principal objections to the use of the condom, medical, religious, and personal, are all based upon the fact that it is interference with a natural function. Some physiologists, sexologists, and urologists believe that prolongation of the period of unsatisfied desire with a correspondingly lengthened period of passive conjestion, consequent upon use of the condom, contribute to prostato-vesiculitis and eventual reduction of fertility in the male. The religious objection is that the condom frustates the prime

purpose of intercourse, procreation. As for the personal objection, not only many men but their sexual consorts likewise dislike the condom because thermal and tactile sensations are attenuated.

The condom, being in addition to a good prophylactic, a good contraceptive, serves a twofold purpose by obviating the unfortunate circumstances of illegitimate pregnancy.

Extragenital venereal infection is not prevented by the condom, but such infection is so uncommon as to be nearly negligible. The combined experience of five recently contacted naval medical officers representing a cumulative period of 64 years, accounted for 10 infections, against thousands of cases with genital pathogenesis.

The ship's service store of a battleship to which I was attached sold, at nearly cost, a great number of a reliable brand of condoms. A change of the officer in charge and discontinuance of this practice was followed by an increase in the venereal list.

Since it would probably be difficult to secure official issue of the condom as a venereal prophylactic, this alternative is suggested: That condoms be sold at cost aboard all ships and stations.

All modern writers I have read distinguish between the chemical prophylaxis of syphilis and gonorrhea. They are in accord in recommending 30 to 50 percent calomel ointment as the medical prophylaxis for syphilis. It is further agreed that the highest percentage of protection occurs when the calomel ointment is applied within an hour following exposure and allowed to remain for a period of hours. Eisendrath and Rolnick in their text Urology, second edition, page 274, state:

> The external application of a 33 to 50 percent calomel ointment immediately after exposure following the washing of the soft parts, is an excellent prophylactic against both hard and soft chancre.

Consensus of recent advice recommends a urethral injection of protargol 2 percent, to be retained 5 minutes, as the most valuable chemical prophylaxis for gonorrhea. Protargol is a protein silver salt, soluble in water, nonaffected by albumen, slightly irritant, penetrating, and gonococcicidal. Argyrol 10 percent, silver nucleinate 10 percent, mercurochrome (220) 2 percent, neosilvol 10 percent, merthiolate 1–5000, protargentum 2 percent, hexylresorcinal and potassium permanganate 1–3000 are likewise, though not so widely, recommended.

There is a discrepancy of opinion among medical officers and Hospital Corps men concerning the purpose of the sanitube in venereal prophylaxis. This is due in large measure to an attempt made during the World War to provide a dual prophylactic for syphilis and gonorrhea by incorporating 0.5 percent protargol or thymol in the calomel ointment. Thymol is freely soluble in fats

and oils; it is antiseptic and germicidal. Those who had knowledge of this combined prophylaxis have retained, fostered, and passed on the informaton. I have been unable to determine why protargol or thymol have been withdrawn from the formulas now used by the services.

The Handbook of the Hospital Corps, United States Navy (1930) page 571 under Venereal Prophylaxis states:

"The formula for the 'sanitube' can be obtained from books on genito-urinary diseases but it is essentially a 30-percent calomel ointment made up with lanolin.

The Venereal Diseases (an outline of their management prepared under the direction of the Surgeon General of the Army for the use of medical officers), third edition, 1920, gives the formula for calomel ointment used in the United States Army as follows:

	Parts
Hydragyri chloridium mite	30
Adeps benzoinatus	65
Cera alba, U.S.P.	5

Maj. B. F. Duckwall and Maj. W. C. Cox of the United States Army (Medical Corps) in An Experiment in the Use of Individual Tubes in the Prophylaxis of Venereal Disease (The Military Surgeon, Aug. 31) state:

The 30 percent calomel is the recognized standard spirocheticide for local use, and is claimed by Vedder and others to act intraurethrally as a gonococcicide.

They selected the following preparation for study:

	Parts
Sodium benzoate	2
Mild mercurous chloride	30
Lanolin, qs. ad	100

The 2 percent sodium benzoate was used as a gonococcicide. Their conclusions were not heartening. It is significant that this formula has not supplanted that above.

An ointment prepared by Ganduceau is intended as a prophylactic against both syphilis and gonorrhea. The formula is:

	Grams
Mercuric cyanid	0.075
Thymol	1.750
Mercurous chloride	25
Wool fat	50
Petrolatum	100

In answer to queries concerning this ointment, the editor of Queries and Minor Notes, J. A. M. A. (Mar. 8, 1924) states:

It will be noted that the ointment consists of a goodly portion of petrolatum. As petrolatum is immiscible with watery solutions, it is doubtful whether antiseptics would be brought in intimate contact with the gonococci.

From this same source, I quote:

The most popular prophylactic ointment is calomel ointment which still has many proponents, particularly in the United States Army, as an effective preventive for syphilis, though *it is not effective against gonorrhea.* [The italic is mine.] It will be remembered, however, that in the United States Army, the application of the ointment is only one step; the soldier is also required to report for prophylactic treatment, in which injections of organic silver compounds are employed.

The directions accompanying the sanitube are misleading. It is assumed by many reading them that the sanitube is a general venereal prophylactic, especially because one-half the contents are to be injected. Most chancres occur externally; the layman is not familiar with intraurethral infection save gonorrhea, and he believes the intraurethral injection protects from gonorrhea. It is a moot question whether the sanitube serves as a mechanical deterent to the gonococcus. Mild mercurous chloride is antiseptic. It is used internally and externally for its disinfectant properties. There is no universally efficacious antiseptic or disinfectant. Tetanus bacillus is said to live in pure carbolic acid. Nowhere do I find mild mercurous chloride recommended primarily as a gonococcide. Some patients who have used a sanitube immediately and properly developed gonorrhea. In most instances the layman is not sufficiently informed concerning complete venereal prophylaxis; nor is the enlisted man concerned with the ingredients of a prophylactic. He procures a little packet from the sick bay or gangway watch, goes ashore, exposes himself, uses the sanitube as directed, and does nothing else as far as prophylaxis is concerned. Three to five days later he develops an acute gonorrhea in too many cases. No authority, so far as I am aware, recommends the sanitube as a preventive of gonorrhea. I questioned confidentially the first 10 men casually encountered. Their rates, the questions asked, and the replies are:

No.	Rate	Does the sanitube protect against syphilis?	Does the sanitube protect against gonorrhea?	Have you had syphilis?	Have you had gonorrhea?
1	Sea 2c	Yes	Yes	No	Yes.
2	AMM 3c	Yes	No	No	No.
3	AMM 2c	Yes	Yes	No	Yes.
4	Mus 1c	Yes	Yes	Yes	Yes.
5	Pvt., U. S. M. C.	Yes	Yes	No	Yes.
6	Tptr., U. S. M. C.	Yes	Yes	No	No.
7	Pvt., U. S. M. C.	Yes	Yes	Yes	Yes.
8	do	Yes	No	Yes	Yes.
9	P. 3c	Yes	Yes	No	No.
10	A. C. M. M.	I don't know	I don't know	No.	No.

While this is a woefully small number upon which to base any definite hypothesis, it is representative of nearly 5 years of unrecorded experience. I hazard there is scarcely a station or ship in

the service where similar results could not be approximated by questioning.

The directions accompanying the sanitube should state its prime purpose—to prevent syphilis—and add that additional prophylaxis, namely, an intraurethral injection of protargol 2 percent (or a similar effective gonococcide) should be taken before the sanitube is used, to prevent gonorrhea. This brings up another difficulty, all modern prophylaxis bulletins recommend the use of the gonococcide previous to that of the sanitube. Herein lies the cause of many cases of gonorrhea. Especially during prolonged liberty or leave, proper gonorrheal prophylaxis is not readily available. With those who indulge in multiple coitus within a relatively brief period, proper chemical prophylaxis is too complicated. The condom is the best present solution for these difficulties. For those who will not use the condom, there is a definite need of a reliable combined chemical preventive of venereal diseases. It does not seem that such a prescription would be impossible to an enterprising pharmacologist. The proprietor of an ethical pharmacy informs me that he has no combined prophylactic for syphilis and gonorrhea. In fact, he supplies venereal prophylaxis only upon prescription.

Pelouze believes that "many of the commercially exploited prophylactics are decidedly inefficient." He has "treated a number of cases of gonorrhea occurring after the apparently proper use of two of them that have gained much reputation among the initiated." One commercial product is purported to be "The Combined Preventive of Sexual Diseases." This is essentially a reproduction of the sanitube. The formula was divulged upon request, but I was asked not to publish it.

There is not an instance in the service where a medical officer on independent duty and dealing with any number of personnel does not encounter gonorrhea in his clinical material. Some medical officers are averse to, disinterested in, or insufficiently informed concerning an acceptable routine modern treatment for this important disease. There are a multiplicity of treatments and it is true that there is no very wide general accord. For this reason, it is urged that an effort be made to standardize in a minimal manner. That excellent book, Gonococcal Urethritis in the Male, by Pelouze, which is on the Supply Table and widely dispersed throughout service activities, might serve as an excellent guide. Men are transferred, occasionally, in violation of paragraph 703, chapter VI, Manual of the Medical Department; more are discharged from treatment before recognized criteria of cure are exercised (see General Order 69, amended). It is my opinion that no infected man, save in grave necessity, should

be ordered to a ship or station minus a medical officer until he is pronounced cured of gonorrhea, or until he has had a minimum of 1 year's continuous antiluetic treatment. Unnecessary hardship for men with recalcitrant infections would be lessened were they allowed liberty under surveillance. Many recrudescences and unfortunate breaks in treatment, favoring chronicity and dissemination would be eliminated, the individual's best interest served, and the ideals of public health subserved.

CONCLUSIONS

Venereal disease incidence in the United States Navy would be decreased if:

1. Abjuration of exposure is emphasized.
2. Thorough bathing and urination following exposure.
3. Drunkenness were avoided.
4. All illicit consorts were considered potentially infections.
5. Condoms were sold at cost aboard all ships and stations.
6. The sanitube were depended upon solely as a spirocheticide.
7. An intraurethral injection of a suitable gonococcicide were used in addition to and preferably before the sanitube.
8. The infected were restrained until cured.

BIBLIOGRAPHY

(1) Modern Clinical Syphilology, Hohn H. Stokes (W. B. Saunders, 1928).
(2) Urology, Eisendrath and Rolnick (J. B. Lippincott, 1930).
(3) Gonoccocal Urethritis in the Male, P. S. Pelouze (W. B. Saunders, 1929).
(4) Practical Therapeutics, H. A. Hare (Lea and Febiger, 1930).
(5) Text Book of Medicine, R. W. Cecil (W. B. Saunders, 1931).
(6) United States Dispensary (Twentieth Edition).
(7) Handbook of the Hospital Corps, United States Navy, 1930.
(8) Manual of the Medical Department, United States Navy, 1927.
(9) General Orders, United States Navy, 1926.
(10) The Venereal Diseases (An Outline of Their Management, Prepared Under the Direction of the Surgeon General of the Army for the Use of Medical Officers), 3d edition, 1920.
(11) Setherstrom, C. A. Development of Venereal Prophylaxis in the U. S. Navy (U. S. Naval Medical Bulletin) 24: 31–42, January 1926.
(12) Morgan, A. G., Prophylaxis and Treatment of Venereal Disease in the U. S., Venereal Disease Information August 20, 1931.
(13) Ashford, M. and Hathaway, L. M., Military Surgeon, February 1932. Hexylresorcinal as a Venereal Prophylactic.
(14) Military Surgeon, August 1931. An Experiment in the use of Individual Tubes in the Prophylaxis of Venereal Disease, Duckwall, B. F. and Cox, W. C.
(15) Hancock, J., Immediate Self-Disinfection After Sex Exposure as a Public Health Activity (Southern Medical Journal, April 1927).
(16) Journal American Medical Association, 82: 812–813, March 8, 1924.

THE INFLUENCE OF INCREASED BAROMETRIC PRESSURE ON THE PULSE RATE AND ARTERIAL BLOOD PRESSURE[1,2]

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Changes have been observed in the pulse rate and arterial blood pressure of men exposed to increased barometric pressure, but no detailed study has been reported. Since physical efficiency and susceptibility to caisson disease may be affected by changes in pulse and blood pressure, the present systematic investigations were undertaken.

Method.—The method used by Schneider and Truesdell (1922) for the study of pulse, blood pressure, and pulse pressure during recumbency, standing, and after exercise was used.

The test was first conducted in the large diving tank at atmospheric pressure and then air pressure applied to equal whatever depth of sea water was desired and the test repeated. Thus, for every test under increased air pressure, there is a corresponding control test at atmospheric pressure done during the same hour. Several tests were performed on all subjects at atmospheric pressure to accustom them to the routine. Four subjects were used in each experimental run at atmospheric pressure and the same four immediately repeated under increased air pressure.

Thirty-one men and officers attached to the Deep Sea Diving School and the Experimental Diving Unit, Navy Yard, Washington, D. C., acted as the subjects for this study. All are experienced divers and accustomed to taking pressure frequently. A routine history of the subject's activity for the previous 24 hours was recorded and a physical examination was conducted prior to every exposure to increased air pressure.

Whenever increased air pressure is referred to in this paper it will be given in atmospheres absolute.

Apparatus.—The experiments were carried out in a vertical cylindrical diving tank, 10 feet 1 inch in height and 9 feet 10 inches in diameter with walls of 2-inch steel, tested to a pressure of 400 pounds per square inch. This tank was used because of the head room afforded which allowed performance of the standard stepping exercise. The tank is fitted with an airtight hatch on the upper end which opens downward into the tank. There are six 4½-inch ports in the side for observation purposes. The tank is well lighted on the inside by electric lights and is equipped with loud-speaker and telephone. For this experiment two folding cots, a chair, and a

[1] From the Laboratory of the Experimental Diving Unit, Navy Yard, Washington, D. C.
[2] Received for publication Jan. 5, 1935.

table were added. The only other apparatus used was a mercury sphygmomanometer, stethoscope, and stop watch.

Experimental data—Pulse rate in recumbency.—The pulse rate of the reclining position at atmospheric pressure for all of our cases ranged between 52 and 108 with an average for the entire group of 124 individual tests of 76.07. The average as given by Schneider and Truesdell (1922) for their group of 200 was 75.21 ± 0.47.

The pulse rate of the reclining position under increased air pressure showed an average slowing of 9.12 beats per minute per individual for all pressures studied. The average diminution found by Heller, Mager, and von Schrotter (1900) was 14.6 beats per minute in 35 men exposed to pressures up to 3.5 atmospheres. Our average slowing of standing pulse for all pressures was 11.1 beats per minute while exercise pulse slowed 12.1 beats per minute. By reference to table 1 the average pulse rate, and the average decrease in pulse rate per individual per minute for each pressure are shown.

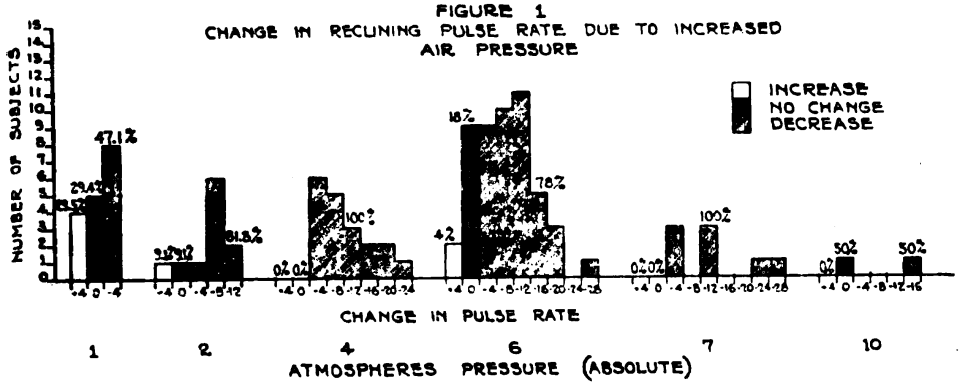
Each increased air pressure test was preceded by a corresponding test on the same individuals at atmospheric pressure. These several atmospheric tests are indicated in table 1 as 1b, 1c, 1d, 1e, and 1f. There was also, as shown in this table, a separate control series of tests (1 and 1a) run at atmospheric pressure and then repeated at atmospheric pressure after a time interval similar to that required to reach the pressure used in the other runs.

TABLE 1.—*Average pulse, blood pressure, and pulse pressure (reclining, standing, and exercise) at atmospheric and increased air pressures*

Atmospheres absolute and differences	Reclining pulse rate	Standing pulse rate	Exercise pulse rate	Reclining systolic blood pressure	Reclining pulse pressure	Standing systolic blood pressure	Standing pulse pressure
1	72.4	83.7	98.8	113.8	48.2	119.3	38.3
1a	71.5	83.7	98.8	116.9	50.0	118.8	36.2
Difference	−0.9	±0.0	±0.0	+3.1	+1.8	−0.5	−2.1
1b	71.3	83.3	99.6	109.8	38.0	115.4	32.5
2	64.7	76.3	92.4	106.2	32.7	113.2	26.9
Difference	−6.6	−7.0	−7.2	−3.6	−5.3	−2.2	−5.6
1c	77.0	91.2	107.1	115.9	50.0	121.3	41.8
4	66.7	79.0	94.7	108.9	41.6	113.9	32.5
Difference	−10.3	−12.2	−12.4	−7.0	−8.4	−7.4	−9.3
1d	77.2	89.0	105.9	114.2	42.7	120.2	35.7
6	69.0	80.0	94.6	107.2	34.9	113.7	26.8
Difference	−8.2	−9.0	−11.3	−7.0	−7.8	−6.5	−8.9
1e	77.0	91.5	107.0	116.2	50.5	122.7	37.5
7	64.5	80.0	95.5	108.5	36.7	112.0	24.0
Difference	−12.5	−11.5	−11.5	−7.7	−13.8	−10.7	−13.5
1f	86.0	100.0	122.0	120.0	66.0	125.0	37.0
10	78.0	84.0	101.0	109.0	30.0	126.0	30.0
Difference	−8.0	−16.0	−18.0	−11.0	−36.0	+1.0	−7.0

The individual range in pulse rate for all pressures above atmospheric is from 52 to 84. The individual change in pulse rate from atmospheric pressure to each increased pressure studied is graphically shown in figure 1. It will be noted in this figure that at 4 and 7 atmospheres 100 percent of the individuals showed a decrease in pulse rate while at 2 atmospheres 81.8 percent showed a decrease, and at 6 atmospheres 78 percent showed a decrease in pulse rate

FIGURE 1
CHANGE IN RECUMBENT PULSE RATE DUE TO INCREASED AIR PRESSURE



during recumbency. At a pressure of 10 atmospheres one subject showed no change while the other decreased 16 beats.

Pulse rate in standing posture.—At atmospheric pressure the pulse rate taken at the end of 2 minutes standing ranged between 64 and 112 while the average for all cases was 88.9. The average for the 200-group of Schneider and Truesdell (1922) was 90.11 ± 0.55.

Under increased air pressure the standing pulse ranged from 56 to 100 for all pressures and the average was 79.9 beats per minute.

FIGURE 2
CHANGE IN STANDING PULSE RATE DUE TO INCREASED AIR PRESSURE

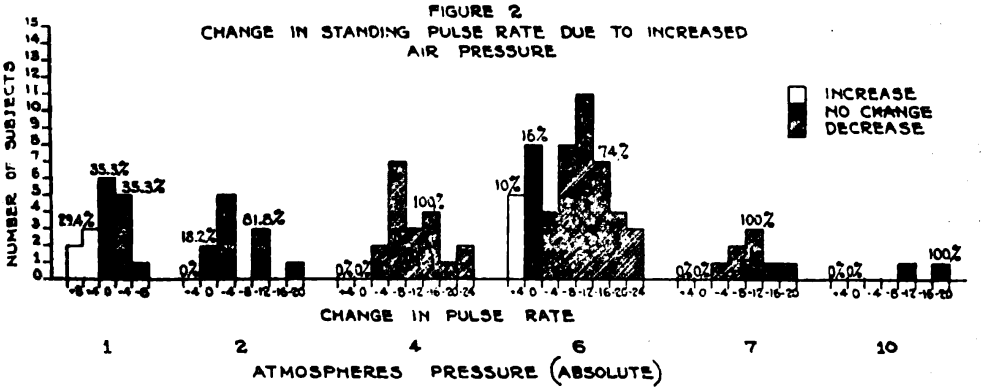


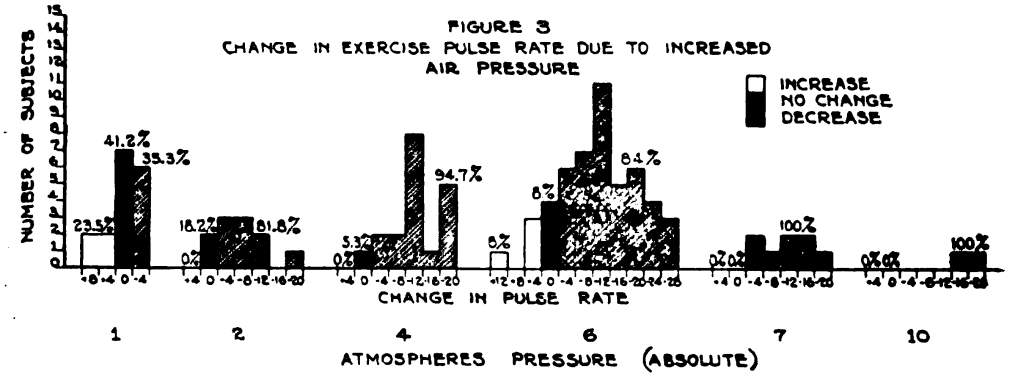
Table 1 shows the average pulse rate and the average decrease in pulse rate per individual per minute at the various increased air pressures in this study. The control run shows a perfect check for the standing pulse rate. These individual checks as well as the individual changes in standing pulse are shown graphically in figure 2. Here again the predominant change under pressure is a de-

crease in pulse rate with 100 percent of the cases showing a decrease at 4, 7, and 10 atmospheres, 81.7 percent showing a decrease at 2 atmospheres, and 74 percent showing a decrease at 6 atmospheres.

Pulse rate after standard exercise.—The pulse taken immediately after exercise at atmospheric pressure ranged from 72 to 136. The average for all cases was 105.6 beats per minute. Schneider and Truesdell (1922) found an average of 102.20±0.19.

Under increased air pressure the exercise pulse ranged from 68 to 120, with the average for all cases being 96.2 beats per minute. The average pulse rate and the average decrease for each pressure is shown in table 1. Again the control runs show a perfect check.

The individual change in exercise pulse rate from atmospheric pressure to the various increased air pressures is shown in figure 3. Here it is shown that at 10 and 7 atmospheres 100 percent of the cases showed a decrease, at 4 atmospheres 94.7 percent showed a



decrease, at 6 atmospheres 84 percent showed a decrease, and at 2 atmospheres 81.8 percent showed a decrease.

Systolic pressure reclining.—The systolic pressure taken following a 5-minute rest period at atmospheric pressure ranged from 96 to 154 mm Hg. The average for all cases at atmospheric pressure was 115.2 mm Hg. Again the average agrees closely with the work of Schneider and Truesdell (1922), in which their mean systolic pressure was 117.6 ±0.53.

Under increased air pressure the range was from 84 to 134 mm Hg, and the average for all cases at increased pressure was 107.96 mm Hg. Not only is there a drop in the average pressure, as noted in table 1, but an analysis of the individual variation shows the following: 54.5 percent of the individuals showed a drop in systolic pressure at 2 atmospheres, 73.7 percent a drop at 4 atmospheres, 74 percent a drop at 6 atmospheres, 75 percent a drop at 7 atmospheres, and 50 percent a drop at 10 atmospheres (2 cases).

Systolic pressure standing.—The range in the standing systolic pressure at atmospheric pressure was from 98 to 154 mm Hg and

the average for all cases was 120.38 mm Hg. This average is almost identical with the 120.26 ±0.18 mm as reported by Schneider and Truesdell (1922) for their large group of 2,000 cases.

Under increased air pressure the range was from 98 to 154 mm Hg and the average 115.76 mm Hg. Not only is this general average for all pressures under the atmospheric average, but reference to table 1 will show that the average for each individual pressure is also less than its atmospheric control.

The individual variation from atmospheric pressure to increased air pressure shows that at 2 atmospheres 45.5 percent of the cases showed a fall in systolic pressure, at 4 atmospheres 78.9 percent showed a fall, at 6 atmospheres 68 percent, at 7 atmospheres 87.5 percent, and at 10 atmospheres 50 percent (2 cases).

Postural change in systolic pressure.—The difference between reclining and standing systolic pressure is usually considered as of importance in affording evidence of the integrity of the vasomotor mechanism, in that there should be a rise in the systolic pressure due to the splanchnic compensation which offsets the increased hydrostatic effect of gravity during standing. At atmospheric pressure 77.42 percent of the cases showed a rise in systolic pressure, 7.25 percent showed no change, and 15.33 percent showed a fall in systolic pressure. The figures of Schneider and Truesdell (1922) for their large group were 55.55 percent increase, 17.95 percent no change, and 26.5 percent decrease.

Under increased air pressure 75.55 percent of the cases showed a rise in systolic pressure, 10 percent showed no change, and 14.45 percent showed a fall in systolic pressure.

Pulse pressure reclining.—The range in pulse pressure at atmospheric pressure was from 22 to 96 mm Hg. The average pulse pressure for all cases was 49.31 mm Hg. The average reclining pulse pressure reported by Schneider and Truesdell (1922) was 47.41±0.14.

Under increased air pressure the range in reclining pulse pressure was from 14 to 64 mm Hg. The average for all cases was 34.4 mm Hg. Table 1 gives the group average for each different pressure used, and the difference between this and its atmospheric control. Not only is the range and entire average lower but the group average for every pressure is seen to be lower than at its corresponding atmospheric control.

The individual variation shows a decrease in the pulse pressure in 72.7 percent of the cases at 2 atmospheres, 89.4 percent at 4 atmospheres, 77.7 percent at 6 atmospheres, 87.5 percent at 7 atmospheres, and 100 percent at 10 atmospheres. The greatest individual decrease in pulse pressure was 50 mm Hg (64 to 14) at 10 atmospheres.

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Pulse pressure standing.—The range in the standing pulse pressure at atmospheric pressure was from 16 to 66 mm Hg and the average for all cases was 37.1 mm Hg. The average reported by Schneider and Truesdell (1922) was 41.58±0.17 mm Hg.

Under pressure the range was from 8 to 52 mm Hg. This range is not only lower than atmospheric pressure, but is lower than the 14 to 64 mm reclining under pressure. The average standing pulse pressure under increased air pressure is 28.04 mm Hg, while the average reclining pulse pressure is 34.4 mm Hg. Table 1 gives the average data for each pressure used.

The individual changes in standing pulse pressure from atmospheric to increased air pressure show that at 2 atmospheres 54.5 percent of the cases showed a lessening in pulse pressure, at 4 atmospheres 73.7 percent, at 6 atmospheres 86.1 percent, at 7 atmospheres 87.5 percent, and at 10 atmospheres 50 (12 cases).

TABLE 2.—*Difference in Schneider indices taken at increased air pressures from those taken at atmospheric pressure*

Pressure atmospheres absolute	Total number of subjects	Increase in index		Decrease in index		No change in index		Total changes in Schneider units			Average change in Schneider units per subject
		Number of subjects	Percent of subjects	Number of subjects	Percent of subjects	Number of subjects	Percent of subjects	Negative	Positive	Difference	
1	17	5	30	8	46	4	23	16	6	−10	−0.588
2	11	9	82	1	9	1	9	1	24	+23	+2.09
4	18	17	89.4	1	5.3	1	5.3	3	62	+59	+3.105
6	50	36	72	8	16	6	12	23	140	+117	+2.34
7	8	8	100	0	0	0	0	0	36	+36	+4.5
10	2	2	100	0	0	0	0	0	10	+10	+5.0

Schneider indices.—From the pulse and blood pressure records a Schneider score was figured for each subject, according to the table as outlined in the Manual of the Medical Department of the United States Navy, both at atmospheric pressure and for each corresponding increased air pressure. Table 2 gives a complete summary of all these Schneider scores. The control series at atmospheric pressure repeated at atmospheric pressure shows 30 percent of the 17 cases decreasing, 46 percent showing no change, and 23 percent increasing in their Schneider score. This gives an average change in Schneider units per individual of minus 0.588. Each increased air pressure series when compared to its paired atmospheric pressure run shows a marked increase in the Schneider indices until at 10 atmospheres there is a plus 5 average unit change per individual. At both 8 and 10 atmospheres 100 percent of the individuals showed an increase in their Schneider score.

TABLE 3.—*Subjects having a Schneider index at atmospheric pressure of less than 10*

Subjects	Atmospheres																	
	1	1a	Difference	1b	2	Difference	1c	4	Difference	1d	6	Difference	1e	7	Difference	1f	10	Difference
J. H. M.	----	----	----	----	----	----	----	----	----	1	10	9	----	----	----	0	8	8
H.— T.	----	----	----	----	----	----	----	----	----	4	9	5	----	----	----	----	----	----
J. A. M.	----	----	----	----	----	----	----	----	----	4	9	5	----	----	----	----	----	----
T. D. F.	----	----	----	----	----	----	−1	7	8	4	13	9	----	----	----	----	----	----
R. E.	----	----	----	----	----	----	----	----	----	6	13	7	----	----	----	----	----	----
J.—M.	----	----	----	8	12	4	----	----	----	6	9	3	----	----	----	----	----	----
J. O. C.	----	----	----	----	----	----	----	----	----	6	12	6	----	----	----	----	----	----
H. G. S.	----	----	----	----	----	----	----	----	----	7	12	5	----	----	----	----	----	----
T. D. F.	----	----	----	----	----	----	----	----	----	7	9	2	----	----	----	----	----	----
J. H. M.	9	10	1	6	10	4	----	----	----	7	12	5	----	----	----	----	----	----
J. M. O.	8	9	1	----	----	----	----	----	----	7	12	5	----	----	----	----	----	----
J. E. G.	----	----	----	----	----	----	----	----	----	8	11	3	----	----	----	----	----	----
W. W. W.	----	----	----	----	----	----	7	11	4	8	12	4	----	----	----	----	----	----
J. A. H.	----	----	----	----	----	----	----	----	----	8	15	7	----	----	----	----	----	----
T. E. G.	----	----	----	----	----	----	----	----	----	8	13	5	----	----	----	----	----	----
L. E. B.	----	----	----	----	----	----	----	----	----	8	14	6	8	15	7	----	----	----
T. D. F.	----	----	----	----	----	----	----	----	----	9	14	5	----	----	----	----	----	----
J. E. G.	----	----	----	9	13	4	----	----	----	9	14	5	----	----	----	----	----	----
J. W. T.	----	----	----	----	----	----	9	14	5	9	13	4	----	----	----	----	----	----
F. A. M.	9	9	0	----	----	----	8	9	1	----	----	----	----	----	----	----	----	----
J. T. K.	----	----	----	----	----	----	4	6	2	----	----	----	----	----	----	----	----	----
J. L. B.	----	----	----	----	----	----	9	12	3	----	----	----	----	----	----	----	----	----
W. A. G.	----	----	----	----	----	----	9	16	7	----	----	----	----	----	----	----	----	----
G. —. T.	----	----	----	----	----	----	----	----	----	----	----	----	7	10	3	----	----	----
J. B. C.	----	----	----	----	----	----	----	----	----	----	----	----	7	13	6	----	----	----
H. H. H.	----	----	----	----	----	----	----	----	----	----	----	----	8	11	3	----	----	----
C. L. C.	----	----	----	----	----	----	----	----	----	----	----	----	9	13	4	----	----	----
J. M. O.	4	4	0	----	----	----	0	12	12	----	----	----	2	11	9	----	----	----
Averages	+0.5			+4.0			+5.25			+5.26			+5.33			+8.0		

Every individual whose Schneider index or score was below 10 units at atmospheric pressure showed an increase or improved index when taken under pressure. All of these low Schneider indices are shown in table 3. The greatest increase or improvement in any individual was 12 and the least was 1 unit. There were 3 individuals with an increase of 9 units, 2 with an increase of 8, 4 with an increase of 6, 9 with an increase of 5, 7 with an increase of 4, 5 with an increase of 3, and 2 with an increase of 2 units. The average increase in Schneider units per individual is progressively greater as the pressure increases.

Cardiac output.—Cardiac output was calculated according to the formula of Furst and Soëtbeer (1907). Using the data for subjects exposed to 6 atmospheres (the largest group) we find that at atmospheric pressure the reclining average cardiac output per individual was 3.79 liters per minute. When repeated at 6 atmospheres the same subjects have an average cardiac output per individual of 2.89 liters per minute. The average decrease in cardiac output is thus almost 1 liter per minute. Of the 47 cases, 39 showed a decrease while 8 showed a slight increase. These results are comparable with those reported by Hawkins, Shilling, and Hansen (1934).

Although all these cardiac outputs were based on theoretical calculations they appear to show a possible significance worthy of careful check by quantitative analyses. Such quantitative experiments are in progress at the present time and will be reported in a subsequent paper.

Relation of increased oxygen tension to pulse and blood pressure.—As the air is compressed the tension of oxygen is increased proportionately. That this may be a factor in slowing the pulse and lowering the blood pressure has been contended by several authors. Simmon (1925) describes cases of hypertension in which a lowering has been noted which persisted for some hours. Steinhaus, Jenkins, and Lum (1930), after 1,289 tests on 3 dogs breathing oxygen at an increased tension, concluded that there was no relation between breathing oxygen and the fluctuation of the pulse. Parkinson (1912) showed that breathing commercially pure oxygen caused a slowing of the pulse. Dautrebande and Haldane (1921) say that when pure oxygen is breathed under increased air pressure of two atmospheres there is a slowing of the pulse. Hill and Phillips (1932) found that a diver, when performing a piece of work at a depth of 300 feet, had an increased pulse rate of only 5 beats per minute. The small increase is attributed to the influence of oxygen, for the diver was breathing air with oxygen the equivalent to 2 atmospheres.

SUMMARY

A study of the pulse, blood pressure, and pulse pressure reclining, standing, and after exercise both at atmospheric pressure and under increased air pressure has been made.

Reclining pulse rate.—The average for all cases at atmospheric pressure was 76 beats per minute. The average for all cases under increased air pressure was 66 beats per minute.

Standing pulse rate.—The average for atmospheric pressure was 88 beats per minute, while under increased air pressure this dropped to 79 beats per minute.

Exercise pulse rate.—The atmospheric pressure average was 105 beats per minute, while under increased pressure this dropped to 96 beats per minute.

Reclining systolic pressure.—The average at atmospheric pressure was 115 mm Hg, which fell to an average of 107 mm Hg under increased air pressure.

Standing systolic pressure.—The average at atmospheric pressure was 120 mm Hg, while under increased air pressure the average was 115 mm Hg.

Reclining pulse pressure.—The average at atmospheric pressure was 49 mm Hg, while at increased air pressure the average was 34 mm Hg.

Standing pulse pressure.—The average at atmospheric pressure was 37 mm Hg, while at increased air pressure the average was 28 mm Hg.

CONCLUSIONS

1. The Schneider index is increased by exposure to increased air pressure.
2. Pulse rate, blood pressure, and pulse pressure are decreased during exposure to increased air pressure.
3. Cardiac output, calculated by the formula of Furst and Soëtbeer (1907), is decreased by exposure to increased air pressure.

REFERENCES

Dautrebande and Haldane. Journ. Physiol. lv, 296, 1921.
Furst, T., and Soëtbeer, F. Deut. Arch. für klin. Med., 90, 190, 1907.
Hawkins, J. A., Schilling, C. W., and Hansen, R. A. In press.
Heller, Mager, and v. Schrotter. Luftdruck Erkrankungen. Wein. 1900.
Hill, L., and Phillips, A. E. Journ. Royal Nav. Med. Serv. xviii 3, 1932.
Parkinson, J. Journ. Physiol. xliv, 54, 1912.
Schneider, E. C., and Truesdell, D. Am. Journ. Physiol. lxi, 3, 1922.
Simmon, H. Physiol. Abs. 10, 581, 1925–26.
Steinhaus, A. H., Jenkins, and Lum. Am. Journ. Physiol. 92, 436, 1930.

THE HAZARD OF CAISSON DISEASE IN INDIVIDUAL SUBMARINE ESCAPE[1]

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INTRODUCTION

Following the disasters of the U. S. S. *S–51* and U. S. S. *S–4* an individual submarine escape appliance was developed and adopted. This is popularly known as the "lung", and for the purpose of brevity this term will be used throughout this paper. Its construction and use has been described by Mankin (1). Since its adoption by the United States Navy all submarine personnel have been trained annually in its use. This training is conducted in the "lung" training tanks at the submarine bases at New London, Conn., and Pearl Harbor, T. H., or with a bell at other submarine concentration points. The U. S. S. *S–4*, salvaged and converted into an experimental vessel, made a final training cruise in 1932, at which time

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most of the men in the submarine service made training escapes from the motor room.

The method of escape from a sunken submarine may be briefly outlined as follows: (1) The "lungs" are distributed individually and tested to see that they are in proper working condition. (2) The "hatch skirt" is then placed in position under the escape hatch so that an air pocket may be maintained. (The newer type submarines have the "hatch skirt" permanently attached). (3) The hatch is undogged so that it will spring open when the external and internal pressures become equal. (4) Flooding the compartment with sea water is now started and continued as rapidly as possible until the internal pressure equals the weight of water over the hatch, at which time the hatch opens and water pours in and air escapes until the level of the water in the compartment reaches the lower edge of the "skirt", air above the water level in the compartment thus being trapped. (5) A buoy carrying a line is released and when it reaches the surface, the end of the line in the submarine is secured. (6) The "lung" is now charged with oxygen, the individual ducks under the edge of the "skirt" and grasping the line slides slowly up through the hatch to the surface. It is contemplated that a submarine salvage vessel will be at hand, whenever possible, to rescue survivors following this escape.

It is evident that a hazard of caisson disease is incurred under this condition of continuous ascent. The present study was undertaken to determine how long an individual could remain at a given depth, as in a submarine during preparation, flooding, and escape described above, and then make a continuous ascent to the surface, at the rate of 50 feet per minute, without developing caisson disease.

METHOD

The diving tank which has been described by Hawkins, Shilling, and Hansen (2), was used for the experiments which were conducted at the Experimental Diving Unit, Department of Construction and Repair, Navy Yard, Washington, D. C., under the direction of Lieutenants Bibby, Clark, and Hansen, former officers in charge. Subjects were exposed in this diving tank to pressure equal to various depths of sea water. During the exposure time they breathed in the air-pocket of the tank and exercised by swimming and diving in the water. The exposure time is the actual time at the depth indicated, plus half the time required to reach this depth. Immediately prior to the end of the exposure, the subject put on the "lung", submerged completely in the water and breathed into the "lung" for 2 minutes after which ascent was made at the rate of 50 feet

per minute. In certain of the experiments the "lung" was charged with oxygen while in other tests compressed air was employed.

The ascent or escape was simulated by the reduction of the air pressure and this constituted the entire decompression received by the subjects. Each subject made daily dives for 5 days a week until the series was completed. Usually eight subjects were used for each depth and time of exposure, this being called a run. In each series the depth was kept constant while the time of exposure was increased by increments of one-half or 1 minute for each succeeding run.

The end-point in each series was the production of caisson disease of severe enough nature to necessitate terminating the series. A recompression chamber was available for the purpose of treatment. These cases of experimental caisson disease have been reported by Shilling, Hawkins, Polak, and Hansen (3).

EXPERIMENTAL RESULTS

A total of 2,143 simulated dives were made over a period of 3 years. These were divided into 12 series and were conducted at depths of 100, 150, 167, 185, and 200 feet, respectively. The data obtained are presented in table 1.

Four series, i. e., 2, 7, 9, and 11 were conducted at a depth of 100 feet with a total of 1,231 individual exposures. The first case of caisson disease occurred following an exposure of 37½ minutes in series 7 breathing oxygen but in series 11 when breathing air in place of oxygen the first case of caisson disease did not occur until an exposure of 43 minutes. It therefore appears safe under these conditions to be exposed to a pressure corresponding to 100 feet of sea water for 37 minutes and then escape to the surface, by continuous ascent at the rate of 50 feet per minute, with the "lung" charged with either air or oxygen. The maximum exposure at the 100-foot depth was 51½ minutes.

Five series were conducted at a depth of 150 feet. In series 3, 8, 10, and 15 the "lung" was charged with oxygen for the escape and in series 16 the "lung" was charged with air. The first case of caisson disease occurred in series 10 and followed an exposure of 18½ minutes. In series 16 in which the "lung" was charged with air the first case of caisson disease occurred following an exposure of 26½ minutes. The longest exposure was 28 minutes in series 15. Considering the first case of caisson disease as the controlling factor, it appears safe under these conditions to remain at 150 feet for 18 minutes and then escape to the surface, by continuous ascent at the rate of 50 feet per minute, with the "lung" charged with either air or oxygen.

TABLE 1.—*Experimental results*

Series	Depth, gage	Number of runs	Individual exposures	Initial exposure time	Increase of exposure time	Final exposure time	Caisson disease			
							First case exposure time	Number of cases	Number of exposures—	
									Before first case	After and including first case
				Minutes	*Minutes*	*Minutes*	*Minutes*			
2	100	75	600	8½	1	31½	-------	0	600	0
7	100	18	75	14½	1	39½	37½	3	56	19
9	100	50	127	32½	1	51½	40½	5	46	81
11	100	59	429	3	1	48	43	5	408	21
10	150	28	70	18½	½	24½	18½	5	4	66
3	150	34	117	10½	1	22½	19½	8	80	37
8	150	18	71	9½	1	21½	20½	2	56	15
16	150	18	54	18	½	27	26½	2	42	12
15	150	74	214	10	½	28	28	5	206	8
4	167	38	141	6½	1	22½	17½	6	95	46
5	185	9	72	6½	1	15½	14½	3	56	16
6	200	23	173	7½	½	16	13½	2	133	40
Total	-------	-------	2,143	-------	-------	-------	-------	46	1,782	361

Only one series each was run at depths of 167, 185, and 200 feet. The first case of caisson disease occurred following exposures of 17½, 14½, and 13½ minutes, respectively. No runs were made breathing air. The preliminary conclusion is drawn that the safe exposure is 17 minutes at 167, 14 minutes at 185, and 13 minutes at 200 feet. However, the experimental data recorded at these depths are considerably less than at 100 and 150 feet.

In the open sea successful "lung" escapes were made from a bell at depths of 100 and 150 feet. At 100 feet 7 individual escapes were made following an exposure of 17 minutes, 6 following an exposure of 22 minutes, 7 following an exposure of 27 minutes, and 7 following an exposure of 32 minutes. In all, there were 27 individual exposures followed by continuous ascent requiring an average of 1 minute. No caisson disease was encountered.

At 150 feet there was 1 escape following an exposure of 12 minutes, 6 following an exposure of 14 minutes, 4 following an exposure of 16 minutes, and 2 following an exposure of 20 minutes; making a total of 13 individual exposures followed by continuous ascent averaging 1.7 minutes per individual. The time of ascent was the entire decompression received, and no caisson disease was encountered.

These dives in the open sea tend to confirm the results obtained in the tank by showing that it is safe to remain at 100 feet for at least 32 minutes and at 150 feet for 20 minutes, and then come to the surface at a rate of 50 feet per minute.

DISCUSSION

From the experimental work here reported, it has been shown apparently safe to remain at 100 feet for 37 minutes, 150 feet for 18 minutes, 167 feet for 17 minutes, 185 feet for 14 minutes, and 200 feet for 13 minutes. In actual "lung" escapes from a bell in the open sea an exposure of 32 minutes at 100 feet and 20 minutes at 150 feet was found safe. In applying these figures to the entire service, the objection may be raised that because of the small number of experimental subjects involved, one might find individuals who would develop caisson disease. While the number of experimental subjects on duty at the Experimental Diving Unit never exceeded 10, still, during the 3 years, a number of changes in personnel occurred, so that in all, 22 men were included in the course of the experiments. Furthermore, when it is remembered that 2,143 dives were made, the data seem sufficiently established to report to the service for information in case of a disaster. It is believed, however, that the results should be confirmed by actual ascents with a large number of subjects.

It should also be remembered that the first case of caisson disease, regardless of the degree of severity, was considered the end-point, despite the fact that for all series a total of 361 dives were made after the first case of caisson disease developed. It is of interest that one man made a total of 120 escapes at the various depths and never developed caisson disease. Eight others made a combined total of 666 escapes with only one attack in each individual.

Granting the accuracy and safety of the figures for the various depths, the time available at the greater depths is, of course, inadequate to flood a submarine compartment and assure the exit of all of the crew. Much discussion has centered around the method of decompression of the men escaping from a sunken submarine. At first, it was thought advisable to have numbered markers attached to the ascending line, requiring a certain number of breaths to be taken at each stop. But this plan slows to such an extent the escape that the men left behind have a much longer exposure and thus it was abandoned in favor of a continuous ascent at the rate of 50 feet per minute. Even this continuous ascent will not give all of the personnel an equal chance, for at least 1 minute is required to properly check each man and start him up the line, and proper ascent from a depth of 100 feet would require at least 2 minutes.

SUMMARY

Two thousand one hundred and forty-three simulated escapes with the "lung" were made at depths of 100, 150, 167, 185, and 200 feet.

It was found safe under these experimental conditions to remain 37 minutes at a simulated depth of 100 feet, 18 minutes at 150 feet,

17 minutes at 167 feet, 14 minutes at 185 feet, and 13 minutes at 200 feet, the subjects coming to the surface at the rate of 50 feet per minute.

It was also found that air could be safely used in place of oxygen to charge the "lung" in making escapes to the surface from depths of 100 and 150 feet by continuous ascent at the rate of 50 feet per minute. Should stops be required on the ascending line from any cause whatsoever the length of time which such stops could be made before developing an oxygen deficiency in the "lung", when charged with air, is still problematical.

Actual ascents were made from 100 feet after an exposure of 32 minutes and from 150 feet after an exposure of 20 minutes, but it is recommended that actual ascents with the "lung" be conducted in sea water with a larger number of subjects in order to confirm the above data.

BIBLIOGRAPHY

(1) Mankin, G. H. "Individual Submarine Escape", U. S. Nav. Med. Bull., 28:18–28; 1930.

(2) Hawkins, J. A. Shilling, C. W., and Hansen, R. A. "Suggested Change in Calculating Decompression Tables for Divers", U. S. Nav. Med. Bull., 33: 327–338; 1935.

(3) Shilling, C. W. Hawkins, J. A., Polak, I. B., and Hansen, R. A. "Caisson Disease and Its Relation to Tissue Saturation with Nitrogen", U. S. Nav. Med. Bull., 33: 4; 1935.

A REVIEW OF THE RELATION BETWEEN SINUSITIS AND PULMONARY DISEASE

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In conducting a survey of recent literature on this subject an outstanding feature noted was the variability of findings and conclusions of various essayists. This variability was most marked on the subject of asthma and sinusitis. The reasons for these paradoxical findings are probably numerous but some of the more apparent sources of error are those common to all clinical research problems. Among them are (*a*) absence of controls in reporting series of cases, (*b*) differences in criteria of diagnosis and standards of cure, (*c*) generalization from too few cases, (*d*) observation over too limited a period of time, (*e*) failure to properly evaluate other factors such as allergy which may be simultaneously present in the patient under observation and lastly the extremely variable personal equation.

The importance of control experiments and criteria of diagnosis can be appreciated when the incidence of sinusitis in the general population is considered. Only a few surveys have been made but

these vary from a high of 72 percent as determined by X-ray evidence to a low of 6 percent determined by routine clinical examination of a group of bank employees. Among 93,000 admissions to three large general hospitals in Rochester, N. Y., 1.2 percent showed enough evidence of sinusitis to warrant a diagnosis on the hospital records. (1) Kern and Schenck quote statistics to the effect that 26 percent of apparently normal controls showed clinically detectable evidence of sinusitis. (2) The former states: "The relation between sinus disease and bronchial asthma is a subject on which there has been a great deal of loose thinking, and, what is worse, a great deal of loose writing." He mentions an instance where a writer quoted him as saying that 85 percent of old asthmatics gave radiographic evidence of sinus abnormality but failed to mention that in a control series of 50 supposedly normal persons a 78 percent incidence was also noted. This practice of citing only those figures which favor a particular theory is apparent throughout the literature.

Such wide average ranges as 6 to 72 percent can be accounted for. The X-ray diagnosis of sinusitis depends upon the finding of thickened membrane or cloudiness due to retained secretions. A thickened membrane may be radiographically demonstrable long after all clinical symptoms have disappeared and the infection cleared. Since there is a catarrhal sinusitis with every head cold it is not surprising to find a high incidence of slight persistent changes in the sinus mucosa.

On the other hand pathological secretions in sinusitis are frequently intermittent and may vary from mucoid to purulent on different days or may be absent at different times in the same day. Clinically, therefore, sinusitis may be easily overlooked. Other clinical criteria of sinusitis are not reliable. Congestion of the nasal mucosa may be due to the common cold and transillumination is notoriously unreliable except in unilateral and fairly marked frontal and maxillary sinusitis. Often pathological secretions are easily demonstrable after shrinkage when prior examination failed to show them. The true incidence of active sinusitis is probably between these extremes depending on seasonal and climatic variations and has been placed at 25 to 50 percent by several observers.

Schenck states that for an average of 4 months following a Caldwell-Luc operation 80 percent of a group of 35 asthmatics were symptom free but in no case was there permanent relief. Statistics in groups of asthmatic patients treated by both radical sinus surgery and conservative nasal applications are uniformly depressing after a 5-year period (3) thus emphasizing the value of prolonged observation.

In spite of such difficulties as previously mentioned in evaluating evidence, certain facts and experiences seem to warrant definite conclusions and suggest rational theories which I will now try to set forth.

That patients with chronic bronchitis, bronchiectasis, and asthma very frequently have associated sinusitis is a universally admitted fact. Although most clinical observations and experimental evidence indicate that the sinusitis is primary in these cases, there is also proof that in certain conditions sinusitis can be the result of chest pathology. Patients with post-tonsillectomy pulmonary abscess, in which previous clinical and X-ray studies of the sinuses were negative, developed a purulent pan sinusitis within a short time (weeks) after the onset of chest symptoms. This was undoubtedly due to spraying of the nose with infectious sputum organisms during cough paroxysms (2). The high incidence of sinusitis in chronic pulmonary abscess and chronic ulcerative tuberculosis with cavitation and mixed infection is probably due to the same cause. This conclusion is further strengthened by failure to find frequent sinusitis in fibroid or miliary tuberculosis (4). The high incidence of sinusitis in bronchiectasis (80 percent by clinical evidence and 100 percent by X-ray findings (2)) may also be due to this mode of infection. I hope to show later that sinusitis is frequently a cause of bronchiectasis but in bronchiectasis due to lung abscess or aspirated foreign bodies, infection of the sinuses is almost sure to follow.

Infection in the upper respiratory tract can be and frequently is the source of pulmonary disease. We are all familiar with a common course of the acute head cold. After a few days, usually about the fourth, the pharynx and/or larynx becomes affected and, finally, a mild form of acute bronchitis is frequently seen. Experience has shown that when the head cold has completely cleared the pharyngitis, laryngitis, and cough quickly disappear. If the nasal infection becomes localized in the sinuses with considerable post nasal discharge cough frequently persists. Often the most distressing symptom of a sinusitis characterized by profuse discharge is cough. In a series of 295 patients with sinus disease almost 20 percent complained of cough, and in nearly 7 percent, cough was the only symptom (5).

Chest examination in these cases shows a few rales, usually in the lower lobes and X-ray pictures show greatly increased trunk shadows near the hilus of the affected lobe with perhaps even some central peribronchial consolidation. Cure of the sinusitis is followed by prompt clearing of the pulmonary lesions both clinically and radiographically (6). Some clinicians have termed this syndrome "bronchosinusitis" or "sinus lung."

That nasal and paranasal secretions reach the lungs has been demonstrated experimentally. Theoretically four routes are possible, viz, direct extension, inhalation, lymph stream, and blood stream extension. Direct extension is difficult to prove but is suggested by a course of the acute head cold which progressively extends downward to pharynx, larynx, trachea, and bronchi, involving each in succession as previously mentioned. Mullin and Ryder (5) dropped India ink into the nostrils of rabbits through a catheter without disturbing their respiration. At post-mortem examination 24 hours later the bronchial lymph nodes and upper lobes of the lungs were found to be black. Miscroscopic examination showed carbon in the bronchioles and alveoli. If these animals were killed shortly after nasal instillation of ink, this carbon was found in the trachea and larger bronchi, indicating that it entered the lungs by aspiration. When emulsions of virulent tubercle bacilli were substituted for ink, pulmonary tuberculosis was produced in the same areas, i. e., bronchial lymph nodes and upper lobes of the lungs. Attention is invited to the dependent position of the upper lobes in rabbits, showing the effect of gravity. Myerson (7) bronchoscoped 100 children immediately after tonsillectomy under ether anesthesia. Seventy-nine showed blood in the bronchial tree. He also found that the normal pulmonary tract rids itself of this blood in from 12 to 15 minutes. Quinn dropped iodized oil into the nasal fossae of 11 sleeping persons by means of a catheter and found that aspiration occurred in 5. In two cases this happened repeatedly. McLaurin (8) showed that this also occurred in the waking state by injecting lipiodol into sinuses by the displacement method of Proetz. In several cases a small amount of oil was found in the bronchial tree 24 hours later. This indicates that there may be individual predisposition to aspiration. In some sinus patients imperfect approximation of the cords due to weakness of the internal tensor muscles of the larynx can be demonstrated by indirect laryngoscopy (9). Chronic inflammation of the pharynx and larynx may possibly be an etiological factor.

The sinusitis and bronchitis patient typically coughs most in the morning. This is due to accumulation of secretions in the trachea and bronchi because of depressed cough reflex. It is also probable that the swallowing reflex in sleep is sufficiently depressed or imperfectly executed in many individuals to permit entry of post nasal discharge into the larynx. The mechanism would probably be inadequate elevation of the larynx from the postpharyngeal wall with imperfect closure of the cords during swallowing. According to Starling the arytenoids rest against the posterior pharyngeal wall except during swallowing, when normally the larynx is elevated

and drawn forward together with a complexity of pharyngeal movements. This theory would explain the high frequency of aspiration during general anesthesia when swallowing is abolished, the lesser frequency during sleep when swallowing may be depressed or imperfectly executed, and the least frequency in the waking state. It would also explain why aspiration and the sinus lung are not universally seen in all cases of purulent sinusitis.

Lymphatic absorption has been demonstrated by injecting suspensions of carbon in the frontal and maxillary sinuses of rabbits. These particles were traced to the submaxillary and internal jugular lymph nodes, the latter corresponding to the retropharyngeal glands in man. Thence they traveled via the lymphatic ducts to the great veins and right heart. From here they were sent to the lungs, where they became lodged (5). G. E. Pfahler (23) reports a case in which lipiodol was injected into both antra for diagnostic purposes. A hyperplastic sinusitis was demonstrated in the left antrum. About 10 weeks later the left sinus was empty and only a small globule of oil remained in the right antrum. Numerous small scattered deposits of lipiodol 2 to 5 mm in diameter were seen in the lymph spaces extending from the zygoma to the clavicle on each side but scattered also across the submental and sublingual regions. On the left side these deposits were traced to the thoracic duct and on the right to the right lymphatic duct at the junction of the jugular and subclavian veins. Another X-ray, taken over a year after the original lipiodol injection, still showed the same distribution with only slight diminution in total deposits. Although this is the only case reported, the author suggests that the lipiodol distribution probably indicates the normal lymphatic drainage of the antra. Childrey and Essex from experimental studies concluded that the normal sinus mucosa is very resistant to absorption. Wood and others hold that sinus discharges set up secondary inflammation in the nasopharynx which acts as a secondary focus from which absorption takes place. From the standpoint of lympyhatic absorption the nasopharyngeal mucosa may be considered as continuous with that of the sinuses.

It has been suggested that the lymphatic route is more important in hyperplastic sinus infections with little or no secretion while inhalation is the most probable route in purulent sinusitis. Sluder showed that the absence of secretion in sinusitis is often more apparent than real. The secretion may be thin, mucoid, and scanty. If spread in a thin layer over the nasal mucosa this type of secretion is very difficult to see.

Although many essayists refer to the lymphatic route to the lungs as clinically established there are many facts which discredit it. For instance, cervical adenopathy which occurs so readily in diseases of the nasopharynx is rarely seen in sinusitis. As previously

mentioned the nasopharynx may be considered continuous with the sinus mucosa from the standpoint of lymphatic drainage. Another argument against the lymphatic route are the X-ray findings in the sinus lung. Wasson (10) reports that these are typically bilateral increase in width and density of the hilus with accentuation of the peribronchial and perivascular markings. Early changes consist simply of a hilus thickening; later, however, markings extend from the hila toward the bases. Only in advanced cases is any parenchymal change seen as evidenced by interstitial connective tissue changes. These findings suggest a bronchial entry of the infecting organisms with peribronchial inflammation. Were a shower of organisms to lodge in the lung from the blood stream a more generalized picture of inflammation with early parenchymal involvement would be expected.

At times early metastatic malignancy in the chest presents the appearance of peribronchial and perivascular infiltration along the bronchi of the hilum region and lower lobes, simulating the sinus lung. Such metastases are usually found in direct extensions of carcinoma of the breast into the mediastinum (24). An excellent example of this perivascular infiltration was seen in our clinic in a case of scirrhus carcinoma of the palate; primary growths elsewhere have also been reported. If cancer growths, which metastasize via the lymph and blood channels, can simulate the sinus lung it might be argued that the infection of true sinus lung probably arrived via the same route. The paucity of such cases, however, indicates that the lymph blood route is probably the exception rather than the rule. It is hard to conceive why other organs are not simultaneously involved more frequently when organisms are using the blood stream as a conveyance from lymph duct to lungs as in cancer metastasis. It seems unreasonable to expect all of the organisms to lodge in the lungs.

From anatomical considerations it is easy to see why chronic sinus infection should be more often primary than chronic pulmonary infections. Spontaneous recovery of the respiratory tract from catarrhal infection is the rule. In the case of the tracheobronchial tree, drainage is usually easy because ciliary action is markedly reinforced by coughing. It is only when destruction of the ciliated epithelium occurs that drainage is impaired because the cilia will bring secretions from the smaller bronchioles to the bronchi and trachea where they are readily disposed of by coughing. As already mentioned, the tracheobronchial tree rids itself of secretions in 12 to 15 minutes (7). Repeated infections with eventual destruction of the ciliated bronchial epithelium would therefore seem to be necessary in order to interfere with drainage and therefore produce a chronic bronchitis.

The nasal accessory sinuses, on the other hand, all communicate with the nose by narrow ostia. A small mucus plug at the orifice or inflammatory swelling of the mucosa lining the ostia may occlude the opening during catarrhal affections of the nose. Deflections of the nasal septum and inflammatory swelling or hypertrophy of the middle turbinate may likewise occlude the ostia. It is, therefore, not surprising, that catarrhal infections frequently become chronic in the sinuses, for impaired drainage during acute inflammation means accumulation of pus under more or less pressure with resultant hypertrophic and eventually destructive changes in the mucosal wall.

This is amply corroborated by clinical experience. The chronic bronchitis case usually gives a history of repeated acute attacks of bronchitis preceding the chronic stage, while the sinus patient frequently can date his sinus infection to a single head cold which localized in one or more of his sinuses. The most important observation from a practical viewpoint is that an early bronchitis is readily cured or arrested when the associated sinusitis is cleared up (6).

French Army medical officers were among the first to stress the importance of investigating the paranasal sinuses in cases of productive cough not due to tuberculosis. Sargeant reports that incorrect diagnoses of tuberculosis had been made in one-third of the supposedly tuberculous soldiers in the French Army during the World War, and that most of these mistakes were in men who had a chronic bronchitis associated with infection of the sinuses. While chronic bronchitis and sinusitis may appear at any time, it is interesting to note that the history of chronic cough frequently dates back to childhood and may be attributed to an attack of measles, whooping cough, or one of the exanthemata. Sinus infection in young children has been considered rare, mainly because the sinuses were thought to be uniformly rudimentary or absent. Because of the difficulties of examining sinuses in young children this belief was allowed to persist unchallenged. The anatomic researches of Onodi, Schaeffer, and Davis have proved that some of the sinus cells are frequently well developed at birth and that their development is governed by no law. This means that well-developed sinuses may be present in a young child just as we know that adults frequently have mere rudiments. Improved diagnostic procedures have shown that sinusitis does occur with much greater frequency than formerly suspected and that it is not an uncommon complication of the childhood contagious diseases and influenza. When the history of bronchitis, therefore, seems to point toward one of these diseases as the etiological factor, it is well to remember that this is not inconsistent with sinusitis being the underlying cause (12). In a series of 196 cases of chronic nontuberculous bronchitis, all but 6 had a definite chronic sinusitis. Of

the 190 sinusitis cases, 164 were treated with uniformly good and, in many cases, brilliant results. The great majority had one or more operations on the sinuses, and the best and quickest pulmonary results were reported in this group. A number were cured by conservative measures alone, however.

Various observers have claimed as many different sinuses as the chief etiologic factor. The maxillary sinuses are indicated most frequently but the ethmoids are almost as constantly blamed. McIntire found the latter and the sphenoids affected in 79 percent of his series (6). The conclusion seems to be that any or all the sinuses can be responsible. Purulent types of sinusitis appear to have the highest incidence of bronchial complications.

Sinus lung has no pathognomonic physical or X-ray findings. The history frequently suggests tuberculosis and the physical findings may not be inconsistent with this condition. The Roentgen findings are those seen in all nontuberculous bronchitis, viz:

(1) Bilateral increase in width and density of the hila.

(2) Accentuation of peribronchial and perivascular markings. The former may be the only sign in early cases, but gradually there is an accentuation of the trunk and linear markings extending usually to the lower lobes, the shadows even extending to the domes of the diaphragm and commonly obliterating the costophrenic angle. In involvement of the upper lobes the shadows radiate upward to the clavicles, but the apices remain clear. Only in advanced cases is there parenchymal involvement as evidenced by interstitial tissue changes. Points of differentiation from incipient tuberculous infections are that in tuberculosis:

1. Hilus involvement tends to be unilateral.
2. The primary focus may be evident.
3. Calcification is not seen in the nontuberculous lesion. This is of more value in children.
4. A tuberculin test may be helpful.

Although roentgenologists disclaim any pathognomic findings, they must be impressed by something in these cases for they frequently report a chest as "suggestive of upper respiratory tract infection" or "disease of the nasal accessory sinuses should be excluded." We have usually found these hunches well founded. In early cases there may be complete clinical and X-ray resolution when the sinusitis is eliminated. If the pulmonary condition has existed for a long time secondary bronchial changes may have occurred and the case is really one of bronchiectasis, in which the outcome is not favorable. The reason for this is that originally the lung pathology consists of a catarrhal inflammation of the bronchi and bronchioles. If this continues for a long time infection may extend deeper into the walls of the tubes. The bronchioles, not having the cartilagi-

nous support of the larger bronchi, are the first to become weakened so that intrabronchial tension such as that produced by coughing results in dilitation or sacculation. These sacculations become filled with purulent exudate which fails to evacuate and acts as a secondary focus of infection. Removal of the original sinus focus cannot be expected to effect a cure any more than removal of periapical dental infection cures advanced arthritis with secondary foci in the jonts even though the arthritic infection was secondary to the dental disease.

Forty-five percent of a series of nontuberculous bronchiectasis cases had a history of gradual onset and 15 percent had chronic cough since childhood. Those cases due to lung abscess or aspirated foreign bodies usually have an acute onset and also have a high incidence of sinusitis. As already indicated, sinusitis found in these cases may be due to spraying of the nasopharynx with infectious organisms during tussive efforts. In those cases giving a history of gradual onset and preceding bronchitis, most observers consider that the sinus infection preceded the bronchiectasis (13). They base this upon the clinical history. It is not surprising to find that bronchiectasis has the highest incidence of sinusitis, since it is possible for infection to occur both before and after the appearance of bronchiectasis. In a series of 50 cases 80 percent showed clinical and 100 percent X-ray evidence of sinusitis (2). Ninety-one percent of bronchiectatic patients with sinusitis had maxillary involvement with frank pus, either alone or associated with other sinusitis. No constant relationship was seen between the degree of sinusitis and bronchiectasis (14). Even though secondary foci are present in the lung, Clerf (13) stresses the importance of treating the sinusitis while bronchoscoping his patients and increasing their general resistance. Many sporadic cases of bronchiectasis have been markedly improved by treatment of the associated sinusitis (15).

The term "asthma" originally included many unrelated conditions, such as cardiac decompensation, chronic bronchitis, and bronchiectasis, characterized by wheezing. At the present time the unqualified term "asthma" has come to mean bronchial asthma, which is recognized as an allergic state. C. Walker (16) recognizes a clinical entity often confused with bronchial asthma because of some similarity of symptoms and calls this symptom complex asthmatic bronchitis. This condition is simply a bronchitis or bronchiectasis characterized by periodic attacks of dyspnoea resembling the allergic spasm of bronchial asthma. The dyspnoea is produced by tenacious mucus partially plugging the bronchi. The foregoing remarks on the relation of sinusitis to bronchitis and bronchiectasis are equally applicable to asthmatic bronchitis.

Bronchial asthma is characterized by spasm of the smooth muscles of the bronchi, with edema and infiltration of the mucosa causing expiratory dyspnea. Frequently there is an accompanying edema and infiltration of the nasal and sinus mucosa. The turbinates become engorged and there is a profuse watery discharge. This is frequently called "nasal asthma" and is really a vasomotor rhinitis and sinusitis. If X-rays of sinuses are taken during this period thickened membrane is reported and a diagnosis of pan-sinusitis may be made. A plate taken between attacks would show a normal mucosa.

The nasal picture of allergy is that of a pale edematous mucosa with abundant clear transudate as distinguished from the red hypertrophic mucosa and purulent exudate of inflammation (17). Usually the symptoms of asthma are so outstanding that the nasal symptoms are forgotten or overshadowed.

Hansel (18) showed that the changes in sinus mucosa are identical with those in the bronchial mucosa in allergy. It is therefore evident that we might expect erroneous diagnoses of sinusitis in allergic individuals. On the other hand, frequent allergic congestions of the nose may interfere with sinus drainage and predispose to secondary infection or true sinusitis.

Statistics as to the frequency of sinusitis in asthma vary widely. Seventy-six percent of 500 asthmatic patients showed X-ray evidence of sinusitis. The ethmoids were involved most frequently. The antra and frontals next and the spheroids least (19). Since 72 percent of a group of apparently normal controls likewise showed X-ray evidence of sinusitis no etiologic relationship can be deduced. In another series of 400 cases of chronic sinusitis of at least 1 year's duration, three-fourths had no evidence of pulmonary disease. About one-fourth had some form of nontuberculous involvement. Only 12½ percent had asthma and of these but 8¾ percent had asthma begin at the same time or after sinus symptoms were first noted (1). It seems, therefore, that sinusitis is at least not a frequent cause of asthma, if at all. In a review of the literature from the standpoint of both the chest and nose specialist as to the incidence of nasal disorder or sinus disease in chronic perennial asthma, figures ranged from 14 to 90 percent and beneficial results from rhinologic treatment varied from 0 to 100 percent (3). Obviously these statistics are worthless.

That nasal treatments and manipulations may result in great relief, and prolonged so-called "cures" in some cases of asthma cannot be denied. However, no one procedure is efficacious in all or even a majority of cases. Voltolini first reported relief from asthma by the removal of nasal polypi. Since then, every region of the nasal cavity, but more specifically the upper and posterior

portion of the septum, ethmoid region, tips of the middle and inferior turbinates, points of contact between septum and turbinates, all the sinuses, and the sphenopalatine ganglion have each been referred to as the most important point, irritation or disease of which may cause asthma (3). A reflex path from the nasal chambers to the lungs is recognized by way of the parasympathetic nervous system but there is a similar path to every viscus in the body. Even psychic stimuli can precipitate or inhibit an asthmatic attack. This "trigger mechanism" may be present in any of the areas previously mentioned but cannot be exclusively claimed by the rhinologist. Experience has shown that almost any type of nasal procedure frequently gives temporary benefit to asthmatics. For example, 80 percent of a group of 35 asthmatic patients were symptom free for an average of 4 months following a Caldwell-Luc operation. But 1 in 5 failed to obtain any relief. When one or more other operations preceded, the results of the Caldwell-Luc were progressively less favorable. In none of these cases and in other patients undergoing other types of sinus operation was there permanent relief (3). The conservative school stress tampons, radiation, vaccines, etc., but statistics in groups treated by both radical and conservative nasal manipulations are uniformly depressing after a 5-year period and valueless for a shorter time. Some favorable results from sinus treatments in asthmatics have been reported when the allergic individual is sensitive to his own sinus organisms or their products. Positive dermal tests can be demonstrated but the incidence of this type is comparatively rare (2). Clearing the sinusitis here may give relief, but if the underlying allergic state persists the individual may soon become sensitive to something else.

Radical sinus surgery in allergic asthma where no secondary sinus infection is present seems as rational as excising a wheal to cure urticaria. Nasal polyps are found in about 30 percent of asthmatics (2). These are of the mucoid type and are now considered to be almost invariably associated with allergy (21). Kern and Schenck failed to find a single case of true mucoid polypus occurring in individuals who did not have either a history of allergic symptoms, a family history of allergy, or positive skin sensitization tests. This conclusion was reached from consideration of about 2,500 cases seen in the department of otolaryngology in the University of Pennsylvania Hospital.

The pathology of mucus polypi is that of hypertrophy and edema of the tissue from which the polyp originates. Mucous glands may be lacking or present in scanty remains. Ciliated cells become increasingly rare in older polyps. The bulk of the polyp consists of loose areolar fibers with large tissue spaces containing albuminous fluid. Lymphatic clefts between areolar fibers contain lymphocytes,

plasma cells, and eosinophiles. The latter are found constantly when the polyp has been recently in contact with allergens; otherwise they may be absent. Other types of polypi are due to glandular hyperplasia and cellular infiltration and may be accompanied by vascular changes, such as angioma. These changes are essentially similar to those of hypertrophic rhinitis and are probably due to infection. Various types of new growth in the nose have also been reported. From the pathological changes seen in mucoid polypi it is apparent that regression to a normal state would be very unlikely. However, in five cases typical mucus polyps completely disappeared after treatment of allergic factor alone.

The removal of these polyps would seem to be indicated, because the operation is comparatively simple and at least temporary improvement in the asthmatic condition frequently follows. Besides, the patient has better nasal aeration after operation and the tendency to secondary sinus infection is diminished. If the causative allergens can be avoided or the patient desentisized the chances of more prolonged cure are, of course, enhanced. If secondary sinus infection is already present, drainage is facilitated. Return of or intensification of asthmatic symptoms is frequently associated with recurrence of polypi which usually take place when exposure to allergens continues. Radical ethmoid exenteration does not seem a logical procedure since the regenerated mucosa will be subject to the same changes as the original. An interesting observation is that out of 1,000 cases of asthma only one had bronchiectasis. Clerf, who believes that the most common cause of bronchiectasis is sinusitis, found no cases of asthma in his entire series of bronchiectasis in children. The flat X-ray findings in these conditions may be similar but Iipiodal failed to show dilation in asthmatics (22).

From all this contradictory chaos, it seems reasonable to conclude that:

1. Sinusitis per se is not a frequent cause of asthma, but that the underlying allergy may predispose to sinusitis.

2. The removal of mucoid polyps is indicated, and if a nasal trigger mechanism can be demonstrated it should be corrected. Otherwise, true sinusitis or secondary infection in asthmatics, representing as it does an intercurrent infection, should be dealt with as in nonasthmatics. The conservative treatment should be favored because the underlying allergy will probably predispose to reinfection of the regenerated mucosa following radical operations. In making a diagnosis of sinusitis in asthmatics care should be taken not to confuse vasomotor changes with infection. The underlying allergy should, of course, be studied and, if specific allergens are found, the patient should either avoid them or be desensitized.

BIBLIOGRAPHY

(1) Bullen, S. S. Incidence of Asthma in 400 Cases of Chronic Sinusitis. J. Allergey. 4. p. 402. July 1933.
(2) Kern, R. A., and Schenck, H. P. Chronic Paranasal Sinus Infection. Relation to Diseases of the Lower Respiratory Tracts. Arch. of Otolaryng. 18. p. 425. October 1933.
(3) Tobey, H. G. Relation of Nasal Sinuses to Asthma. Sect. on Laryng. Otol. and Rhin. Trans. A. M. A. 1931.
(4) Eadie, C. M. Nasal Sinusities in Relation to Pulmonary Tuberculosis. M. J. of Australia. 1. p. 345. March 1933.
(5) Mullin, W. V. A Review of Sinus-Chest Infections. Amm. of Otol. Rhin. and Laryng. 41. p. 794. September 1932.
(6) McIntire, R. T. Pulmonary Symptoms Incident to Infection of the Accessory Sinuses of the Nose. U. S. Naval Med. Bull. 18. June 1923.
(7) Myerson, M. C. Laryngoscope. December 1922.
(8) McLaurin, J. G. Chest Complications of Sinus Disease. Amm. Otol. Rhin. and Laryng. 41. p. 789–93.
(9) Daily, L. The Maxillary Antrum as a Focus of Infection. Texas State Journal of Medicine. 26. p. 414–20. October 1930.
(10) Wasson, W. W. Relationship of Sinus Disease to Chest Diseases in Children. Radiology. 22. p. 432–44. April 1934.
(11) Kistner, F. B. Infection of Accessory Nasal Sinuses as the Cause of Chronic Nontuberculous Bronchitis. Northwest Medicine. 26. p. 203. April 1927.
(12) Knight, R. W. The Relation of Sinus Infection to Respiratory Disease. Canadian Med. Assoc. J. 18. p. 54. January 1928.
(13) Clerf, L. H. Bronchiectasis Associated with Disease of Nasal Accessory Sinuses. Arch. of Otolaryngy. 6: 28–35. July 1927.
(14) Quinn, L. H., and Meyer, O. O. Relationship of Sinusitis and Bronchiectasis. Arch. of Otolaryng. 10: 152. 165. August 1929.
(15) Palmer, A. Importance of Sinus Diseases as a Focus of Infection. E. E. N. & T. Monthly. 12: 227–30. July 1933.
(16) Weldon, W. A. Relation of Nasal Sinuses to Asthma. Kentucky M. J. 31: 360–32. August 1933.
(17) Dean, L. W. Relationships between Diseases of the Nose, Throat, and Pulmonary Diseases. J. Missouri M. A. 31: 13–14. January 1934.
(18) Hansel, J. K. Clinical and Histopathological Studies of Nose and Sinuses in Allergy. J. Allergy 1: 43. November 1929.
(19) Smith, A. F. Nasal Pathology Associated with Bronchial Asthma. Penn. M. J. 35: 607–09. June 1932.
(20) Hurd, L. M. Asthma in Relation to Nasal Sinusitis. Arch. Otolaryng. 17: 557–60. April 1933.
(21) Kern, R. A., and Schenck, H. P. Importance of Allergy in Etiology and Treatment of Nasal Mucuous Polyps. J. A. M. A. 103. no. 17. October 27, 1934.
(22) Manges, W. F. Pulmonary Disease as the Result of Naval Accessory Sinus Infection. Penn. M. J. 35: 240. January 1934.
(23) Pfahler, G. E. A Demonstration of the Lymphatic Drainage from the Maxillary Sinuses. Am. Journal of Roentgenology and Radium Therapy. 27: 352–6. March 1932.
(24) Sante, I. R. The Chest. Annals of Roentgenology. vol. XI. 1931.

THE CIVILIAN DOCTOR'S PART IN A NATIONAL MILITARY EMERGENCY

By Henry A. Monat, Lieutenant, Medical Corps, United States Naval Reserve

Then to the rolling Heav'n itself I cried,
Asking, "What Lamp had Destiny to guide
Her little children stumbling in the dark?"
—*Omar Khayyam.*

With memory's dying ashes of the 1914 conflagration, the realist who knows how changeable and temperamental nations may be, pre-

paredness to meet the national emergency in all its phases stands out in flaming letters!

The task thrust upon the shoulders of a civilian doctor in the case of modern warfare is tremendous. Whether he is in a community situated in the midst of military operations or be he behind the lines, his duties are important and difficult.

The moment mobilization is declared, the civilian doctors should organize and have a program similar to the following made out:

1. Education and cooperation of the public, as to: (*a*) Gas and bacterial warfare; (*b*) hygiene and prevention of communicable diseases; (*c*) raising the public's morale.
2. Arranging additional hospitalization for the wounded and sick.
3. Arranging safe, hygienic quarters as emergency homes in case of air raids or bombardments.
4. Arranging for a quick evacuation of insane and acutely ill patients in case of military peril to the community.
5. Stocking sufficient drug and serum supplies.

The education of the public must begin early and be accomplished by means of a very active propaganda, using radio, cinema, newspapers, and demonstrations. The doctors should enroll sanitary volunteers from amongst the civilian component of the community, whose work should be supplementary to that of the physicians, and under their guidance. These volunteers would demonstrate to the public the proper use of gas masks; they would organize gas-mask drills; they would be taught by the doctors how to use a pulmotor or administer oxygen in case of an emergency. They would also be taught to disseminate and instruct the populace as to the dangers in using uninspected food or water supplies.

All civilian school teachers should be enlisted as such sanitary volunteers to instruct their pupils in the A, B, C of hygiene, anti-aircraft, and bombardment drills, and in the use of gas masks. These volunteers would inspect frequently every livable quarter in the community, and the unsanitary ones ordered for immediate sanitary improvement.

In cases of certain citizens protesting the perforce hygienic alteration the case should be adjudged by the sanitary inspector—a physician.

Any unsanitary or hygienically suspicious quarters would undergo a thorough delousing and fumigation. All refuse would have to be incinerated immediately. An intensive rat-exterminating campaign would have to be instituted. All plumbing would have to be inspected by competent civilian plumbers and enforced correction or replacement of unfit plumbing made.

The sewage-disposal plant would have to be managed competently so as to prevent the spread of communicable diseases.

All of the population would have to be vaccinated against typhoid, small pox, and cholera, and be given clearance certificates, to be kept by all the civilians for inspection at any time. A forceful delousing of any unsanitary-looking individual would have to be enforced.

Establishing at terminals delousing stations so that no one could arrive in the community without proper health certificates. Guarding of all the public highways by civilian militia so that no one could enter without a clean bill of health, and also so that no food got into the community without first having been properly inspected and passed. An active bacteriological service would have to be established. The water supply would have to be checked often. All places where food and nonalcoholic drinks were sold or dispensed would have to be bacteriologically inspected at frequent intervals.

In the case of an outbreak of a communicable disease in a home, a most strict quarantine under seal would have to be enforced, together with frequent inspections.

Raising the morale of the public is another duty of a civilian doctor in case of war. It is up to him to prevent mass hysteria and phobia. This can be accomplished by proper supervision of amusements. He would have to see to it that the cinema and theaters gave amusing plays, that the music halls included light musical numbers in their programs, and that the newspapers published only stories of an entertaining character.

With the inevitable shortage of food amongst the civilian population of a community during a war, the proper dispensation of food rations becomes a matter of grave importance, and should be undertaken with the greatest possible tact. The public would have to be educated and advised by the physicians in charge as to the optimum food intake for an individual, the caloric and vitamin values of various articles of food and their relation to health. The physicians would have to see to it that essentially important foods, such as milk, fresh meat, and fresh vegetables, were available for hospital patients and babies. They would have to instruct the public as to which of the "food substitutes" (so well remembered during the late war by certain belligerent countries) are harmless.

The doctors would have to see that all the vacant suitable places, such as large concert halls, auditoria, and schools, be converted into emergency lazarets, ambulatoria, and clinics, to be kept in readiness for the incoming casualties, either "wounded in action" or convalescents, as the occasion might be. A portion of the citizenry would have to be instructed in emergency nursing, bandage-making, dressings, sterilization, and minor surgical technique.

Doctors would also have to investigate all of the available basements, especially the ones constructed of concrete, which would afford a certain amount of protection in the case of air raids or bombard-

ment. They would have to see that these basements were livable, kept in a hygienic condition, and, if possible, to have a sufficient water supply, so that in case of military peril the population could be given shelter.

A workable arrangement would have to be made with other hospitals within a safe vicinity, so that in the case of the terrain of military operations approaching a given community, the insane, the acutely ill, and the bed-ridden patients could be quickly and safely evacuated and transferred there.

They would see that there was a sufficient drug and serum supply; enough to tide the community over its trying period.

In addition to the above herculean duties, the civilian doctor would have to be further burdened by the additional patients left in his charge by his colleague in the military service, who must, of necessity, take care of those actively engaged in the fighting units.

In conclusion, we may see that even though the civilian doctor in a national military emergency may not be taken by the Valkyre to Valhalla, his contribution in case of war in the future is as important and as patriotic as any one of his colleague's on the field of glory.

ALLERGY, AUTOINTOXICATION, AND INDICANURIA

By J. R. Sayers, Lieutenant, junior grade, Medical Corps, United States Navy

It is my desire to treat a number of related conditions that apparently arise directly or indirectly from a chronic gastrointestinal disease of dysfunction, and to dwell particularly on the latter.

The subject of autointoxication is one that has been much discussed, little understood and dismissed by the majority of us as being either of little consequence or about which we could do nothing definite.

Drawing an analogy from the continued action of focal infection on the general well being of the human organism, in producing such diseases as affect the cardiovascular system, and the joints in particular, that is the action of circulating toxins, it has been conceived by many investigators and writers that similar results may be obtained from abnormal absorption by the mucosa of the gastrointestinal tract. It is not, therefore, necessary to set forth further discussion to validate or invalidate this assumption.

It is impossible to state what the underlying pathology is, if indeed there be any, or what may be the patho-physiological condition, but at least one may state with safety that the gut is irritated directly proportional to the systemic symptoms. This latter statement is borne out by the degree of abdominal tenderness found on deep abdominal palpation.

The symptoms are almost entirely subjective, except perhaps for quickening of the pulse and respiration on slight exertion and the above-mentioned tenderness of the abdomen. The subjective symptoms are almost myriad, as might be expected from any chronic toxic condition. The most common complaints are, headache, backache, general malaise, undue fatigueability, cardiac distress, syncope, decrease of mental acuity, and generally of indigestion. For some reason the patient seldom complains of abdominal tenderness, although they may complain of mild abdominal cramps associated with a mild diarrhea, which is usually followed by slight constipation. The stools are seldom well formed and indicanuria is usually present either during or following an attack.

This latter fact has proven to be of utmost importance in my observations and experiments. Methods of analysis, diagnosis and treatment based on its presence or absence in the urine are worthy of description. The presence of indoxyl in the urine has been known for many years, and was associated with intestinal toxemia and other sources of protein putrefaction. Consequently my first efforts were directed toward following the recognized procedure of prescribing a diet that was practically protein free, that is consisting principally of fruits, vegetables, and cereals. Much to my dismay, my early patients failed to improve, some even becoming worse. The thought finally occurred to me that of two people on the same diet, one would remain normal and the other become ill. On closer examination I found that all these patients exhibited signs of tenderness of the abdomen, which to me meant probable irritation or a low-grade inflammation of the intestinal mucosa.

On pondering this thought I conceived the idea that perhaps the selective ability of the mucosa, for absorption, was either lowered or destroyed, thus allowing foreign substances to enter the blood stream. On testing the urine of patients three times a day I discovered that the amount of indoxyl present varied from none to an intense reaction and coincided with the period of digestion of certain meals, also that its presence was coincident to the height of the patients subjective symptoms. This, of course, led to the belief that some specific food or drink was perhaps the etiologic factor.

The next move, of course, was to chart the articles ingested and to test the urine at least three times daily. At the same time the diet is controlled in such a manner that an offender may be detected by correlation of the food chart with the urinary findings. Shortly it became evident that certain articles were responsible for the major symptoms and coincided with the indicanuria.

It has been my practice to place each individual on what I call a basic diet. This diet consists of articles of food that I have found to be most frequently tolerated, such as adult beef, white bread, car-

rots, English peas, and apples. In the event that the urine remains clear on this diet one other article of food is added daily until a positive urine is noted, then the last article added is considered as an offender and is removed from the diet. As soon as the urine is again clear the process is repeated.

This procedure is slow, tedious, and occasionally has some puzzling trends, but withal is well worth the effort in view of the fact that improvements in the patient's condition is usually pronounced. The general vitality is noticeably increased, and the secondary conditions usually become symptomless.

As I have previously mentioned, a low protein diet was of no avail. It is then not surprising that the chief offenders are found in other classes of foods. My experience has been that the leafy vegetables are the chief offenders, Broccoli, cauliflower, cabbage, tomatoes, and asparagus are the most prominent. Of the citrous fruits, grape fruit and lemons are the most common, and of the stone fruits sour plums and apricots appear to lead the list. In considering the meats, veal, fowl, rabbit, and pork are most commonly offensive in the order named, while adult beef is almost universally well tolerated. Cereals are usually well tolerated, except oats, bran, and grain heart products. The condition is, however, one of individual susceptibility, and each case must be worked out accordingly.

As a matter of general discussion, I am led to believe that the condition is primarily one of susceptibility to specific food proteins. There is, however, no proof that the allergic condition is not secondary to a primary intestinal irritation of some other origin. Due to the fact that the subjective symptoms at each attack are commensurate with the amount of indoxyl secreted in the urine, it is reasonable to assume that the degree of toxemia varies accordingly. This is borne out by the severity of secondary symptoms, such as increases in arthralgia, increase of premature systoles, and shortening of the breath.

Due to rapid change of station of duty, both of patients and myself, I have been able to observe only a limited number of patients over a long period of time. These few have been quite instructive. A small number of hypertrophic arthritic cases, so diagnosed by the aid of X-ray, have improved or become symptom free. These cases were all vertebral or sacroiliac. Two cases of cardiac arythmia, one of premature systoles with a low T-wave, the other a paroxysmal type of tachycardia, improved commensurate with the degree of restriction to a tolerated diet. A large number of cases of chronic prostatitis of unknown etiology have shown marked improvement. One case of dry scaly eczema of an acute type was seen, diagnosed as due to citrus fruits and cleared in less than 10 days after removing them from the diet. Reference has been made of these few cases

only to direct attention to the disease conditions that are due to autointoxication, or that at least improve on a well-tolerated diet.

In conclusion I wish to state my theory relative to the three apparently associated conditions. That to certain people an article or articles of diet are irritants to the digestive tract; that under the mild inflammatory condition thus produced the mucosa of the gut loses its ability of selective absorption, thus permitting toxic substances to enter the circulation, and these toxins have the same effect as toxins from other foci of infection, depending on their affinity and the individual's reaction.

CAMPHORATED OIL IN THE TREATMENT OF MINOR INDUSTRIAL WOUNDS

By D. N. McINTURFF, Jr., Lieutenant, junior grade, Medical Corps, United States Navy

In the treatment of minor industrial wounds our first concern is to return the patient to his work with the least pain possible and with maximum prevention of primary and secondary infection.

The great bulk of our cases is made up of lacerations and burns, so that this article is directed at their treatment exclusively.

The prevention of pain begins immediately after the patient sustains the injury, for he realizes that if it is possible to continue work he will be allowed to do so. Thus the fear of losing time is dispelled, and he enters the dispensary in a cooperative mood, minimizing rather than exaggerating his injury.

What we consider the prerequisites of an ideal antiseptic are as follows: (1) It should be strongly inhibitory to bacterial growth; (2) it should not hurt, burn, or adversely alter the wound or surrounding tissue; (3) it should make dressing changes possible without sticking and tearing the new or injured tissue; (4) it should produce no general toxic symptoms; (5) it should be inexpensive, easily handled, and free from offensive odor. We have found that camphorated oil fills these requirements. The purpose of this article is to show the usefulness of this drug as we have found it in 100 consecutive cases.

In a bacteriological study of various fats and oils, Lohr, in Berlin, found that they are usually free from bacteria. He found that bacteria ordinarily found in wounds such as streptococci and staphlococci, and bacillus coli, perished in cod-liver oil.

In 1932 G. R. R. Hertzberg reported the wide range of use of camphorated oil. His enthusiasm for its benefits led him to use it in wounds of every size and type, including compound fractures, postoperative fascial plane infections of the abdominal wall, septic cellulitis, osteomyelitis, and in the treatment of burns, with uniform satisfaction. He attributes the results to bacterial inhibition and

stimulative action the oil has on granulation, while being nontoxic itself.

Our treatment of lacerated wounds is to first flush the wound out thoroughly with ether which is allowed to evaporate. This is followed by stitching if necessary. The wound is then dressed with sterile gauze saturated with camphorated oil. If the wound is so situated that surgical rest cannot be obtained otherwise, a splint is applied. Tetanus antitoxin is used when necessary.

The use of ether follows the suggestion of P. Koristenskiy, who reports its use in open wounds in 1,000 consecutive cases in which healing by first intention occurred in 93 percent of the cases. Ether acts as a local anaesthetic which assists in suturing, as well as being bactericidal, and influential in inducing phagocytosis. It is a tissue irritant but the oil dressing which follows makes this transitory.

The treatment of second-degree burns is first to apply amertan, a tannic acid jelly, to get the benefit of coagulation, followed the next and subsequent days by gauze dressing saturated with camphorated oil. Used in this manner, reepithelization is especially rapid since there is no secondary infection and no trauma associated with dressing renewal which is done daily.

Since adopting this routine in November we have treated the following consecutive cases:

Site	Lacerations	Second-degree burns	Site	Lacerations	Second-degree burns
Finger or fingers	41	2	Lower leg	3	1
Toe or toes	10	0	Upper leg	0	1
Hands	10	6	Face or scalp	10	0
Lower arm	8	4			
Upper arm	1	3	Total	83	17

Results.—No primary or secondary infection resulted in any of the cases. In mutilative wounds of the fingers, which occur quite frequently, no attempt is made to improve the appearance of the member the first day. Often partial amputation is seriously considered, but because of previous good results, conservative treatment has been given preference. In a day or so the amount of devitalization can be seen and removal at that time is tolerated much better. In such cases the amount of scar tissue is minimal, since there has been no aggravation by infection. Contractures are prevented by splinting.

In the burn cases, the results are particularly gratifying; healing and reepithelization going on together, with the patient continued at his work. One burned area measured 8 inches by 6 inches and included the anterior surface of the knee joint. The patient was a

Negro and the new, bluish epithelium could be easily followed as it progressed from the entire margin to complete, uninterrupted healing.

None of the patients complained of pain, but on the contrary often comment on the contrast found between the oil treatment and other forms they have previously undergone.

Our statistics show that the number of accidents is usually higher on Friday than any other day. This makes redressings quite a problem, for although the dispensary is always open, the yard does not work the remainder of the week. The workmen often live considerable distance from the yard. Many have no cars, etc. In such cases rather than have them go to a drug store, or apply home remedies, we give them some bandage and a small bottle of the oil to take home. Their instructions are to daily remove the outer dressing, resaturate the underdressing without disturbing it, and rebind the wound. If any throbbing or unusual pain develops they are instructed to come to the dispensary at once. So far this arrangement has been very satisfactory, and they often return Monday morning with the wound nearly healed.

CONCLUSION

1. We believe that ether followed by camphorated oil dressing is the ideal method of treatment of lacerations as shown by 83 consecutive cases in which there was no primary or secondary infection.

2. The treatment of 17 consecutive cases of second-degree burns is given and the beneficial use of camphorated-oil dressings following the primary use of amertan is shown.

REFERENCES

(1) Lohr, William, Cod Liver Oil Bandage. Archiv fur klinische cherurgie, Berlin, September 21, 1934.

(2) P. Korestenskiy, Ether in the Treatment of Freshly Infected Wounds. Vrack. Gaz. October 31, 1930.

(3) G. R. R. Hertzberg, Camphorated Oil, An Ideal Wound Antiseptic. M. J. and Rec. December 2, 1932.

CLINICAL NOTES

A CASE OF PERIPHERAL NEURITIS FOLLOWING ARSENICAL POISONING WITH SEVERE EXFOLIATIVE DERMATITIS

By W. G. KILBURY, Lieutenant, Medical Corps, United States Navy

C. F. S., private, United States Marine Corps, aged 23 years (serving with First Brigade, United States Marines, in Haiti), and later transferred to the naval hospitals at Norfolk, Va., and Chelsea, Mass.

Chief complaint (on admission to naval hospital at Chelsea, Mass.).—Numbness in right and left feet and ankles.

Family history.—Irrelevant. (Single.)

Past history.—Usual childhood diseases. In good health up to 1932, when he first contracted venereal disease.

Venereal history.—Gonococcus infection of urethra in January 1932. Chancroid penis in December 1932. Chancroid penis in July 1933. Syphilis, 1933.

Present illness.—While serving with the Marines in Haiti, in July 1933, patient was admitted with a chancre on his penis. He was sent to duty under active antiluetic treatment. Receiving biweekly injections of neoarsphenamine of 0.45 gram.

Due to a severe chronic prostatitis occurring during the course of treatment, his treatment was extended.

Following the thirteenth injection of neoarsphenamine, the patient noticed some itching of his face and pimples over the face and neck. This was rapidly followed with puffiness under his eyes, and he was hospitalized immediately.

The edema spread rapidly to his chest and extremities, and following this he had an extensive generalized dermatitis with delirium. During this illness the patient lost 40 pounds in weight. After 2 months' illness, he gradually regained his weight and strength but began to have sharp pains in his toes. He had difficulty in walking, his feet felt numb, and he had a bilateral foot drop. His knees also felt weak and at times failed to support his weight. This was more pronounced in his left knee, at first, but later both knees would at times buckle under his weight. During this period of about 6 months following his original admission he still had multiple ulcers and small abscesses over his chest and lower abdomen and extremities. Patient was transferred to the United States, and while in the Norfolk Naval Hospital his condition greatly improved. He was given physiotherapy; massage, active and passive motion; hot-air baths to feet and ankles; infra-red lamp to spine, sinusoidal wave to extremities, and daily exercise. His skin became clear and he gained considerable weight. Later he was transferred to the United States Naval Hospital, Chelsea, Mass., in order to be near his home. He was admitted to this hospital on June 5, 1934. On admission, his chief complaint was, as stated previously, "numbness in both feet and ankles."

Physical examination.—Patient is a young American white male, age 24 years, well nourished and developed, weighing 165 pounds. Height 68 inches. Does not appear ill. Temperature 98.4, pulse 78. Respiration 20.

Head.—Symmetrical. No deformity. Numerous scars over face.

Eyes.—Pupils equal, regular, and react to light and in accommodation.

Ears, nose, mouth, teeth, and throat.—Essentially negative.

Neck.—Few cervical lymph nodes palpable.

Chest.—Expansion equal, symmetrical and good. Pitted scars over chest and back.

Lungs.—Resonant, no rales. Breath sounds normal.

Heart.—Negative. Blood pressure 130/80.

Abdomen.—No masses, no tenderness. Numerous pitted scars over abdomen.

Genitalia.—Several small pustules on scrotum. Contents normal. Scar on glans penis.

Rectal and prostate.—Several moist lesions on inter gluteal fold. No hemorrhoids. Sphincter tone good. Prostate gland moderately tender, normal consistency. Seminal vesicles strip readily.

Extremities.—Skin appears flabby, color good, appears normal otherwise.

Reflexs.—Knee jerks equally present but diminished.

Ankle jerks.—Absent. Slight limitation of dorsi flexion both feet. Tactile sensation diminished both feet. Tendency to steppage gait.

Romberg.—Negative.

Sensation to heat and cold.—Normal.

Elbow jerks.—Active.

Abdominal and cremasteric reflexes.—Present. No ankle clonus or Babinski. Mentality is clear.

LABORATORY FINDINGS (FIELD HOSPITAL, FIRST BRIGADE, UNITED STATES MARINES, PORT AU PRINCE, HAITI)

BLOOD—WHITE CELLS

	W. b. c.	Bands	Segmented	Lymphs	Mono.	Eos.	Juv.	Baso.
Oct. 4, 1933	12,600	10	69	16	3	3		
Oct. 5, 1933	21,400	4	76	17	3			
Oct. 6, 1933	13,800	13	65	20	1	1		
Oct. 9, 1933	10,500	17	60	13	1	6	3	
Oct. 11, 1933	13,400	11	56	24	2	5	2	
Oct. 12, 1933	13,750	19	48	23	2	2	5	1
Oct. 13, 1933	13,200	9	70	14	3	3	1	
Oct. 16, 1933	16,000	12	70	15	3			
Oct. 18, 1933	11,800	11	65	17	4	4		
Oct. 21, 1933	11,000	8	70	15	3	4		
Oct. 23, 1933	12,800	8	69	16	3	4		
Oct. 26, 1933	15,400	3	52	30	1	14		
Oct. 30, 1933	17,000	4	51	30	3	12		
Nov. 2, 1933	13,850	7	38	29	2	24		
Nov. 7, 1933	11,800	8	65	24		3		
Nov. 16, 1933	12,150	5	64	28		3		
Nov. 23, 1933	12,500	3	65	27	2	3		
Dec. 1, 1933	10,050	8	44	41		7		

BLOOD—RED BLOOD CELLS, ETC.

	R. b. c.	Hgb.	Platelets	Kahn	Sedimentation	Index
Oct. 9, 1933	3,300,000	67				
Oct. 19, 1933	2,770,000	60				
Oct. 23, 1933						
Oct. 26, 1933	2,950,000	58	222,950			
Nov. 2, 1933	3,160,000	62		Negative	1 hour, 28 minutes.	
Nov. 9, 1933	3,200,000	70	265,600			
Nov. 16, 1933	4,210,000	75				
Nov. 23, 1933	4,460,000	81	300,200			
Dec. 1, 1933	4,720,000	88	340,000			

LABORATORY FINDINGS (FIELD HOSPITAL, FIRST BRIGADE, UNITED STATES MARINES, PORT AU PRINCE, HAITI)—continued

BLOOD—RED BLOOD CELLS, ETC.=Continued

Urine	Reaction	Specific gravity	Albumin	Sugar	Microscopic
Oct. 7, 1933	Neutral	1.009	Positive (negative 1-10).	Negative	Motile bacteria, w. b. c. 1-2 h. p. f.
Oct. 9, 1933	do	1.012	Negative	do	Numerous w. b. c.
Oct. 11, 1933	Acid	1.010	do	do	Numerous w. b. c., many. epithelium.
Oct. 13, 1933	Alkaline	1.015	One plus	do	Many motile bacteria.
Oct. 21, 1933	Neutral	1.018	Negative	do	W. b. c. too numerous to count.
Oct. 24, 1933	do	1.020	Positive (negative 1-10).	do	W. b. c. 6 per h. p. f., few epithelium.
Oct. 28, 1933	Acid	1.021	Negative	do	W. b. c. 15 per h. p. f., occassional r. b. c.
Oct. 30, 1933	do	1.020	One plus	do	W. b. c. 10-12 per h. p. f.
Nov. 2, 1933	do	1.012	Negative	do	Numerous w. b. c.
Nov. 4, 1933	Neutral	1.010	do	do	Occasional w. b. c.
Nov. 6, 1933	Acid	1.004	do	do	Numerous w. b. c.
Nov. 7, 1933	do	1.011	do	do	14-16 w. b. c. per h. p. f., few epithelium.
Nov. 10, 1933	do	1.020	do	do	6-8 w. b. c. per h. p. f.
Dec. 2, 1933	do	1.020	do	do	Few w. b. c.

LABORATORY FINDINGS (NORFOLK NAVAL HOSPITAL, PORTSMOUTH, VA.)

March 30, 1934. Urinalysis negative except for white blood cells.

April 4, 1934. R. b. c., 4,400,000; w. b. c., 7,000; hgb., 85 percent; urine negative, except for a few white blood cells.

April 18, 1934. Urinalysis negative except for white blood cells.

April 26, 1934. W. b. c., 9,800; r. b. c., 4,600,000; hgb., 90 percent.

LABORATORY FINDINGS (UNITED STATES NAVAL HOSPITAL, CHELSEA, MASS.)

June 6, 1934. Urinalysis negative for albumen and sugar. No w. b. c. or r. b. c.

June 6, 1934. Blood Kahn, negative; r. b. c., 4,900,000; w. b. c., 7,200; hgb., 90 percent.

November 6, 1934. Blood Kahn, negative.

COMMENT ON LABORATORY FINDINGS

Laboratory data were given in some detail because the interesting changes can be easily seen and followed. It is noted that at no time was there a granulocytopenia. On the contrary, there was an absolute and a relative increase in granular cells, which at the onset was almost treble. There was a marked eosinophilia during the fourth week of the disease. Monocytes remained in general at their usual absolute value. An interesting feature is the marked drop in hemoglobin and red blood cells, most marked at the end of the second week, following the onset of the disease. A platelet count at 3 weeks after the onset was somewhat below normal but gradually increased. While in the naval hospital at Chelsea, Mass., as a patient, his convalescence was uneventful. Physiotherapy and general hygienic measures were continued, and upon discharge from hospital on

December 4, 1934, the peripheral neuritis cleared up to a great extent with the exception of slight numbness over the plantar surface of the left foot. There was also slight limitation of dorsi flexion of left foot. Repeated blood Kahn's were negative and the patient was considered fit for duty exactly 17 months following his original admission.

SUMMARY

Peripheral neuritis following arsenical poisoning with severe exfoliative dermatitis is considered very rare following antiluetic medication.

Stokes mentions one case of peripheral neuritis following a very large total dosage of neoarsphenamine, but considers the condition extremely rare.

Beeson reports a severe grade of peripheral neuritis with exfoliative dermatitis.

Moore and Keidel have likewise noted similar cases.

MOLLUSCUM CONTAGIOSUM

REPORT OF CASE

By Bruce V. Leamer, Lieutenant, Medical Corps, United States Navy

The patient, J. O., age 26, private, United States Marine Corps, reported to the regimental hospital, Shanghai, China, on February 1, 1935, complaining of "numerous warts" in the left axillary region and down the left side of body.

Family history and past medical history irrelevant.

Present illness.—About 2 months prior to the time he came to the hospital, patient had the axillary hair shaved by one of his friends because of "crabs." Two weeks later he noticed the appearance of a few warts in the axillary region. These gradually increased in number and size until there were about 50, varying in size from a pinhead to a small pea. Patient gave no history of any fever or other organic disturbances and has not been annoyed by itching or other irritation, except that perspiration causes burning and smarting in the axillary region.

Etiology.—Going into the etiology of this particular infection, the patient states that the man who shaved him had a number of warts on his face which he was constantly "picking at." He thinks he was infected in this way; unfortunately, this man was transferred from the regiment before the patient reported his own infection.

Physical examination was grossly negative except for the skin condition. In the left axillary region there were approximately 30 discrete, waxy, globular, epithelial tumors, whitish in color; some having minute rounded orifices at the apex from which a thick, cheesy exudate could be expressed. Several of the tumors were hemorrhagic, due to irritation and scratching. There were several small areas covered with a dried serosanguinous exudate which the patient stated had been "warts" similar to the others. He had scratched the tops off and they had healed up.

Treatment.—The first 10 days the lesions were treated by the application of an ointment of sulphur, resorcin, and salicylic acid. This treatment resulted in no improvement. The lesions were then incised, the contents expressed by pressure, and dry dressings applied. All the lesions healed in from 7 to 10 days. After a period of 3 weeks there was no evidence of any new "tumors" forming. The diagnosis was confirmed by microscopic examination.

Molluscum contagiosum is a comparatively uncommon affection. It is a mildly contagious disease of the skin, characterized by the occurrence of small, waxy, globular, epithelial tumors which are whitish or pinkish in color and often have minute rounded orifices at their apices.

There are no constitutional or subjective symptoms. The lesions are unquestionably due to some external infection. They usually start as firm, pin-point, globular, or acuminate tumors with broad bases. As they increase in size they may become slightly flattened and unbilicated. They may attain to the size of a small pea. As the tumors reach maturity they become somewhat softened and some may break down and supperate, eventually healing. As a rule, if untreated, the lesions persist for months. Occasionally some of the tumors may heal spontaneously. They vary in number from 1 to 40 or 50. There are several cases reported in which there were as high as 500.

The lesions are mainly on the face, breasts, genitalia, and inner surfaces of the thighs. However, no region is exempt.

The disease occurs in birds and animals as well as in man. It is autoinoculable and undoubtedly contagious, although the causative agent has not as yet been isolated. Infection in bathing pools, from the interchange of bathing suits; in gymnasiums and turkish baths is common. Physicians may be accidently inoculated from their patients and experimental inoculations have been produced. The incubation period is from 2 to 10 weeks. The majority of patients are children and the inmates of children's homes are more susceptible than children reared under more favorable circumstances.

The diagnosis is based on the size, color, and appearance of the lesions and the fact that the contents of the little tumors can be expressed through the central orifices. Fibromata, milia, comedones, and verruca are to be excluded.

The disorder is harmless, but the lesions, if untreated, are usually persistent. If the growths are numerous, lotia alba, 5 percent ammoniated mercury ointment, or sulphur, salicylic acid, and resorcinal ointment are sometimes beneficial. In the majority of cases it is better to incise the little tumors with a sharp bistoury, squeeze out their contents, and apply tincture of iodine to the inside of the cavities. Engman recommends simple incision, allowing the blood to flow into the cells of the lesions and dry.

CAVERNOUS SINUS THROMBOSIS

REPORT OF A CASE

By R. W. TAYLOR, Lieutenant, junior grade, Dental Corps, United States Navy

The cavernous sinus is located at the side of the body of the sphenoid bone. It extends from the superior orbital fissure anteriorly to the apex of the petrous portion of the temporal bone posteriorly. The lateral wall of the sinus contains the oculo-motor, trochlear, and abducent nerves; also the ophthalmic and maxillary divisions of the trigeminal nerve and internal carotid artery with its sympathetic plexus.

The cavernous sinus receives the venous blood return from a widely distributed area. Through the medium of the ophthalmic veins, tributaries, and related facial veins, the eyelids, the cheek, and the external nose are drained through the cavernous sinus. From the mucuous membranes of the sphenoidal and posterior ethmodial air sinuses numerous small veins pass through the bony walls. There are small veins which traverse the foramen ovale and the foramen Vesallii and communicate with the pterygoid plexus situated in the infratemporal fossa. In this manner the cavernous sinus is brought into indirect venous relation with the soft palate, the fauces, the pharynx, and the alveolar processes of the maxilla and mandible. In case of septic thrombosis from a primary focus of these areas, the cavernous sinus may be secondarily infected through the medium of the inferior ophthalmic vein and the pterygoid venous plexus. The cavernous sinuses communicate with each other by means of the anterior and posterior intercavernous sinuses.

From the location of the primary infection and lack of clinical evidence showing involvement of the angular or ethmoidal veins, it is highly possible that the infection in the following case reached the cavernous sinus through the pterygoid venous plexus.

F. G., Matt., second class, age 25, Filipino. On May 16, 1935, a carious aching lower right second molar tooth was removed under conduction anesthesia on board patient's ship. X-ray equipment not available, the diagnosis being made clinically.

May 17, 1935. Postoperative examination revealed no complication. Patient dismissed.

May 22, 1935. Returned and complained of pain in region of extracted tooth. A degenerated clot removed and iodoform gauze dressing inserted in alveolus. Patient admitted to sick list.

May 23, 1935. Showed signs of increased pain. Alveolus irrigated and fresh iodoform gauze dressing inserted. Examination revealed no edema of buccal tissues and no apparent cause of pain. Medical officer consulted. Sedatives administered. Morning temperature normal, but increased by evening to 102.

May 24, 1935. Buccal tissues revealed first signs of swelling. Medical officer consulted. Temperature, 102. White blood count, 14,950; juveniles, 2; bands, 17; segmented, 72; lymphs, 9. Urinalysis: Specific gravity, 1.012; acid; negative for albumin and sugar.

May 25, 1935. Temperature normal. Pus drained from buccal tissues between bone over socket and periosteum. White blood count, 16,000; bands, 11; segmented, 60; lymphs, 29.

May 26, 1935. Patient showed improvement. Appetite good. Pus drained and iodoform gauze dressing inserted. Temperature morning, 100.4; afternoon, 102. W. b. c., 17,700; bands, 10; segmented, 61; lymphs, 25.

May 27, 1935. Temperature normal. Relatively little pus drained. Edema has moved posteriorly over ramus and has involved temporal region. Iodoform gauze dressing inserted. Patient complained of weakness and pain in the involved area. W. b. c., 18,800; juveniles, 5; bands, 22; segmented, 57; lymphocytes, 14. Transferred to U. S. S. *Relief* for further treatment and disposition.

Diagnosis: Osteomyelitis, mandible, right at tooth no. 18.

Chief complaint: Pain, swelling, and disability right side of mandible.

Family history: Mother dead, cause unknown. Father living and well, one brother and three sisters.

Past history: Mumps and osteomyelitis. Denies venereal disease.

Physical examination: Patient admitted stretcher case. A well-developed Filipino male, 25 years old, 64 inches in height, 140 pounds in weight. Skin clear. Scalp normal. Ears normal. Mouth: There is considerable swelling with pain, tenderness over right side of face near the angle of the mandible. Patient unable to open jaws except for about one-half inch. There is pain, tenderness, and fluctuation in the vicinity of recently extracted tooth. Neck: Post cervical glands palpable. No thyroid enlargement. No tremors. Chest: Expansion good and equal. Lungs clear. Heart: Apex fifth inter-space regular. No murmurs. Abdomen: No tenderness, rigidity, or masses. Genitals negative. Extremities negative. Blood pressure 104–60. Reflexes normal.

May 30, 1935. Area opened through the mouth and free drainage established. Rubber dam drain inserted.

June 1, 1935. Further drainage established. Suppuration has advanced over an area from third molar to first premolar tooth.

June 3, 1935. Condition showed improvement clinically. Swelling considerably reduced.

June 5, 1935. External drainage established over lower right mandible. X-ray showed definite osteomyelitis extending from angle to mental area on right side. Patient's condition showed no improvement. Temperature 102 to 104. Laboratory findings: Kahn negative. Urinalysis negative. W. b. c., 16,300; juveniles, 2; band forms, 20; segmented, 60; lymphocytes, 15.

June 6, 1935. There was moderate edema of both lids of right eye. Temperature 102 to 104.

June 7, 1935. Eye examination: Moderate proptosis with moderate edema of upper lid, marked chemosis of inferior half of globe which covers lower lid and prevents closure of palpebral fissure. No clinical evidence of thrombosis along the facial or angular veins. There was a horizontal diplopia increasing in the field of movement of the right external rectus muscle with limitation of movement in the same field. W. b. c., 24,400; R. b. c., 4,090,000; Hgb., 80 percent; juveniles, 4; band forms, 12; segmented, 71; lymphocytes, 11. Diagnosis changed to thrombosis cavernous sinus, right side. 200 cc antistreptococcus serum intravenously at 4 p. m. 60 cc intramuscularly at 10 p. m.

June 8, 1935. Condition about the same. Continued to have considerable drainage from the jaw. Eighty cc of staphylococcus antitoxin administered intravenously.

June 9, 1935. Blood culture taken June 7, 1935, positive for staphylococcus albus. There was beginning edema of lids of left eye. Moderate headache. Good drainage from the mandible.

June 10, 1935. Transferred to United States Naval Hospital, San Diego, Calif., for further treatment. Admitted to hospital only partially conscious. X-ray of right mandible exhibited moderate absorption throughout posterior half of mandible. There was no evidence of new bone formation. W. b. c., 11,200; Kahn, negative; coagulation, 6 minutes.

June 14, 1935. W. b. c., 10,900; sedimentation index, 21; temperature, 103; pulse, 140; respiration, 30. Symptoms became worse until both eyes showed marked proptosis and patient had symptoms of general sepsis. Blood cultures positive for staphylococcus albus. Patient showed signs of meningeal involvement and expired June 16, 1935.

Autopsy findings.—1. Osteomyelitis, mandible, right.

2. Cavernous sinus thrombosis.

3. Thrombosis of sinuses enclosed in the meninges and thrombosis along the anterior and posterior central gyri, at which site a leptomeningitis is present.

4. Broncho and hypostatic pneumonia in both lungs.

5. Numerous septic thrombi in both lungs and other viscera.

6. Very early arteriosclerosis.

7. Staphylococcic septicemia.

8. Parenchymatous changes in solid viscera due to toxemia.

NAVAL RESERVE

MEDICAL CORPS

PROMOTIONS, THIRD QUARTER, 1935

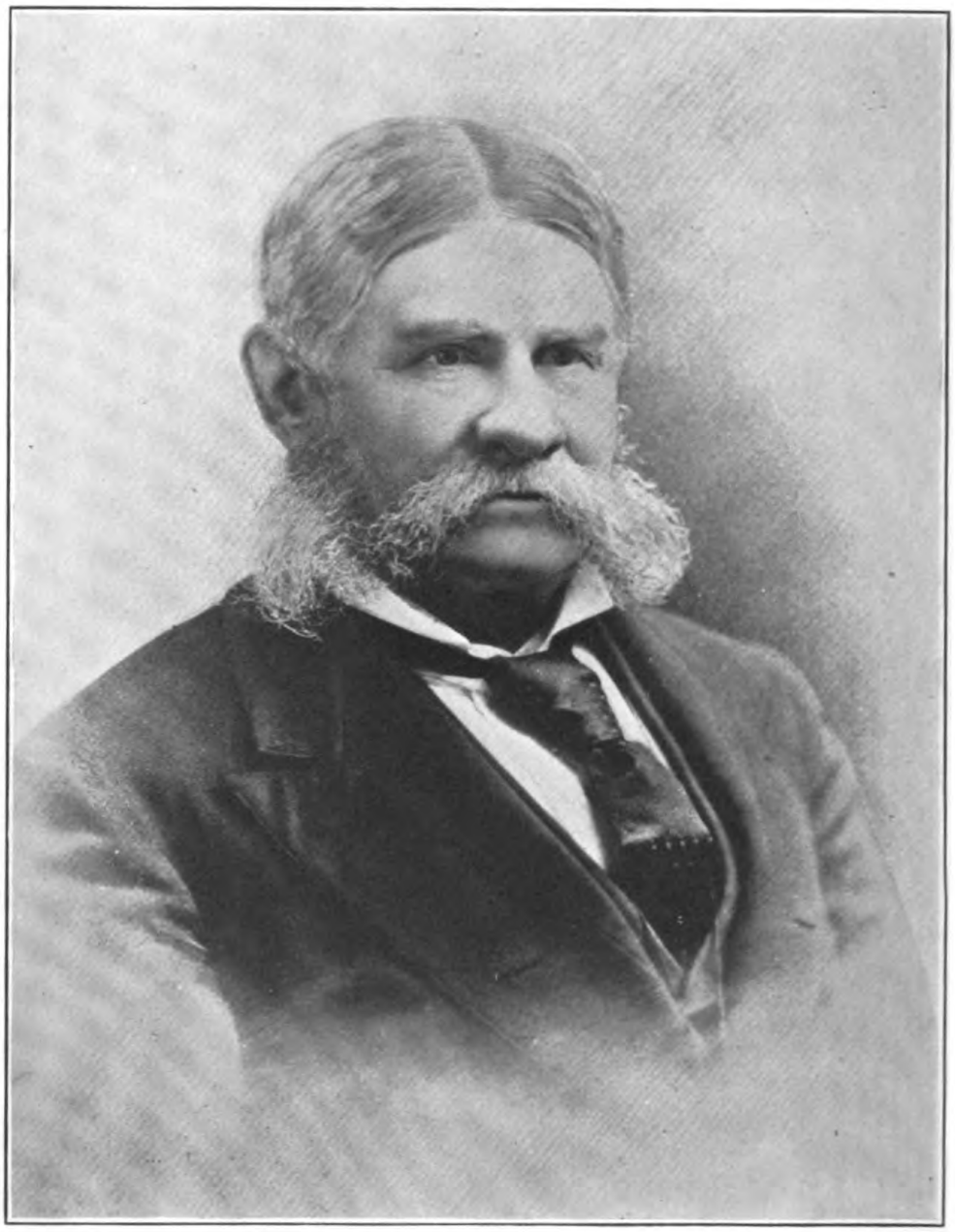
Lt. Clarence J. M. Hofer, MC–V(G), USNR. from Lt. (jg), MC–V(G), USNR. June 20, 1935.

Lt. Oscar H. Fulcher, MC–V(S), USNR. from Lt. (jg), MC–V(S), USNR. June 20, 1935.

Lt. Eric Dolph Pearson, MC–V(G), USNR. from Lt. (jg), MC–V(G), USNR. July 16, 1935.

MEMBERS OF THE NAVAL RESERVE AT THE MEETING OF THE AMERICAN COLLEGE OF SURGEONS AT SAN FRANCISCO AND OAKLAND, OCTOBER 28 TO NOVEMBER 1, 1935

A considerable number of members of the Naval Reserve who are Fellows of the American College of Surgeons were present at the Twenty-fifth Clinical Congress of the college at San Francisco and Oakland. The Surgeon General of the Navy, Rear Admiral P. S. Rossiter, Medical Corps, and the personnel officer of the Bureau of Medicine and Surgery, Capt. G. E. Thomas, Medical Corps, U. S. Navy, were present at this meeting. All reserve officers attending the meeting were the guests of Captain Thomas at an informal social gathering held at the Fairmont Hotel in San Francisco. The hospital ship *Relief* was in San Francisco harbor during the period of the meeting of the college. Many medical officers from the fleet and from shore stations on the Pacific coast availed themselves of the opportunity to attend the sessions.



JOSEPH BEALE

The eighth Chief of the Bureau of Medicine and Surgery and the fourth Surgeon General of the United States Navy.

NOTES AND COMMENTS

JOSEPH BEALE, SURGEON GENERAL OF THE NAVY, 1873–77

The fourth to hold the office of Surgeon General of the Navy and the eighth to be Chief of the Bureau of Medicine and Surgery was Joseph Beale, born on December 30, 1814. He was a native of Pennsylvania and appointed from that State as an assistant surgeon in the Navy on September 6, 1837. His first sea duty was on the old sailing sloop *John Adams* on which he served in the East India Squadron as it was then called, from 1839 to 1841. After service in the Home Squadron and the Naval Hospital, New York, he was again on the *John Adams*, this time on the coast of Africa engaged in the prevention of the slave trade. He was commissioned as a surgeon on April 19, 1848, the anniversary day of Lexington.

During the Civil War he was on the steam sloop *Susquehanna* in the West Gulf Blockading Squadron from 1861 to 1863. He thus participated in the attack on Forts Jackson and Phillip and the capture of New Orleans by Farragut. He was on duty at the Naval Hospital, Chelsea, Mass., from 1863 to 1865, and after the war fleet surgeon of the Asiatic Squadron. He was serving as president of the board of medical examiners when appointed Chief of the Bureau of Medicine and Surgery by President Grant in 1873.

Beale was one of six very able Surgeons General who followed in succession after the Civil War: J. M. Foltz, J. C. Palmer, Joseph Beale, William Grier, J. W. Taylor, and P. S. Wales. Under these men many important advances were made. The Annual Reports of the Surgeon General, begun in the sixties, were continued and improved. These reports have been of enormous value to public-health authorities and to life-insurance actuaries. They are unique in their standardization and accuracy and have been a real contribution to knowledge regarding morbidity and mortality trends.

Under Surgeon General Beale's administration, the naval hospital at Pensacola was constructed (1875).

The size and constitution of the Medical Corps in Beale's time, approximately 10 years after the Civil War and 10 years before the beginning of the steel Navy, is of interest. The number and distribution of medical officers in various ranks were:

Medical directors	15
Medical inspectors	15
Surgeons	50
Passed assistant surgeons	32
Assistant surgeons with rank of master	21
Assistant surgeons with rank of ensign	23
Total	156

Surgeon General Beale held office until February 2, 1877. His death occurred on September 23, 1889.

THE MISSION OF THE MEDICAL CORPS OF THE NAVY

The ideal of military tactics has always been the concentration of a superior force against an enemy. Napoleon declared that victory was with the biggest battalions. The Confederate cavalry leader, Forrest, is credited with the picturesque statement that victory was won by him who "got there fustest with the mostest." The mission of a military medical department, stated in its briefest form, is conservation of manpower, so as to enable the military leader to fulfill the mission expressed by Forrest. A slightly more elaborate description is as follows: (1) Proper procurement of manpower (careful recruiting); (2) conservation of manpower (care of the sick and wounded, and prevention of disease); (3) elimination of the physically unfit.

It is of interest that the peace-time mission of the medical department is almost identical with the war-time mission, in other words, to maintain a state of readiness for any emergency. An English naval medical officer of the present day has stated that the first duty of a surgeon of a man-of-war is "to render the ship an efficient fighting unit as far as lies in his power." Farenholt, of our own Medical Corps, has expressed the surgeon's primary duty on board ship as: "(1) Keeping the health of the personnel up to the highest efficiency; (2) arranging for the care of the wounded in, and more particularly after, action."

The statement of the mission of the Medical Corps of the Navy placed on the cover of this issue of the United States Medical Bulletin was elaborated by another medical officer of our Navy, the naval hygienist Gatewood, and he has expressed it in a brief and striking phrase: "*To keep as many men at as many guns as many days as possible.*"

THE NEW REVISION OF THE SUPPLY TABLE OF THE MEDICAL DEPARTMENT

The revised Supply Table has been distributed to all medical activities during the past year and the supply depots are now filling requisitions based upon it. This new revision does not contain any radical changes. It was designed rather to bring the Supply Table up to date by the inclusion in it of the numerous changes which have been made since the last printing.

The revision of the Supply Table of the Medical Department is a task of very considerable magnitude. It is necessary that it shall

have on it the items needed for the treatment of the sick and injured of the Navy, and that the most recent developments in medicine and surgery must be considered and made available if of value. Furthermore, the question of value and practicability of supply and use in times of national emergency is always a question that must be answered before an item is deleted or added. All the factors to be considered in regard to each item are not always easy to determine. Medical officers should submit suggestions and recommendations from time to time with reference to their experience. Criticisms, particularly along the following lines, are valuable:

(*a*) Were the supplies furnished during the year of good quantity and quality?

(*b*) Were they sufficient, under normal conditions?

(*c*) Were they delivered in the numbers as requested?

SOME COUGH MIXTURES

During this season of the year, when the acute respiratory diseases are usually on the center of the medical stage, a busy practitioner always finds plenty of need for cough mixtures. The following from various sources are published as furnishing some prescriptions of value.

While unanimity of opinion does not exist in regard to the matter there is a tendency to prefer cough mixtures containing alkaline citrates in the early dry irritative stage and to use expectorant mixtures in the exudative stage of respiratory infections. The essential feature of the expectorant mixture is its ammonium ion generally given as ammonium chloride, ammonium carbonate, or aromatic spirits of ammonia.

ALKALINE COUGH MIXTURES

℞
Sodii Citratis, ℥ss (15.0 gm).
Syr. Ipecacuanhae, ʒss (2.0 cc).
Succus Limonis, f℥i (30.0 cc).
Syr. Simplicis, f℥ss (15.0 cc).
Aquae, q. s. ℥iv (120.0 cc).
Sig.: Teaspoonful every three (3) hours.

℞
Sodii Citratis, ℥ss (15.0 gm).
Syr. Ipecacuanhae, ʒss (2.0 cc).
Syr. Limonis, ℥i (30.0 cc).
Aquae, q. s. ℥iv (120.0 cc).
Sig.: Teaspoonful every two to three (2 to 3) hours.

COMBINATION ALKALINE AND EXPECTORANT COUGH MIXTURE

℞
Potassii Citratis, ʒii (8.0 gm).
Syr. Ipecacuanhae, ʒss (2.0 cc).
Spt. Ammoniae Aromaticae.
Syr. Tolu, ää ℥ss (15.0 cc).
Aquae, q. s. ℥iii (90.0 cc).
Sig.: Teaspoonful every two to four (2 to 4) hours for cough.

EXPECTORANT COUGH MIXTURE

℞
Ammonii Chloridi, ʒii (8.0 gm).
Spt. Chloroformi, ℥i (4.0 cc).
Mist. Glycyrrhizae Comp., ℥ss (15.0 cc).
Syr. Pruni Virginianae, ℥i (30.0 cc).
Aquae, q. s. ℥iv (120.0 cc).
Sig.: Teaspoonful every two to three (2 to 3) hours.

SEDATIVE COUGH MIXTURES

℞
Ammonii Chloridi, gr. xxiv (1.6 gm).
Syr. Ipecacuanhae, f℥ss (2.0 cc).
Elix. Terpini Hydratis et Codeinae, q. s. f℥iii (90.0 cc).
Sig.: From ½ to 1 teaspoonful every three (3) hours.

℞
Sodii Citratis, ℥ss (15.0 gm).
Succus Limonis, f℥i (30.0 cc).
Tr. Opii Camphoratae, f℥ii (8.0 cc).
Syr. Ipecacuanhae, f℥ss (2.0 cc).
Syr. Zingiberis, q. s. f℥ii (60.0 cc).
Sig.: One teaspoonful *t. i. d.*

℞
Codeinae Sulphatis, gr. v (.30 gm).
Ammonii Chloridi, ʒiss (6.00 gm).
Syr. Acidi Citrici, f℥i (30.00 cc).
Aquae, q. s. f℥iv (120.00 cc).
Sig.: One teaspoonful *t. i. d.*

NEW VIEWPOINTS IN SURGERY

Conservatism has always been a feature of medicine. Hippocrates was a conservative, and the dependence on the healing powers of nature advocated by the Father of Medicine has been justly regarded by all great clinicians since as one of the fundamental concepts of the science and art of medicine. In the practice of surgery the conservative attitude has become more and more apparent.

There is a notable restriction in the number and particularly in the extent of operative procedures. The delay in opening empyemas, the limitation of operation in osteomyelitis and cellulitis, the decline in the excision of tubercular glands and joints, are all examples of these restrictions. A part of this conservatism is now to be found in the statement from studies of recent statistics that facial carbuncles with operation show a higher mortality than where nonoperative treatment has prevailed. Of course, there is always the doubt in statistical evidence regarding surgical conditions, especially that cases are parallel in character, and it is usually the less virulent infections that show early improvement and escape operation. The lack of deep fascia in the face to limit the spread of infection which thus has direct access to the muscles, the abundant venous supply, and the movement of the muscles of facial expression are the factors which make these cases so frequently fatal. In no other infection is the surgeon confronted with so important a problem in treatment as the necessity for a decision as to the opening of these infections, the best time to open, and the extent of the operative wound. Experience of generations extending back perhaps to primitive man has established the value of evacuation of pus. The relieving of pain and tension and the entry of fresh immune plasma into the formerly distended tissue spaces are undoubtedly favorable to the healing process and of assistance to nature in carrying this out. It is in the selection of the time and extent of operative interference that surgery is becoming more conservative.

FELLOWSHIP IN THE AMERICAN COLLEGE OF SURGEONS AND AMERICAN COLLEGE OF PHYSICIANS

For 1935 the Surgeon General has recommended the following for Fellowship in the American College of Surgeons:

Lt. Comdr. Albin H. Cecha, Medical Corps, United States Navy.
Lt. Comdr. George E. Mott, Medical Corps, United States Navy.
Lt. Joseph E. Evans, Medical Corps, United States Navy.

In the American College of Physicians, Comdr. Lyle J. Roberts, Medical Corps, United States Navy, has been recommended for

Fellowship, and Lt. George A. Cann, Medical Corps, United States Navy, for Associateship.

CONTRIBUTION TO THE HISTORY OF SYPHILIS

Two officers of the Medical Corps of the Navy have contributed extensively to the literature in connection with the history of syphilis. As is known to many, Rear Admiral C. S. Butler, Medical Corps, has been a strong advocate of the view that the disease is of Old World origin and was not, as some medical historians aver, unknown in Europe until after the discovery of America, the disease having been in their opinion like such well-known natural products as potatoes, maize, and tobacco, indigenous to the New World. Capt. R. C. Holcomb, Medical Corps, United States Navy, retired, has also recently upheld the view that the disease was known in Europe before the Columbian voyages. Many will recall his article in the October 1934 Bulletin, entitled "Christopher Columbus and the American Origin of Syphilis." He recently has contributed to the Medical Life an article on the antiquity of syphilis. He produces strong evidence to show that the numerous leprosaria of the Middle Ages were probably treating much syphilis. He believes that the references in the literature showing that much of the so-called "leprosy" was of venereal origin are so numerous that it is difficult to escape the conclusion that a venereal disease with constitutional symptoms existed at that time in Europe.

The history of this disease is one of great interest, containing many obscure places waiting for the scholar to explore. It is a credit to our Corps that two of our members have contributed extensively to the literature on this subject.

THE COMMON COLD

There is no escape from death and taxes and, from both common experience and recent studies, it would seem that colds should be added to these two other inevitable consequences of life. One of the most interesting surveys of the subject is one made by the Public Health Service (Collins and Grover) on the families of medical officers of the United States Army, Navy, and Public Health Service, and the faculty members of a number of universities. Nearly 3,000 persons were studied in a period of 33 months. A year's record was also kept on 1,872 college students in 1924. Semimonthly reports were made.

The records when analyzed show that the incidence of minor respiratory attacks was nearly two per person per year. About one-

half were reported as head colds, one-fourth as influenzal in character, and one-fourth as a combination of sore throat, bronchitis, and coryza. The students reported an incidence of three cases per person per year, with three-fourths of the cases described as coryza. Males and females showed apparently the same incidence rates. There is, however, a difference between the sexes in regard to certain symptoms at specific ages.

There are interesting differences in the seasonal incidence. Coryza reaches a peak in the fall, and influenzal-like attacks reached a peak in the late winter or early spring.

THE FORTY-THIRD ANNUAL MEETING OF THE ASSOCIATION OF MILITARY SURGEONS OF THE UNITED STATES

The forty-third annual meeting of the Association of Military Surgeons was held in New York City October 3–6, 1935. The headquarters was the Hotel Waldorf-Astoria, and all the principal meetings were held there. Outstanding medical men of the various Government services, including a number of national and international eminence, presented papers of much interest. The Surgeons General of the Army and Navy, Major General Reynolds and Rear Admiral Rossiter, were both present. There were scientific exhibits by the medical departments of the Army, Navy, Public Health Service, and Veterans' Administration. In addition there was a fine exhibit by the College of Pharmacy of Columbia University. There were technical exhibits by many drug, biological, surgical instrument, and pharmaceutical houses. The usual banquet and other social events were held, and entertainments for the ladies included a visit to West Point and trips to Radio City and Rockefeller Center. The attendance was as large as any in recent years, and the meeting as a whole was one of the most successful held by the association. The Reserve officers and National Guard were particularly well represented. The new president is Dr. Charles M. Griffiths, Medical Director of the United States Veterans' Administration. The first vice president, who will succeed him in October of 1936, is the Surgeon General of the Navy, Rear Admiral P. S. Rossiter, Medical Corps, United States Navy.

BOOK NOTICES

Publishers submitting books for review are requested to address them as follows:

The EDITOR, UNITED STATES NAVAL MEDICAL BULLETIN,
Bureau of Medicine and Surgery, Navy Department,
Washington, D. C.

(For review.)

PRINCIPLES AND PRACTICE OF MEDICINE, by *Sir William Osler* and *Thomas McCrae, M. D.;* 1,245 pages, 38 illustrations. New twelfth edition. D. Appleton-Century Co., New York and London. Price $8.50.

It is more than 40 years ago that the first edition of Osler's Practice appeared. The germ theory of disease was yet new. In that first edition Osler says that Eberth, Koch, and Gaffky have shown that there is a specific microorganism associated with typhoid fever. In the second edition he reported a new feature, Behring's discovery of diptheria antitoxin. He strongly recommended its use. Mild cases were to receive 1,000 units and severe cases 3,000 units of antitoxin. The successive editions of Osler continue to record the medical progress that brought in endocrines, vitamins, allergy, basal metabolism, electrocardiography, salvarsan, complement fixation, and blood chemistry.

This twelfth edition is from the hand of the late Thomas McCrae, long associated with Osler and brother of the medical poet who wrote "In Flander's Field." He has revised every edition of Osler since the ninth, which appeared just after Sir William's death. Without altering the style that made this so great a textbook, the book from which the greater number of our present generation of English-speaking physicians learned the practice of medicine, Dr. McCrae has brought the text completely up to date. Some of the new subjects are prolyvalent sera in pneumonia, bacteriophage therapy, all methods of hyperpyrexia and their value, the newer therapy of amebiasis, malaria, syphilis, degenerative myelitis, cestode diseases, and the latest ideas regarding the use of such important therapeutic agents as ultraviolet light, X-ray, insulin, and dextrose. Many new subjects have been added in order that nothing current in internal medicine might be omitted, and numerous parts of the book have been entirely rewritten.

Osler was one of the great masters of style among scientists, and for clarity and precision stands beside such men as Darwin, Huxley, LeConte, Asa Gray, and other great writers of scientific prose. To rewrite anything he has written is therefore a difficult task. Dr. McCrae, however, has done it well. There is one passage later editors of Osler have left out, and which is deplored by all lovers of the old textbook. This is the description of the rash of measles quoted from Sydenham's original description.

Osler's Practice of Medicine has become an institution among medical men rather than a mere book. As long as splendidly revised new editions such as this are made available from time to time it will continue to rank with Gray's Anatomy as one of the indispensable texts of medical literature.

A Textbook of Histology, *by Alexander A. Maximow, Late Professor of Anatomy, University of Chicago; and William Bloom, Associate Professor of Anatomy, University of Chicago.* Completely revised with 662 pages with 530 illustrations, some in colors. W. B. Saunders Company, Philadelphia and London, 1934. Price, $7.

The late Professor Maximow was not only a great histologist, he was also a talented artist and his histological drawings are such as to place his work beside the many great anatomical illustrators of the eighteenth and nineteenth centuries. The text has been carefully revised and Professor Bloom has made this edition an outstanding work on the subject.

The Autonomic Nervous System, Anatomy, Physiology, and Surgical Treatment, *by James C. White, M. D., Assistant Professor of Surgery, Harvard Medical School.* 386 pages, illustrated. The Macmillan Co., New York. $7.

This is a fine monograph on the treatment of disease by surgery directed at the sympathetic nervous system. A most valuable feature for the surgeon is the carefully tabulated results and evaluations of results with this form of operative treatment.

Diagnostic Methods in Internal Medicine, *by S. A. Loewenberg, M. D., Associate Professor of Medicine, Jefferson Medical College.* 1,032 pages, 547 illustrations, some in color. Third revised edition. 1935. F. A. Davis Co., Philadelphia. $7.50.

Dr. Loewenberg's book is well known for its excellence and this new edition is one of which both author and publisher may well be proud. Well written, well illustrated, and handsomely bound, it represents an excellent work for either the general practitioner or the specialist. The section on the interpretation of laboratory findings is a useful feature that will appeal to every medical man.

MICROBIOLOGY AND ELEMENTARY PATHOLOGY, *by Charles G. Sinclair, Major, Medical Corps, U. S. Army.* 377 pages, 102 illustrations, some in color. Second edition, 1935. F. A. Davis Co., Philadelphia. $2.50.

Excellent text intended primarily for nurses.

MANUAL OF CLINICAL LABORATORY METHODS, *by Pauline S. Dimmitt, Ph. G.* 156 pages, 36 engravings, including 7 full-page color plates. F. A. Davis Co., Philadelphia, 1935. $2.

Elementary manual for nurses and technicians.

A TEXTBOOK OF STERILIZATION, *by Weeden B. Underwood, B S. in E. E., Research Engineer, American Sterilizer Co.* 124 pages, illustrated. American Sterilizer Co., Erie, Pa., 1935. $2.

This is a well-written manual covering the physics and engineering features of various sterilizers and autoclaves. It should be a useful reference book for surgeons and bacteriologists and hospital executives particularly.

THE AMERICAN ILLUSTRATED MEDICAL DICTIONARY, *by W. A. Newman Dorland,* 17th edition, 1935. W. B. Saunders Co., Philadelphia. Price $7.50.

This is the new revised edition, brought entirely up to date, of the well-known medical dictionary that has for years been on the regular book list of the Supply Table of the Medical Department of the Navy. Neither in subject matter nor in binding has the book declined but still maintains its high position as a medical man's reference book.

A TEXTBOOK OF ANATOMY AND PHYSIOLOGY, *by J. F. Williams, M. D.,* 606 pages, illustrated. Fifth edition. W. B. Saunders Co., Philadelphia, 1935.

A new edition of this excellent textbook for nurses and for schools.

DENTAL RADIOLOGY HANDBOOK, *by Houghton Holliday, B. A., D. D. S.* 120 pages, illustrated. The Macmillan Co., New York. Price $2.

Essentials of dental radiology in a simple and compact form.

LIVING ALONG WITH HEART DISEASE, *by Louis Levin, M. D.* 126 pages. The Macmillan Co., New York. Price $1.50.

An excellent little manual for the layman on the plan of Joslin's Diabetic Manual and intended as an aid to the treatment prescribed by insuring more intelligent cooperation of the patient.

DIAGNOSIS AND TREATMENT OF SKIN DISEASES, *by J. H. Swartz, M. D., and Margaret G. Reilly, R. N.* 316 pages, illustrated. The Macmillan Co., New York. $3.50.

A manual combining the knowledge of a specialist with the detailed directions for carrying out treatment by a nurse who has been in the Skin Clinic of Massachusetts General Hospital for a number of years.

SURGICAL DISEASES OF THE CHEST, *by Evarts Ambrose Graham, A. B., M. D., F. A. C. S. Professor of Surgery, Washington University School of Medicine, St. Louis; Jacob Jesse Singer, M. D., F. A. C. P., Associate Professor of Clinical Medicine, Washington University School of Medicine, St. Louis; and Harry C. Ballon, M. D., C. M., F. A. C. S.* Lea & Febiger, Philadelphia, 1935. Price, $15.

In this book the authors have correlated and presented a vast amount of information relative to all diseases of the thorax which are amendable to surgical treatment. Not only is the brilliant original work of the authors presented but full recognition is paid to the work of others in this field. A voluminous bibliography at the end of each chapter affords reference to the literature on its subject. This book should be equally as valuable to the internist as to the surgeon because of its comprehensive treatment of all morbid processes of the thorax and its contained structures.

The section on empyema is especially valuable. It presents the results and knowledge gained by a wide experience in the treatment of this condition.

The surgical treatment of pulmonary tuberculosis is extensively presented in all its phases, including operations on the phrenic nerve, thoracoplasthy, pneumothorax, and oleothorax.

SYNOPSIS OF REGIONAL ANATOMY, *by T. B. Johnston, M. B., Ch. B., Professor of Anatomy, University of London, Guy's Hospital Medical School.* 483 pages, illustrated. Third edition. Lea and Febiger, Philadelphia, $4.50.

This is a carefully written and complete compend of anatomy.

DISEASES OF THE NERVOUS SYSTEM, *by Smith Ely Jelleffe, M. D., Ph. D., and William S. White, M. D.* 1,175 pages, 497 engravings, 13 plates. Sixth edition, 1935. Lea and Febiger, Philadelphia, $9.50.

This is a complete revision of this standard text, rewritten and enlarged. The sections on the endocrinopathies, and the vegetative nervous system, have included the latest knowledge on these subjects. The authors have presented a view of man as a unit in the universe, subject to its laws and engaged in the transformation and delivery of energy. The difficulties which he encounters result in the disturbances which end in mental and nervous disease. It is a view of man and his troubles similar to that expressed by the old Greek tragedies and one the late Mr. Thomas Hardy utilized in his novels. In this conception of mental and nervous disorders this book throws light from a somewhat different angle and lights up many dark areas in that field of medicine.

DISEASES OF THE THYROID GLAND, *by Arthur E. Hertzler, M. D., Professor of Surgery, University of Kansas.* 348 pages, illustrated. Third edition, 1935. The C. V. Mosby Co., St. Louis. $7.50.

This is a fine monograph by one of the leading American surgeons and gives a well-rounded view of the diagnosis and treatment

of the various pathologic conditions of the thyroid. There is an excellent chapter by Dr. Victor E. Chesky on the hospital management of goiter patients.

A TEXTBOOK OF LABORATORY DIAGNOSIS, *by Edwin E. Osgood, M. A., M. D., Assistant Professor of Medicine and Director of Laboratories, University of Oregon School of Medicine.* 585 pages, illustrated. Second edition. P. Blakiston's Son & Co., Inc., Philadelphia. $6.

This is a revised edition of this splendid laboratory manual. It has probably the most complete index of any medical book in print, an exceedingly useful feature in a book of this type. The publishers state that the binding is water resisting and vermin proof.

PREVENTIVE MEDICINE AND HYGIENE, *by Milton J. Rosenau.* Sixth Edition. D. Appleton-Century Company, New York and London. $10.

A review of this widely known and accepted text and reference book must partake of the nature of an announcement of a new edition rather than a comprehensive discussion of its merits and shortcomings. The ever-widening scope of preventive medicine is shown by the inclusion in this edition of information on contraception, maternal mortality, heart disease, diabetes, ringworm, snake poisoning, and psittacosis.

ARTHRITIS AND RHEUMATOID CONDITIONS. THEIR NATURE AND TREATMENT, *by Ralph Pemberton, M. D., F. A. C. P., Professor of Medicine, Graduate School of Medicine, University of Pennsylvania.* 455 pages, 69 engravings, and a colored plate. Second edition. 1935. Lea and Febiger, Philadelphia. $5.50.

This is a carefully prepared monograph on this group of conditions whose etiology and treatment contain so many puzzling problems.

ANESTHESIA IN DENTAL SURGERY, *by Sterling V. Mead, D. D. S., M. S.* 482 pages, 144 illustrations. The C. V. Mosby Co., St. Louis. $6.50.

A complete, well written, and well illustrated text on this subject.

APPLIED ORTHODONTICS, *by James D. McCoy, M. S., D. D. S., Associate Clinical Professor of Oral Surgery, University of Southern California.* 336 pages, 220 engravings. Fourth edition. 1935. Lea and Febiger, Philadelphia. $4.50.

Revised edition of a well-used textbook. Emphasis is placed on slight forces applied over long periods as the ideal in treatment.

A TEXTBOOK OF CLINICAL NEUROLOGY, *by Israel S. Wechsler, M. D., Professor of Clinical Neurology, Columbia University.* 826 pages, illustrated. Third edition. W. B. Saunders Co., Philadelphia. $7.

This is a new and completely reset edition of this fine textbook. There is an interesting introduction to the history of neurology that is remarkably fine and one of the few in the English language.

A TEXTBOOK OF GENERAL BACTERIOLOGY, *by Edwin O. Jordan, Ph. D., Professor of Bacteriology at the University of Chicago and Rush Medical College.* Illustrated. 11th edition. 1935. W. B. Saunders Co., Philadelphia. Price, $6

This is a new edition of a classic text brought completely up to date. Jordan's book has the great advantage of being a general work not confined only to pathogenic organisms. The latter are covered, however, in a very complete manner. The filterable viruses also receive considerable space.

CLINICAL DIAGNOSIS BY LABORATORY METHODS, *by James C. Todd, M. D., and Arthur H. Sanford, M. D.* 792 pages, 370 illustrations, 29 in color. 8th edition. W. B. Saunders Co., Philadelphia. 1935. Price, $6

This is a new and thoroughly revised edition of this splendid manual of clinical pathology long well known to our profession both here and in Canada and England. The Index of Laboratory Findings in Important Diseases is alone worth the price of the book. Nowhere can be found a more succinct account of the laboratory characteristics of the principal diseases.

CLINICAL ATLAS OF BLOOD DISEASES, *by A. Piney, M. D., and Stanley Wyard, M. D.* 110 pages and 38 illustrations, 34 in color. P. Blakiston's Son & Co., Inc., Philadelphia, 1935. Price, $4

A new edition of this convenient little hand atlas of blood diseases. One feature is an informative little glossary covering blood pathology.

A TEXTBOOK OF FRACTURES AND DISLOCATIONS, *by Kellogg Speed, M. D., Professor of Clinical Surgery, Rush Medical College of the University of Chicago.* Third edition, thoroughly revised 1,000 pages, 1,042 engravings. Lea and Febiger, Philadelphia, 1935

This is a new edition of a book well known to medical officers of the Navy, as it has been for a number of years on the standard book list of the Supply Table. The previous edition was published in 1928.

DISORDERS OF THE BLOOD, *by L. E. H. Whitby, M. D., and C. J. C. Britton, M. D.* 543 pages, illustrated including 8 colored plates. P. Blakiston's Son & Co., Inc., Philadelphia. 1935. Price $7

This is a book by two eminent English haemotologists and compresses into a reasonable compass the essentials of present knowledge of this subject. A feature is the emphasis on the relative rarity of primary disease of the hæmopoietic system and the fact that changes in the peripheral blood are usually a symptom of some other system of the body. These changes in the blood are clearly and adequately described.

APHASIA, *by Theodore Weisenburg, M. D., and Katherine E. McBride, Ph. D.* 634 pages. The Commonwealth Fund, New York. 1935. Price $5

A study of aphasia largely from the psychological side. Elaborate studies of cases are given, with the results of numerous tests used in the psychological laboratory.

THE ANATOMY OF THE NERVOUS SYSTEM, *by Stephen W. Ranson, M. D., Professor of Neurology and Director of Neurological Institute, Northwestern University Medical School.* Fifth edition. 501 pages, 381 illustrations, some in color. W. B. Saunders Co., Philadelphia and London. 1935. Price $6.50

A new and revised edition of a work extensively used as a textbook in many medical schools. Although an anatomical work, relation to function is constantly linked to structure.

CLINICAL PARASITOLOGY AND TROPICAL MEDICINE, *by Damaso de Rivas, M. D., Professor of Parasitology, Graduate School of Medicine, University of Pennsylvania, and Carlos T. de Rivas, M. D., Pathologist of Santo Tomas Hospital, Panama.* 367 pages, 144 engravings and a colored plate. Lea and Febiger, Philadelphia. 1935. Price $5

This excellent work aims to be a manual of tropical medicine not too large and bulky and yet comprehensive enough to give all essentials. The authors have succeeded well in their purpose.

COLLECTED PAPERS OF THE MAYO CLINIC AND THE MAYO FOUNDATION, *edited by Richard M. Hewitt, B. A., M. A., M. D., and Lloyd G. Potter.* Vol. XXVI, 1934, published May 1935. W. B. Saunders Co., Philadelphia. Price $11.50.

These collections from the Mayo Clinic and Foundation, representing as they do the papers and reports originating at one of the great medical centers of the world, contain a vast amount of important material on the latest developments in medicine and surgery. Nearly every field is touched, the subjects ranging from skin condition to disturbances of the functions of the parathyroids and the presence of arsenic in the brain. Surgery and X-ray are not forgotten and public health problems are included. William Mayo, Charles Mayo, and E. Starr Judd all have contributed papers. One by Charles Mayo on the necessity for the medical profession to study the alcohol problem in a scientific manner is of particular interest, as well as another article by him which paradoxically enough is on "Some of the Properties of Water." The Mayo Clinic volumes are so important that every active practitioner should have them in his library or have access to them in some institutional library.

FOOD AND BEVERAGE ANALYSES, *by Milton A. Bridges, B. S., M. D., Director of Medicine, Department of Correction Hospitals, New York.* 246 pages, limp binding. Lea & Febiger, Philadelphia, 1935. Price $3.50.

Dr. Bridges has an excellent and original motto on the flyleaf, which is an excuse for all professional books at least:

Now go, write it before them in a table, and note it in a book, that it may be for the time to come for ever and ever.—Isaiah 30:8.

He has well carried out the instructions of this wise old prophet, and his manual gives a complete analysis of practically all foods and beverages, including the vitamin and mineral contents.

THE DIVISION OF PREVENTIVE MEDICINE

S. S. Cook, Commander, Medical Corps, United States Navy, in charge

TOXIC EFFECTS OF ARSENICAL COMPOUNDS EMPLOYED IN THE TREATMENT OF DISEASE IN THE UNITED STATES NAVY, 1934

By S. S. Cook, Commander, Medical Corps, United States Navy, and H. D. Campbell, Chief Pharmacist's Mate, United States Navy

Since November 1924 medical officers of the Navy have been required to make monthly reports of the number of doses of arsenicals administered and a separate account of each case in which ill effects are noted. During the 10 years, 1925–34, in which this information has been compiled 966,767 doses of arsenicals have been administered and 741 reactions have been reported.

Previous articles dealing with the information obtained from these reports were published in the September 1925, January 1927, January 1929, July 1930, October 1931, October 1932, April 1933, October 1933, October 1934, January 1935, and October 1935 numbers of the United States Naval Medical Bulletin. Cases of arsenical dermatitis occurring during the year 1934 were published in the October 1935 number of the United States Naval Medical Bulletin. The present article deals with all cases, except arsenical dermatitis, which were reported during the year 1934. In this installment are also presented some comparative figures from the experience of previous years.

TABLE 1.—*Arsenical reactions 1934*

Classification	Neoarsphenamine and Sulpharsphenamine reactions			
	Mild	Severe	Fatal	Total
Vasomotor phenomena	41	7	0	48
Arsenical dermatitis [1]	22	20	1	43
Table reactions	15	0	0	15
Blood dyscrasias	3	4	1	8
Jarisch-Herxheimer	3	2	0	5
Liver damage (jaundice)	1	2	0	3
Gastro-intestinal	3	0	0	3
Arsenical neuritis	0	1	0	1
Hemorrhagic encephalitis	0	0	1	1
Total	88	36	3	127

[1] Case histories were published in the October 1935 number of the bulletin. Included in the above table are reactions caused by sulpharsphenamine which were classified as follows: Vasomotor phenomena, 6 mild and 2 severe; table reactions, 2 mild. Total, 10 reactions.

TABLE 2.—*Arsenicals administered during the year 1934 for all diseases including syphilis*

Drug	Dose				
	0.9 to 3.0 grams	0.9 gram	0.6 to 0.9 gram	Less than 0.6 gram	Total
Acetarsone:					
Navy	0	0	0	0	0
All others	0	0	0	26	26
Arsphenamine:					
Navy	0	0	81	2,773	2,854
All others	0	0	0	11	11
Bismarsen:					
Navy	0	0	0	95	95
All others	0	0	1	78	79
Neoarsphenamine:					
Navy	0	461	35,603	61,868	97,932
All others	0	4	3,586	16,277	19,867
Silver arsphenamine:					
Navy	0	0	0	0	0
All others	0	0	0	3	3
Sulpharsphenamine:					
Navy	0	0	64	1,661	1,725
All others	0	0	1	815	816
Tryparsamide:					
Navy	2,579	0	0	0	2,579
All others	401	0	0	0	401
Total	2,980	465	39,336	83,607	126,388

TABLE 3.—*Arsenicals administered during the 3-year period, 1932–34, for all diseases including syphilis*

Drug	Dose				
	0.9 gram to 0.6 gram	0.9 gram	0.6 gram to 0.9 gram	Less than 0.6 gram	Total
Acetarsone:					
Navy	0	0	0	0	0
All others	0	0	76	729	805
Arsphenamine:					
Navy	0	0	81	2,993	3,074
All others	0	0	0	54	54
Bismarsen:					
Navy	0	0	0	117	117
All others	0	0	1	140	141
Neoarsphenamine:					
Navy	0	4,475	133,974	180,934	319,383
All others	0	373	17,159	47,914	65,446
Silver arsphenamine:					
Navy	0	0	0	47	47
All others	0	0	0	148	148
Sulpharsphenamine:					
Navy	0	18	96	3,028	3,142
All others	0	3	10	4,921	4,934
Tryparsamide:					
Navy	7,270	0	0	10	7,280
All others	7,157	0	0	5	7,162
Total	14,427	4,869	151,397	241,040	411,733

TABLE 4.—*Deaths and severe reactions following the administration of 880,490 doses neoarsphenamine, 1925–34. Ratio of deaths and severe reactions to doses*

Classification	Deaths		Severe reactions		Deaths and severe reactions	
	Number	Ratio to doses 1 to —	Number	Ratio to doses 1 to —	Number	Ratio to doses 1 to —
Hemorrhagic encephalitis	14	62,892	0	----------	14	62,892
Arsenical dermatitis	8	110,061	137	6,427	145	6,072
Vasomotor phenomena	6	146,748	54	16,305	60	14,675
Blood dyscrasias	4	220,123	11	80,045	15	58,699
Acute renal damage	2	440,245	4	220,123	6	146,748
Acute yellow atrophy of the liver	2	440,245	0	----------	2	440,245
Liver damage (jaundice)	0	----------	12	73,374	12	73,374
Jarisch-Herxheimer	0	----------	2	440,245	2	440,245
Polyneuritis	0	----------	1	880,490	1	880,490
Border-line hemorrhagic encephalitis [1]	0	----------	1	880,490	1	880,490
Arsenical neuritis [2]	0	----------	1	880,490	1	880,490
Total	36	24,458	223	3,948	259	3,400

[1] First classified during the year 1933.
[2] First classified during the year 1934.

TABLE 5.—*Deaths following administration of arsenical compounds, 1919–34*

Year	Arsphenamine	Neoarsphenamine	Kind not specified	Total
1919	2	0	1	3
1920	1	1	0	2
1921	3	1	0	4
1922	0	4	0	4
1923	0	1	0	1
1924	1	2	0	3
1925	0	2	0	2
1926	0	4	0	4
1927	1	4	0	5
1928	0	6	0	6
1929	0	3	0	3
1930	0	3	0	3
1931	0	0	0	0
1932	0	4	0	4
1933	0	7	0	7
1934	0	3	0	3
Total	8	45	1	54

NUMBER OF PERSONS TREATED FOR SYPHILIS AND FOR OTHER DISEASES

On December 31 of each year each activity records and reports to the Bureau of Medicine and Surgery, on NMS Form A, the number of persons in that command who have a history of syphilis, and the number of those in the command who were treated during the year with an arsenical compound, heavy metal, or other treatment. The census also requires the recording and reporting of the number of persons who were treated during the year with an arsenical compound for a disease other than syphilis. This census does not take into account those individuals who left the service during the year.

In the table which follows, treatment data have been separated into that given to active service personnel and that given to all

others. The term "all others" includes Veterans' Administration patients, dependents of naval personnel, retired naval personnel, and native populations of insular possessions.

TABLE 6.—*Syphilis and arsenicals, United States Navy, 1934*

	United States Navy	All others	Total (persons)
Strength, Dec. 31, 1934	111,840	----------	111,840
Syphilis census, Dec. 31, 1934	15,039	----------	15,039
1. Number of persons treated for syphilis with:			
(a) Arsenicals:			
Arsphenamine	151	42	139
Bismarsen	10	20	30
Neoarsphenamine	7,071	967	8,038
Silver arsphenamine	0	1	1
Sulpharsphenamine	239	19	258
Tryparsamide	104	68	172
Total	7,575	1,117	8,692
(b) Other treatment:			
Bismuth compounds	5,913	1,011	6,924
Mercury compounds	1,438	194	1,632
Potassium iodide	380	108	488
Mixed treatment (specific mixture, etc.)	194	24	218
Total	7,925	1,337	9,262
Total of (a) and (b)	15,500	2,454	17,954
2. Number of persons treated for other diseases:			
Arsphenamine	1	0	1
Neoarsphenamine	191	3,036	3,227
Sulpharsphenamine	0	54	54
Fowler's solution	98	0	98
Bismuth compounds	1	88	89
Mercury compounds	0	4	4
Total	291	3,182	3,473
Grand total	15,791	5,636	21,427

In table 6 it will be noted that 291 service personnel and 3,182 non-service personnel were treated for diseases other than syphilis during the year 1934.

Of the 291 naval personnel, 253 were treated for Vincent's infection, 17 for acne vulgaris, 1 for yaws, and 20 for other diseases.

Of the 3,182 persons in the group "all others", 3,143 were treated for yaws, 31 for Vincent's infection, and 8 for other conditions.

HEMORRHAGIC ENCEPHALITIS

Ratio of deaths to persons treated for syphilis with arsenical compounds, 1931, 1932, 1933, 1934

	Number of persons	Deaths	Ratio 1 to—
1931	15,763	0	----------
1932	12,245	2	6,123
1933	9,378	1	9,378
1934	8,692	1	8,692
Total	46,078	4	11,520

In comparing deaths from hemorrhagic encephalitis with the number of doses of neoarsphenamine administered, all diseases for which this compound was given are considered.

	Number of doses of neoarsphenamine	Deaths	Ratio 1 to—
1931	95,444	0	
1932	128,540	2	64,270
1933	138,490	1	138,490
1934	117,799	1	117,799
1925-34	880,490	14	62,892

During the 10-year period, 1925–34, there were 33,527 doses of arsphenamine administered with 1 death from hemorrhagic encephalitis. In this same period 34,205 doses of tryparsamide and 17,197 doses of sulpharsphenamine were given with no deaths.

Neoarsphenamine.—(1—1934) A patient had been given a diagnosis of syphilis because of positive clinical and serological findings. Arsenical treatment was started on April 14, 1934, with a 0.3 gram injection of neoarsphenamine. On April 21 he was given 0.45 gram and on April 28, May 5 and 12, 0.6-gram injections, an average of 88 milligrams per day. As concurrent treatment he was given 1 cubic centimeter intramuscular injections of bismoid on April 10, 13, 17, 20, 24, 27, May 1, 4, 8, and 11, 1934.

At 10 a. m. May 12 the patient received his fifth injection of neoarsphenamine. Immediately after the injection he was given 1 gram of sodium thiosulphate intravenously. About 3 minutes later he complained of nausea and dizziness and went into a convulsive state, which lasted about 2 minutes. He regained consciousness, answered a few questions, and vomited, bringing up a small amount of mucoid material. He was placed in bed and given 1 cubic centimeter of adrenalin (1–1,000) subcutaneously. Convulsive seizures occurred every few minutes with various patterns of cerebral irritations noted in the different seizures. Sodium amytal was given by rectum with some apparent benefit. The patient became unconscious after the second convulsion. Following the amytal the seizures were about 1 hour apart.

Laboratory reports.—Urinalysis prior to injection of neoarsphenamine was negative for albumin. Dickens' test on catheterized specimen, 3 plus arsenic elimination. Urine: Light amber; specific gravity, 1.014; sugar and albumin, negative; microscopical, negative. Blood: Red blood count, 5,110,000; white blood count, 28,000; differential, juveniles, 2; band forms, 12; segmented, 64; lymphocytes, 14; eosinophiles, 8.

Treatment.—

10:20 a. m. Sodium thiosulphate, 1 gram intravenously.

11 a. m. Adrenalin (1–1000), 1 cubic centimeter intramuscularly.

11:20 a. m. Morphine sulphate one-fourth grain and atrophine sulphate one one hundred-and-fiftieth grain.

12:00 noon. Adrenalin (1–1000), 1 cubic centimeter intramuscularly.

12:10 p. m. Sodium thiosulphate, 1 gram intravenously.

12:30 p. m. Sodium amytal, grains V1, by rectum.

1:30 p. m. Sodium amytal, grains V1, by rectum.

1:50 p. m. Glucose (50 percent solution), 50 cubic centimeters intravenously.

3:15 p. m. Temperature 104.6 degrees by axilla. Patient comatose. Stertorous respirations. Pulse 98, respirations 36. Blood pressure 146/88.

3:42 p. m. Convulsion, lasting a minute.

3:45 p. m. Pallor and cyanosis. Pulse too rapid to count. Respirations 20 per minute. Adrenalin (1–1000), 1 cubic centimeter intramuscularly.

4 p. m. Patient pronounced dead, 6 hours after the injection of neoarsphenamine.

Autopsy findings.—Encephalitis acute; congestion and edema of brain and lungs; congestion of spleen and kidneys; submucous petechial hemorrhages of small intestines.

LIVER DAMAGE

Neoarsphenamine.—(114—1934) Two reactions occurred in the same individual.

A patient whose diagnosis of syphilis was established on June 22, 1934, began treatment on June 28, when he received 0.45 gram of neoarsphenamine. This was followed by 0.6 gram injections on July 12 and July 19. The last dose was dissolved in 7 cubic centimeters of sterile distilled water and injected in 2 minutes. The patient received 1 cubic centimeter intramuscular injections of bismosol on June 29, July 3, 6, 10, 12, 17, 20, 24, August 10, 14, 17, 21, and 24, 1934.

Two days after the last injection of neoarsphenamine the patient noticed loss of appetite, a sense of oppression after eating, and a sense of weight in his epigastrium. Everything tasted badly. He was nauseated but did not vomit. He had slight fever, eructations of gas, and occasional feeling of distention. He had no definite abdominal pain. He complained of extreme weakness. His urine was dark brown and positive for bile. There was a faint icteroid tint of sclerae, but the jaundice appeared more clearly on mucous

membranes and skin. He had slight tenderness over gall bladder. His sclerae developed a deep yellow color. Icterus index 18. Blood: White blood count, 6,000; hemoglobin, 80 percent; bands, 2; segmented, 54; lymphocytes, 39; monocytes, 5. The sclerae were clear, his skin was normal, and the urine was free of bile. On August 7, 17 days after onset, he was returned to duty with no symptoms.

On August 9, he was given a 0.3-gram injection of neoarsphenamine. Following the injection he felt feverish, had loss of appetite, and developed itching of the skin. Recurrence of his cholangitis was noted. Exfoliative dermatitis developed with recovery in 110 days. (This reaction is described in the October 1935 Bulletin under "Case 115—1934.")

(116—1934) A patient in whom the diagnosis of syphilis was established on August 9, 1932, received 26 injections of neoarsphenamine, 3 injections of sulpharsphenamine, 27 injections of bismuth, and 1 course of mixed treatment between the dates of August 20, 1932, and July 1, 1934.

A fourth course of arsenicals was started on July 14, 1934, when the patient received 0.3 gram of neoarsphenamine. On July 28 and August 11 he received 0.3 gram; on August 19, 0.45 gram; and on September 1, 0.3 gram dissolved in 5 cubic centimeters of sterile distilled water and injected in approximately 2 minutes. About 30 minutes later he vomited. On September 3 there was some puffiness of the face with slight redness of the skin of the chest and neck. Temperature 102° F. A physical examination was negative, except for slight pain on pressure over the epigastrium. Three days later he developed jaundice. Nausea and vomiting persisted for 5 days. His temperature gradually returned to normal. The patient was returned to duty fully recovered in 31 days.

(117—1934) A patient developed hypertrophied tonsils with grayish ulceration. Dark field examination of ulcerations on tonsils was positive for *Treponema pallidum* on June 19, 1933. Kahn blood tests were 4 plus on June 19 and July 6. Treatment was immediately instituted, and by November 21 he had received a total of 27 injections of neoarsphenamine and 29 injections of bismuth.

The patient reported for arsenical treatment on December 30, 1933, but his urine showed 2 plus albumin and the injection was withheld. On January 5, 1934, his urine was 1 plus for albumin. The next day he began a fourth course of arsenical treatment, receiving a 0.3 gram injection of neoarsphenamine. The dose was dissolved in 10 cubic centimeters of water and given in 2½ minutes. One hour later he complained of slight nausea. He was given 1 gram of sodium thiosulphate intravenously and went on liberty in the afternoon. Four days later, January 10, he showed a moderate icterus with icterus index of 80.

January 11. Temperature, 99; pulse, 50; respirations, 18. Blood: Red blood count, 4,500,000; white blood count, 6,200; hemoglobin, 85 percent; blood platelets, 199,000; reticulocytes, 1.8 percent; urine, 2 plus albumin.

January 15. Temperature ranged from 99° to 100° F. Icterus index 86. No complaints.

January 17. Urine showed many granular casts and 4 plus albumin.

January 19 and 22. Biliary drainage done but results were not satisfactory. An X-ray showed no evidence of gallstones. On January 19, temperature, pulse, and respirations became normal but icterus remained intense.

January 28. Blood: Red blood count, 4,420,000; white blood count, 6,100; hemoglobin, 88 percent; blood platelets, 229,000; recticulocytes, 5.5 percent; urine contains 4 plus albumin with casts.

January 30. Icterus index 220.

The generalized icterus gradually subsided, and on March 16, 1934, he was considered fully recovered after 65 sick days.

BLOOD DYSCRASIAS

Neoarsphenamine.—(106—1934) The patient contracted syphilis in February 1924, and was apparently reinfected in November 1933. From February 1924 until August 1933 he received 14 injections of neoarsphenamine, 7 injections of arsenicals (type not stated), and 5 injections of mercury. Following his apparent reinfection the patient was given 0.3 gram of neoarsphenamine on February 3, 1934; 0.45 gram on February 6, 13, 20, 27, and March 6; and 0.5 gram on March 30 and April 7. As concurrent treatment he was given intramuscular injections of bismuth, 0.13 gram each, on March 2, 9, April 5, 16, 26, and May 4.

On May 6, 29 days after the last injection of neoarsphenamine he was admitted to the sick list with a chronic scaly eruption on both feet and ankles. The patient had been treated for similar complaints several times during the past 16 months without complete healing. Recently the condition of the feet and ankles had become worse. The present acute symptoms began several days ago with the appearance of erythematous macular spots over both legs, and scattered discrete patches over various portions of the body, hands, arms, and head. In addition to the eruption, both hands and feet became edematous. The area in and about the thighs itched considerably, resulting in numerous scratch marks. Progressive weakness, rapid fatigue, a generalized petechial eruption, tachycardia with cardiac palpitation and visual disturbances (blurred vision and metamorphopsia), were noted early. A marked pallor without emaciation was present when patient first reported symptoms. A slight fever,

99.6° F., was present when first placed on the sick list. The weakness rapidly became more marked. Pain in precordial region followed exertion. Headache became severe. On admission to a naval hospital, May 12, his temperature was 100.6° F., pulse 88, and respirations 20 per minute. The pallor was marked. Pitting edema of the lower extremities was present, but the swelling of the hands had subsided. The trichophytosis lesions of the ankles were extensive and oozing a bloody fluid. Above the lesions was a dusky erythema extending to above the knees. Scattered petechial hemorrhages were present on the thighs, abdomen, scrotum, chest, and buccal mucosa. There were numerous small retinal hemorrhages, flame shaped and irregular shaped, but no clouding of the vitreous. There was one small subconjunctival hemorrhage. Retinoscopic examination revealed that there were also areas of " cotton wool " exudate and pallor of disks. The interpretation of the eye findings was that they were not due to simple hemorrhagic retinitis, but were characteristic of an arsenical amblyopia. The teeth were in poor condition with receded gums, fetid breath, and a dark line on gums considered typical of heavy metal salivation. The tonsils appeared chronically inflamed. Heart sounds were faint. There was a systolic murmur but no thrill over the precordium. The heart was not demonstrably enlarged. Blood pressure 144/64. Lung sounds were normal. The abdomen was normal on palpation, neither the liver nor spleen being palpable. The slight fever persisted for 7 days, during which time the pulse continued under 100 per minute. The edema of the lower extremities subsided in 2 days and the lesions showed marked improvement. The patient felt much better after the first blood transfusion on May 13, and stated that the cardiac pain and palpitation had subsided. No new petechial spots were noted and those present began to fade. The headaches subsided. The gums improved and the dark line disappeared. The pallor was less marked.

On the evening of May 22, 10 days after admission to the hospital, the patient was seized with a severe attack of pain in the left chest requiring morphine for relief. The temperature became elevated and the pulse and respiration increased. Physical examination revealed a loud friction rub over the painful area. Pain diminished by the next day and subsided after 3 days. X-ray on May 28 showed left pleural effusion with fluid level to third interspace and remainder of lung clear. The fever rose to 105.4° F. on May 25 following a blood transfusion and receded to 100° F. on May 27. The weakness and pallor became more marked. The heart rate had been 122 per minute during the height of the fever but slowed to 100 on May 27. Due to the weakened condition of the patient aspiration of the fluid was not attempted. The patient complained of dull abdominal pain. On the morning of May 28 he complained of nausea and later

vomited. On the afternoon of May 28 dyspnea and subnormal temperature with a cold clammy skin developed. The pulse rate reached 118 per minute and respirations 38. The mental state was clear until late in the evening, when he lapsed into coma and died quietly at 12:35 a. m., May 29, 1934, 17 days after admission to the hospital; 23 days after admission to the sick list; and 52 days after the last injection of neoarsphenamine.

Blood

Date	R. b. c.	W. b. c.	Hgb.	Polys.	Lymphs.	Monos.	Eosin.
May 12, 1934	1,300,000	3,200	30-40	54	42	2	
May 17, 1934[1]	1,600,000	1,500	25	8	11	1	1
May 21, 1934	1,800,000	2,650	40	41	52	2	5
May 25, 1934	1,020,000	2,200	25	59	37	2	2
May 28, 1934	990,000	2,050	20				

[1] Only 21 cells found on slide.

No nucleated red cells seen at any time. No variations in size or staining of red blood cells noted. Only an occasional platelet seen on stained side.

Autopsy findings.—Autopsy performed 2 hours after death. Body that of an adult male about 35 years of age, weight about 200 pounds, height about 5 feet and 10 inches. Muscular development about average. No cyanosis or lividity. Healing superficial ulcerations about both ankles discolored with gentian violet dye. Numerous tattoo designs on arms and legs. Numerous petechial spots from pin head to one-half centimeter size on left hip, thighs, abdomen, and scrotum. No post-mortem rigidity. The subcutaneous fat was well preserved. The left pleural cavity contained about 4 liters of thin, blood-tinged fluid. The left lung was completely collapsed and felt normal throughout. The right lung was normal except for some recent adhesions to the diaphragm. The right pleural cavity contained no free fluid. A few petechial spots of one-half centimeter size noted on the parietal pleura. The pericardial sac contained no free fluid. The heart had been displaced about an inch to the right, but resumed its normal position when the left pleural cavity was drained. The heart was of normal size. On the posterior surface were numerous petechial areas of small size. The coronary arteries were unusually thin and soft. The cavities were empty. The valves were normal. Under the endocardium were numerous petechial spots, mostly of pinhead size. They were especially numerous on the papillary muscles of the left ventricle. The heart muscle appeared pale but of normal thickness. The aorta was normal except for three small atheromatous plaques. The stomach and intestines were normal. The liver was normal but of pale appearance. The spleen was small and soft and appeared normal on section. The pancreas was normal.

The kidneys were of normal size. The capsules stripped readily, and the cortex appeared of normal thickness. There was more than the usual amount of fat about the calyces. The left kidney appeared to be much paler than the right. There were numerous small petechial hemorrhages throughout the substance of the right kidney, but none on the left. The bladder was empty. The prostate was not enlarged. No petechial hemorrhages were seen on the visceral or parietal peritoneum. The bone marrow of the ribs and sternum appeared pale, fatty, and acellular. The brain was not examined.

Gross pathological findings on autopsy.—Specimen consisted of six pieces of bone, including marrow, kidney, spleen, adrenal, three sections of liver, and two sections of lung.

Liver: The liver cells show all stages of degeneration, from that of a mild granular condition to cytoplasm necrosis and atrophy, leaving only the reticular framework. The atrophy is more marked about the central veins but there are scattering foci both midzonal and marginal. Some of these degenerated cells, especially about the central vein, are laden with bile pigment. The sinusoids are dilated and partially filled with laked erythrocytes. The Von Kupffer's cells contain hemosiderin pigment.

Spleen: The capsule trabeculae appear normal. There is a decrease in number of the malpighian corpuscles leaving only a thickened wall arteriole. Those that are present are indistinct in outline and atrophic in appearance. The sinusoids are dilated and congested with erythrocytes. There is moderate endothelial proliferation and the pulp contains a goodly number of plasma cells. Granules of hemosiderin pigment are scattered throughout the tissue.

Kidneys: All sections are quite similar. There is nothing unusual about the glomeruli except capillary congestion. The epithelium lining the proximal convoluted tubules shows granular degeneration with some atrophy. The vascular bed is congested throughout.

Lung: The pleura shows moderate fibrous thickening with compression of the adjacent underlying alveoli. In these compressed areas the alveolar spaces are partially filled with masses of iron-containing macrophages and an occasional one containing carbon. In other areas the bronchi appear essentially normal. There is a tendency to alveolar collapse; their walls are of normal thickness and the capillaries congested. There is a scattering of heart-failure cells throughout all lung sections. The lymphoid tissue appears atrophic.

Adrenals: The cortical cells are granular and foamy in appearance. The sinusoids are congested. The medullary portion of the organ shows nothing unusual except congestion.

Heart: The fibers stain poorly, are fragmented, and the cross striations are indistinct. There is a slight amount of lipochrome pigment about the nuclear poles. The coronaries appear normal.

Bone marrow: There is marked decrease of cellular elements with fat replacement. The granular cells are few. With difficulty is a nucleated red cell found and the megakaryocytes are extremely rare, and those seen appear degenerated. The reticulo-endothelial structures are also atrophic.

The pathological findings are those seen in arsenical toxicosis with bone marrow involvement.

Pathological diagnosis.—Poisoning, neoarsphenamine; hypoplastic bone marrow, marked; toxic parenchymatous degeneration, liver; myocardial degeneration; splenitis, subacute, toxic; parenchymatous degeneration, kidneys and adrenals; chronic passive congestion, lung, kidney, and adrenal.

(107—1934) A patient had a history of four chancroids on his prepuce on June 22, 1931. Dark-field examination and a Kahn blood test were negative. On July 17, a generalized maculo-papular rash and general adenopathy developed. A Kahn blood test was reported 4 plus the same day.

From August 3, 1931, to September 6, 1933, he received 44 injections of arsenicals and 43 injections of bismosol.

On March 7, 1934, a fifth course of treatment was instituted, the patient receiving a 0.3 gram injection of neoarsphenamine.

Two days later the patient noted symptoms of illness but did not report to the sick bay until the following morning at which time he complained of nausea, vertigo, and swelling of the ankles. Examination revealed hemorrhagic purpura of both legs. Temperature, 101.2° F.; pulse, 84. Red blood count, 4,200,000; white blood count, 15,800; Differential: polymorphonuclears, 70; lymphocytes, 28; eosinophiles, 2. His condition rapidly improved under treatment and he recovered in 5 days.

(108—1934) A patient in whom a dark-field examination of the primary lesion was positive for *Treponema pallidum* on September 15, 1934, was given a 0.3-gram injection of neoarsphenamine on that date. On September 18 he was given 0.45 gram, and on September 25 a second 0.45-gram injection, which was dissolved in 10 cubic centimeters of water and injected in 3 minutes.

Two hours later he was admitted to the sick list complaining of chills and fever. His temperature was 102° F., pulse 120, and respirations 24. He had redness of the skin on his chest which was suggestive of a macular rash. His throat was reddened and angry. Urine: Appearance, clear; reaction, acid; specific gravity, 1.018; albumin, faint trace; sugar, negative. Blood: Red blood count, 4,430,000; white blood count, 3,300; hemoglobin, 80 percent. Differ-

ential: Juveniles, 17; band forms, 72; segmented, 4; lymphocytes, 15; eosinophiles, 1; monocytes, 7. The red blood cells showed a moderate degree of anisocytosis and poikilocytosis. A majority of the cells were well hemoglobinized. No polychromasia or stippling was noted.

Under palliative treatment he recovered in 9 days.

Subsequent blood examinations were:

October 16. Red blood count, 4,800,000; white blood count, 6,050; hemoglobin, 90 percent. Differential: Band forms, 27; segmented, 18; lymphocytes, 36; eosinophiles, 1; basophiles, 3; monocytes, 15.

October 29. Red blood count, 4,750,000; white blood count, 4,250; hemoglobin, 90 percent. Differential: Band forms, 11; segmented, 41; lymphocytes, 35; eosinophiles, 4; basophiles, 2; monocytes, 7.

(109—1934) A patient after exposure on December 1, 1933, developed an intraurethral chancre on January 1, 1934. On January 22 and 27, Kahn blood tests were 4 plus. On January 29 a macular rash developed on trunk, thighs, and arms. Arsenical treatment was instituted on January 31, with a 0.3-gram injection of neoarsphenamine. On February 7, 14, and 21 he received 0.6-gram injections.

On February 22 he awoke feeling chilly but did not report his condition. The next morning he complained of a sore throat, chills, and fever. His tonsils and pharynx were swollen and injected. His temperature was 103° F., pulse 104, and respirations 20. Blood pressure was 104/60. Urine: Appearance, clear, amber; reaction, acid; specific gravity, 1.022; albumin, slight trace; sugar, negative. Microscopical: Few epithelial cells and an occasional pus cell.

February 24. Temperature, 103° F. Ulceration left tonsil spreading.

February 25. Diagnosis changed to septic sore throat. Temperature, 103.2° F.

February 26. No improvement in condition of throat, ulceration extending.

March 1. Blood picture showed a marked shift to the left. Patient complained of dull pain over left side of chest. Examination showed numerous coarse moist râles over left side. Moderate dullness on percussion over left base. X-ray examination showed a bronchopneumonia left side.

March 2. Evidence of bronchopneumonia both sides. Heart action fair. General condition somewhat worse.

March 5. Ulcer on left tonsil healing rapidly. Right side of chest apparently clearing.

March 7. General condition much improved. Apparently patient had pseudocrisis this morning.

March 8. Throat ulcer practically healed.

March 10. Few scattered râles right and left base, slight impairment of percussion. Breath sounds come through. Slight enlarge-

ment of cardiac dullness. Aortic and mitral areas normal. Slight accentuation of first sound at apex, pericardial friction rub at apex. Blood pressure 122/66.

March 12. Cardiac examination: There is both a pericardial and bronchial friction rub loudest at the apex, a soft blowing double mitral murmur, to and fro, heard over base of heart but not transmitted, both A2 and P2 accentuated. The same double murmur is heard at right sternal margin, probably transmitted from mitral area.

March 15. Electrocardiogram report: Sinus tachycardia with a rate of 116. S–T segments 1 and 2, 1 mm. elevation above base line. Otherwise tracing within normal limits.

March 16. Pleural rub still heard over left lung, pericardial rub still present.

March 18. Patient complained of precordial pain on coughing.

March 20. Precordial pain still present. Left base is dull on percussion. Pericardial rub and many rales heard. Bedside X-ray of chest shows a change in the appearance of the heart shadow. It is now nearer "jug" shape with broadening of the base. The left border is convex, apex well to the left. There is still some unresolved pneumonia, right midlobe and probably left base. The heart shadow is now suggestive of a pericardial effusion.

March 22. Patient stated he had the best night he had since he has been sick.

March 27. Patient continues to improve.

April 6. There is no pericardial friction rub, heart sounds good. Rales are heard at right and left base.

April 7. Teeth and gums are in very poor condition, an apparent Vincent's infection.

April 10. Patient is getting stronger all the time.

April 12. Patient continues to improve.

April 17. Gaining in strength. Is sitting up in a chair.

April 24. He has gained much in strength.

May 1. Patient is in good condition.

May 25. Four lower central incisors have been extracted because of pyorrhea and recession of gums. Gums now healed. Patient looks well, and feels well.

June 4. Temperature, pulse, and respirations, normal. Complains of occasional shortness of breath on exercise. No other symptoms.

June 13. Temperature, pulse, and respirations, normal. Chest examination negative. No shortness of breath.

June 21. Blood picture normal. Weight is better than usual and response to exercise is excellent.

June 25. To duty, 123 days after onset of first symptoms.

Blood

Date	Red blood count	Hemoglobin	White blood count	Myelocytes	Juveniles	Band forms	Segmented	Lymphocytes	Monocytes	Eosinophiles	Basophiles
Feb. 22 [1]	5,390,000	70	5,900					92	3		
Feb. 26 [2]	4,410,000	70	2,400	12	33	7		47			
Feb. 27			3,350								
Feb. 28			2,750								
Mar. 1 [3]			3,375	21	5	4		51			1
Mar. 2 [4]	3,750,000	80	1,500	24	7	1	2	52	2		
Mar. 3 [5]	4,180,000	80	1,460	31	4	11		47	1		
Mar. 4 [6]			6,050	18	20	14		42	2		2
Mar. 5 [7]	4,500,000	80	10,600	16	16	32	9	13	1		
Mar. 6 [8]	3,860,000	77	13,950	8	4	44	22	19	1		
Mar. 7	4,020,000	70	14,800		2	22	54	18	3	1	
Mar. 8 [9]	3,410,000	70	12,750	1	5	26	49	11	5		1
Mar. 9	4,500,000	81	15,950			19	61	13	4	2	1
Mar. 10	4,120,000	80	19,500	2	4	14	63	14		1	2
Mar. 11 [10]			12,400		7	5	13	13		1	
Mar. 12	4,160,000	80	15,800		2	27	56	12	2	1	
Mar. 14 [11]	4,290,000	80	12,250					18	5		
Mar. 15	3,960,000	85	10,700			16	55	25	2	1	1
Mar. 16	4,380,000	75	10,750		4	27	48	16	5	1	
Mar. 17	4,260,000	75	7,800		3	26	45	24		2	
Mar. 18			11,600		1	14	51	25	2	1	
Mar. 19	4,350,000	80	11,600		1	22	52	21	1	1	1
Mar. 22			8,450								
Mar. 23			6,400		1	19	54	21	2	3	
Mar. 24 [3]			9,250		4	30	33	21	4	5	1
Mar. 26			6,550			9	49	28	6	8	
Mar. 28 [2]			6,850			8	52	24	11	4	
Mar. 29			6,600			7	55	33	5		
Mar. 30			7,400			11	44	37	4	7	
Apr. 1			7,800			12	50	35		2	
Apr. 2			6,400			7	41	33	15	4	
Apr. 3			6,700			7	48	26	13	4	2
Apr. 4			6,350			8	35	45	7	5	
Apr. 5			6,550			20	45	23	10	2	
Apr. 6			5,900			6	56	27	8	3	
Apr. 7			5,800			10	39	31	1	1	1
Apr. 9			5,700			12	31	33	20	3	1
Apr. 11 [3]			5,150			12	35	33	14	4	1
Apr. 13			5,800			3	38	39	14	3	1
Apr. 16			4,750			13	21	45	12	7	2
Apr. 17 [3]			6,450			8	21	52	10	7	3
Apr. 18			6,300			11	37	49	3		
Apr. 19			6,450			25	39	24	12		
Apr. 20			5,450			4	33	46	11	6	
Apr. 21			5,750			8	22	49	15	4	2
Apr. 23			6,500			7	15	60	12	4	2
Apr. 24			5,800			6	35	41	16	1	1
Apr. 25			6,300			12	24	47	11	6	
Apr. 26			6,450			9	32	38	15	4	2
Apr. 28			6,250			13	24	49	13	1	
Apr. 30			7,850			21	27	40	12		
May 2			7,050			8	47	36	9		
May 3			7,250			10	37	39	4	6	4
May 4			8,950			8	30	51	9	2	
May 8			6,500			11	42	35	12		
May 11			6,650			6	34	55	2		3
May 17			5,950			8	33	41	10	6	2
June 4	5,120,000	90	6,750			13	43	42	2		

NOTE.—Additional cells were as follows:

[1] Transitional 4.
[2] Turck's cell 1.
[3] Pre-erythrocytes, 21; blast cell, 14.
[4] Turck's cell, 1; myeloblast, 9.
[5] Blast cell, 2; Turck's cell, 4.
[6] Turck's cell, 2; normablast, 1.
[7] Premyelocytes, 10.
[8] Blast cell, 1; premyelocytes, 1.
[9] Blast cell, 1; Turck's cell, 1.
[10] Myeloblast, 1.
[11] Transitional, 1; polymorphonuclears, 76.

(110–1934) A patient was given a diagnosis of syphilis on August 11, 1934, because a dark-field examination of an indurated penile lesion was positive for *Treponema pallidum*. A Kahn test was 1

plus on August 14. Arsenical treatment was instituted on August 11, with a 0.225 gram injection of neoarsphenamine. On August 15, 22, 29, and September 5, he received 0.45 gram injections.

Two days later, September 7, the patient noted a slight discomfort in his throat, heaviness of the tongue, and soreness of his gums. He felt weak and that night he was restless, with increasing sore throat. The next morning he started to perform his regular duties but felt weak and feverish. He then reported to the sick bay. His face was flushed and his eyes were injected. His gums and posterior pharyngeal wall were slightly inflamed. Swallowing was difficult. His temperature gradually rose to 103° F., pulse, 110, and respirations, 25.

September 9. Temperature, 101° F.; pulse, 108; respirations, 25 at 7 a. m. The throat was more painful and the gums were more swollen. The lymphoid tissue at base of tongue on right side was greatly swollen and painful. At 2 p. m. his temperature was 104° F., pulse 120, respirations 30. All subjective symptoms had increased. At 8 p. m. his temperature was 103° F., and a white blood count was 4,600. By 10 p. m. his temperature rose to 105° F.

September 10. Temperature, 103° F.; pulse, 115; respirations, 20. The throat was very painful. Cervical adenopathy increased. Sibilant high-pitched rales were heard in bases of both lungs but there was no evidence of consolidation. He coughed slightly but had no chest pains. Blood pressure 112/70. At 10 a. m. a white blood count was 2,750. Differential: lymphocytes, 96; polymorphonuclears, 4; hemoglobin, 90 percent. Smear from throat showed Gram positive cocci; morphologically staphylococci and short chain streptococci; few Gram positive diplococci, morphologically pneumococci. Urine: color, amber; reaction, acid; specific gravity, 1.015; albumin, 4 plus; sugar, negative; microscopical: Loaded with fine and coarse granular casts, few leucocytes, many epithelial cells, and many urates. Blood culture was sterile after 72 hours. Icterus index 22.5. Bedside X-ray of chest: Moderate thickening at the hilum with a few lymphoid nodules. Moderate peribronchial thickening in the central lung field, somewhat more marked in the lower lobes than in the upper. No visible areas of pneumonic consolidation. Findings essentially negative.

September 11. Throat showed little change. Numerous sibilant and sonorous rales throughout both lung fields. Patient stated he felt better and throat seemed to be less painful. Temperature, 103.4° F.; pulse, 102; respirations, 20.

September 12. Throat appeared the same, great difficulty in swallowing. Platelet count 304,640; 80 percent granular white cells. Intake of fluids 2,940 cubic centimeters during the past 24 hours. Temperature, 104° F.; pulse, 120; respirations, 22.

September 13. Temperature remittent, 102–104° F. Slight nonproductive cough. Numerous sibilant and sonorous rales throughout both lung fields. Throat shows little if any improvement.

September 14. Coagulation time, 3 minutes, 45 seconds; bleeding time, 1 minute, 30 seconds; temperature, 102–103° F.; pulse, 120; respirations, 20–40. Numerous medium and coarse rales throughout both lung fields. Throat appeared unchanged. Patient slightly cyanotic at times. Fluid intake 730 cubic centimeters. Patient took little nourishment.

September 15. Slept well last night following morphine. Throat appeared less red and apparently somewhat improved.

September 17. Temperature, 99° F.; pulse, 82; respirations, 20. Acute condition of throat subsiding. Patient took semisoft diet.

September 18. Inguinal glands and sternal button excised for histopathological examination: Gross examination, myelocytes, 1; juveniles, 1; band forms, 1; lymphocytes, 23; eosinophiles, 3; basophiles, 1; monocytes, 70. Blood picture: Red cells appear essentially normal. There is an absolute decrease in granular cells and lymphocytes. There is an absolute monocytosis, the majority of which show marked toxic degeneration. Blood platelets appear essentially normal in size and number. In the absence of any anemia and no deficiency of blood platelets the picture does not suggest any form of leukemia, but is suggestive of (1) agranulocytosis, infection; (2) agranulocytosis, due to some drug of the benzene group.

September 21. Temperature normal. Lungs clear. Throat clear. Cervical and inguinal glands are becoming smaller.

September 24. Patient feels good.

September 27. Patient up in a. m. and p. m. Appetite good.

September 28. Reports on additional blood smears: Gross examination, specimen consists of two portions of tissue, the largest of which measures 4.2 centimeters by 1.1 centimeters; it is irregularly "mitten" shape, yellowish in color, and rather rubbery in consistency. On cross section of the growth it is whitish and encapsulated. The other portion of tissue is bonylike in structure, measuring 0.6 centimeter in thickness, 2.3 centimeters in length. It is brownish in color, section divided through the middle. Specimen completely imbedded. Microscopic examination, section from the lymph gland shows a moderately fibrosed capsule. The germinal centers of the lymph follicles are large and hyperplastic. There is congestion of the cortical portion of the gland with accumulation of serum in this area and about the subcapsularlymph sinuses. There is no evidence of neoplastic change. The bone marrow appears to have a normal qualitative and quantitative cellular make up. The more mature types of granular cells appear slightly decreased. All other elements are essentially normal. There is no evidence of

leukemia. Pathological diagnosis: Hyperplastic lymph node; essentially normal bone marrow.

October 1. Temperature normal. Throat clean. Icterus index 12.

October 5. Report of blood picture: There is a marked regenerative left shift accompanied by a marked toxic granulation and degeneration of all granular cells. There is considerable evidence of regeneration and improvement over previous blood pictures, despite the toxic granulation and suggestion of some possible bone-marrow involvement. Impression: Poisoning, neoarsphenamine.

November 9. To duty 63 days after the onset of the first symptoms.

Blood

Date	Red blood count	Hemoglobin	White blood count	Myelocytes	Juveniles	Band forms	Segmented	Lymphocytes	Monocytes	Eosinophiles	Basophiles
Sept. 10	4,800,000	85	4,300	-------	-------	-------	36	58	6	-------	-------
11 [1]	-------	-------	4,050	-------	-------	-------	-------	-------	-------	-------	-------
12 [2]	-------	-------	3,600	-------	-------	-------	-------	-------	-------	-------	-------
13	-------	-------	3,200	-------	-------	-------	-------	-------	-------	-------	-------
14	-------	-------	3,000	40	6	6	32	-------	-------	-------	-------
15	4,060,000	80	8,200	31	13	9	21	23	-------	2	-------
17	-------	-------	13,800	29	16	15	27	11	-------	2	-------
18	4,190,000	80	14,200	18	9	11	37	20	1	2	1
19	-------	-------	22,000	12	9	10	48	17	1	1	1
20	4,210,000	80	19,200	3	8	13	59	15	2	-------	-------
21	-------	-------	14,800	2	7	15	56	16	2	2	-------
22	-------	-------	8,600	-------	5	10	66	15	2	1	1
24	-------	-------	8,400	-------	-------	3	63	24	1	2	1
26	-------	-------	11,200	-------	-------	3	63	30	3	1	-------
27	4,180,000	80	8,800	-------	-------	-------	-------	-------	-------	-------	-------
Oct. 3	4,280,000	80	7,600	-------	-------	1	55	35	4	2	3

[1] All cells stain poorly.
[2] Platelets 304,640. Granulocytes 80 percent, nongranulocytes 20 percent.

(111—1934) A patient who was exposed August 27, 1933, developed an indurated penile ulcer, inguinal and right epitrochlear glandular adenopathy, and a frontal headache. A Kahn blood test was 4 plus on September 26, 1933.

From September 26, 1933, to April 3, 1934, he received an unstated number of injections of neoarsphenamine for a total dosage of 13.35 grams, and 9 intramuscular injections of bismosol, 0.1 gram each.

From April 5 to June 25, 1934, he received 20 intramuscular injections of bismosol, 0.1 gram each, as intercurrent treatment.

On July 5, 1934, a third course of arsenical treatment was instituted, with an 0.3 gram injection of neoarsphenamine. On July 9, 12, and 16 he received 0.45 gram injections.

On July 16 he noted bleeding of the gums, and that night some fever blisters on his lower lip turned black. The next day a small abrasion on the left hand was found to be purpuric and a small pustule on the left leg, which had been scratched, was also purpuric. There were two small faint purpuric spots on the left thigh while both legs showed a number of very small purpuric spots. The site of the injection showed a small hemorrhage.

July 17. Blood: Bleeding time, 5 minutes; coagulation time, 3 minutes and 45 seconds; platelets, 325,000; white blood count, 7,000; Differential: band forms, 6; segmented, 62; lymphocytes, 28; monocytes, 2; eosinophiles, 1.

July 18. The gums stopped bleeding in the afternoon.

July 20. Blood: Bleeding time, 2 minutes and 30 seconds; coagulation time, 4 minutes; platelets, 365,000; white blood count, 9,300; Differential: band forms, 6; segmented, 68; lymphocytes, 24; monocytes, 1; eosinophiles, 1.

July 21. All purpuric spots have faded. The patient had been continued on duty under treatment.

(112—1934) A patient who was exposed on June 10, 1933, developed two indolent lesions on the frenum of his penis on July 4. On July 7 a dark-field examination was positive for *Treponema pallidum.*

From July 12 to December 2, 1933, he received 17 injections of neoarsphenamine, total dosage 7.2 grams, and 6 injections of bismosol. From April 4 to July 23, 1934, he received 9 injections of bismosol as intercurrent treatment.

On July 26, 1934, a 0.227 gram injection of neoarsphenamine was administered as the first injection of a third course of treatment. On August 2 and 9 he received 0.45 gram injections. The latter dose was dissolved in 15 cubic centimeters of sterile distilled water and injected in 1 minute.

About 4 minutes later he became dizzy, vomited several times, and complained of headache. The conjunctivae were injected. His temperature was 100.8° F.; pulse, 102; respirations, 22. Urine was negative. Dickens' test was positive. Blood: 11 a. m., white blood count, 2,700; 3 p. m., red blood count 5,980,000; white blood count, 5,500; Differential: segmented, 8; band forms, 6; juveniles, 4; myelocytes, 2; lymphocytes, 77; basophiles, 2.

Under treatment recovery was complete in 20 hours.

On August 22, a blood count was as follows: White blood count, 7,100; Differential: segmented, 66; lymphocytes, 21; monocytes, 10; eosinophiles, 3.

(113—1934) A patient who was exposed on August 11, 1933, developed a typical chancre, about 2 centimeters in diameter with a punched-out appearance, on the dorsal side of glans penis on August 28. On September 2, a dark field examination was positive for *Treponema pallidum* and on December 4, a Kahn blood test was 4 plus.

From September 19 to December 28, 1933, he received 10 injections of neoarsphenamine, total dosage 5.55 grams, and 15 intramuscular injections of mercury bichloride, one-sixth grain each, and

from January 5 to May 21, 1934, he received 16 injections of mercury bichloride, one-sixth grain each.

On June 12, 1934, he was given a 0.3 gram injection of neoarsphenamine as the first injection of a second course of arsenical treatment. On June 26, he received a 0.45 gram injection, and on July 3, 0.6 gram. Immediately after this injection the patient noted several skin lesions on the chest, shoulders, and upper arms. The lesions faded the next day.

On July 12 he was given 0.6 gram of neoarsphenamine and approximately 12 hours later the patient noted a few skin lesions. They were brownish-red macular patches, 1 to 1½ centimeters in diameter without scales. There was no pruritus and the patient had no other complaints. Arsenical treatment was discontinued.

On August 13 an examination revealed two typical mucous patches on the under surface of the tongue. Examination was otherwise negative except for a few skin lesions of a dull bronze color which remained on the shoulders and arms. The next day the patient was given a 0.2 gram injection of neoarsphenamine as a test dose. Two hours later the skin manifestations recurred, but were of a bright red color. The patient had no complaints. Urine, negative.

August 15. Urine, negative; red blood count, 4,790,000; hemoglobin, 90 percent; white blood count, 4,950. Differential: band forms, 4; segmented, 66; lymphocytes, 25; eosinophiles, 2; monocytes, 2.

The patient was not admitted to the sick list, but under treatment the lesions faded in approximately 1 week.

On September 18 an examination revealed only a slight discoloration of the skin at the sites of the former lesions.

ARSENICAL NEURITIS

Neoarsphenamine.—(124—1934) A patient was infected on March 29, 1934, and on April 9 two dark field examinations of a typical chancre on the sulcus of his penis were positive for *Treponema pallidum.* He had beginning inguinal adenopathy and a Kahn blood test was 1 plus. The same day he received a 0.3 gram injection of neoarsphenamine. The course was continued and on April 12, 16, 19, 23, 26, 30, May 3, 7, 10, and 14 he received 0.45 gram injections, an average of 131 milligrams per day. As concurrent treatment he received 1 injection of bismosol and 25 days' treatment with mercury rubs.

Following the last injection of neoarsphenamine the patient noted a tingling sensation in the thumbs and index fingers. On May 17 he was given another 0.45 gram injection of neoarsphenamine. Following this injection he noted the same sensations, and in addition had tingling in the balls of the feet and toes. Four further injections of neoarsphenamine, 0.45 gram each, were given on May 21, 24,

28, and 31. Following these injections there was little, if any, increase in the symptoms.

May 28. White blood count, 7,000. Differential: segmented, 65; lymphocytes, 30; eosinophiles, 2; monocytes, 3.

June 12. The patient was admitted to the sick list with acute cholangitis with temperature 99° F., general malaise, lassitude, and anorexia. He had an icteroid tint of sclerae and tenderness over epigastrium on pressure. The cholangitis symptoms subsided in about a week. White blood count, 12,800. Differential: band forms, 4; segmented, 60; lymphocytes, 23; eosinophiles, 9; monocytes, 4. Urine: Appearance, dark amber; reaction, acid; specific gravity, 1.020; albumin and sugar, negative. Microscopical: Few white blood cells and much mucus.

June 13. Urine: Negative except for an occasional white blood cell.

June 15. Complains that paresthesia of hands and feet is more marked than previously.

June 17. Patient somewhat depressed, and complains of marked paresthesia of hands, forearms, feet, and legs.

June 18. Lumbar puncture: Pressure, 14 millimeters mercury; cells, 1; globulin, no increase; Kahn, negative.

No ova or parasites were found in feces on June 13, 14, and 18.

June 20. At times the patient had felt a vibrationlike sensation above the knees. He also had felt " messed up " in walking and could not control his muscles so well. An examination showed a diminished touch sensation in toes and balls of feet and of the first three fingers of hands. Sensation to pressure present but delayed. Pain sensation present. Heat and cold sensation present but somewhat delayed. Tendon reflexes of knees plus, of ankles diminished, of biceps plus, of wrist diminished. Superficial abdominal and scrotal reflexes plus. Babinski no response. No ankle clonus. Rhomberg negative.

June 25. Sensation of hands and feet slightly improved.

June 26. White blood count, 5,000; Differential: band forms, 4; segmented, 63; lymphocytes, 29; eosinophiles, 1; monocytes, 3.

July 6. Started on potassium iodide, grains X, three times daily.

July 13. Some improvement in sensation.

July 22. Patient had 4 grams of sodium thiosulphate intravenously. He appeared well but still complained of some discomfort in feet in afternoon.

August 1. Sedimentation time 5 millimeters drop 1 hour. Red blood count, 4,110,000; hemoglobin 80 percent; white blood count, 10,000; Differential: segmented, 40; lymphocytes, 46; monocytes, 1; eosinophiles, 13.

August 10. Condition same.

August 13. Diagnosis changed to multiple neuritis. Started on high vitamin diet and potassium iodide, grains 15, three times daily.

August 14. Slightly improved. Patient now on potassium iodide, grains 25, three times daily, high vitamin diet, and physiotherapy.

August 15. Icterus index 12. Van den Bergh negative.

August 18. Feces negative for ova and parasites. Urinalysis negative.

August 28. Has early iodide rash; iodide discontinued.

August 31. Subjective sensations improving more rapidly. Moderate steppage gait. Evidences improved behavior and outlook in place of former anxiety and fretfulness.

September 4. Basic condition improving slowly. Today presents purulent urethral discharge. Smear shows numerous pus cells, no organisms.

September 15. No urethral discharge. Prostate normal, smear from same shows few gram positive cocci and diplococci and many pus cells.

October 6. Continues to improve slowly. Stool shows ova of Necator americanus.

October 15. Allowed exercise in gymnasium.

October 30. Convalescing. Physiotherapy continued.

November 16. Given thyroid treatment for hookworm.

November 19. Stools negative for ova or parasites.

November 27. Patient recovered, 207 days after the onset of the first symptoms.

GASTRO-INTESTINAL SYMPTOMS

Neoarsphenamine.—(125, 126, 127—1934) Three reactions occurred in the same individual. A patient had a penile lesion in May 1927, which was negative for *Treponema pallidum* and which healed in a few days. Diagnosis of syphilis was made August 30, 1928, when he reported to the sick bay because of pain in his right leg in the region of the sciatic nerve. He had general adenopathy and a Kahn blood test was 4 plus. From September 21, 1928, to January 18, 1932, he received 20 injections of arsenicals, 78 mercury inunctions, and 18 injections of bismosol.

On October 19, 1934, he was given a 0.3 gram injection of neoarsphenamine as the first injection of a fourth course of arsenical treatment. The course was continued and on October 27, 31, and November 8, he received 0.45 gram injections. As concurrent treatment he received intramuscular injections of bismuth salicylate, 0.13 gram each, on October 25, 30, and November 6. Following the last injection of neoarsphenamine the patient felt nauseated and vomited up a small amount of blood-tinged sputum. The patient stated that

he had always felt nauseated following an injection of neoarsphenamine and that he often vomited. He was retained on the sick list for 2 days, during which time it was learned that he had been drinking to excess.

On November 15, after he had received a 0.5 cubic centimeter subcutaneous injection of adrenalin, he was administered a 0.45 gram injection of neoarsphenamine. Again he was nauseated and vomited but did not report these symptoms at the time.

On December 13 he was again given a 0.5 cubic centimeter intramuscular injection of adrenalin, following which he was given a 0.2 gram injection of neoarsphenamine which was dissolved in 10 cubic centimeters of water and injected in 3 minutes. Four minutes later he began to feel nauseated and vomited mucus and bright red blood. He complained of headache and general malaise. His temperature was 101° F., pulse, 90; respirations, 20. With the exception of injected conjuntivae and mouth which contained bright red blood diluted with saliva and gastric juice, a physical examination was negative. Urine: Appearance, amber; reaction, acid; specific gravity, 1.020; albumin and sugar, negative. Microscopical: Red blood cells, none; white blood cells, numerous. Blood: White blood count, 7,000; Differentials; polymorphonuclears, 75; lymphocytes, 25.

Fifteen minutes after the onset of the symptoms he was given 1 gram of sodium thiosulphate intravenously. Fifteen minutes later he was given one-fourth grain of morphine sulphate. Fifteen minutes after the injection of morphine he stopped vomiting, after having vomited approximately 10 cubic centimeters of blood. He dropped off to sleep and slept several hours. On awakening he felt entirely well. Recovery was considered complete in 45 minutes after the onset of the first symptoms.

VASOMOTOR PHENOMENA

Neoarsphenamine.—(62, 63—1934) This patient experienced three reactions. Two were mild vasomotor phenomena and the third a fatal arsenical dermatitis. (The fatal reaction is described in detail in the October 1935 issue of the Bulletin under "Case No. 64—1934.")

The patient contracted syphilis in February 1934. Diagnosis of syphilis was made because of a typical Hunterian chancre on his penis which contained *Treponema pallidum.*

On March 12 he was given a 0.3-gram intravenous injection of neoarsphenamine, following which he was nauseated and became so dizzy he had to lie down for several hours. In the evening he had a chill.

On March 20 he was given 0.6 gram of neoarsphenamine, and similar symptoms occurred as after the previous injection.

Heavy metal treatment was instituted on March 16 and continued until May 1, during which time he received 9 intramuscular injections of bismuth salicylate of 0.13 gram each.

Arsenical treatment was continued, and on March 27, April 3, 10, and 17 he received 0.6-gram injections of neoarsphenamine. Previous to the sixth or last injection the patient had a slight skin rash. Following the injection he developed exfoliative dermatitis and died 33 days later.

(2—1934) This patient developed a penile lesion which was positive for *Treponema pallidum* on November 7, 1932. On November 9, a Kahn blood test was 4 plus.

From November 7, 1932, to May 5, 1933, he received 16 injections of neoarsphenamine, and from November 7, 1932, to June 21, 1934, he received 86 injections of bismosol and 9 injections of mercury.

On September 25, 1934, a third course of arsenical treatment was instituted, with 0.3 gram of neoarsphenamine. On October 2 he received 0.45 gram and 20 minutes later developed diarrhea, vomiting, and profound shock. Despite treatment he remained pulseless for 12 hours. The pulse then gradually returned to normal. Nausea persisted for 24 hours and then ceased. He had severe albuminuria without suppression.

Recovered in 5 days.

(3—1934) A patient acquired syphilis on November 6, 1933. Diagnosis of syphilis was made about 1 month later because of generalized adenopathy and a 4 plus Kahn blood test. A recheck Kahn blood test was also 4 plus. He received 10 injections of neoarsphenamine, 2 injections of mercury, and 1 injection of bismuth subsalicylate in February 1934.

He began a second course of neoarsphenamine treatment on March 10, 1934, receiving a 0.3-gram injection. About 30 minutes later a man in the same compartment heard the patient groaning and saw him shaking in his bunk. He talked at that time, but soon had another convulsion. He then became comatose, with labored breathing and a full bounding pulse. Without regaining consciousness he went into a third convulsion. The sequence of events were: A cry as if in pain, tonic spasm, then clonic movements during which patient was unable to breathe. He became very cyanotic, pupils dilated, and finally took a breath. He was admitted to a hospital in a semicomatose condition. He had drowsiness and clouding of consciousness the next morning. The consciousness gradually returned in about 4 days.

March 15. He was given a 0.25-gram intravenous injection of arsphenamine.

March 20. He was considered recovered but was retained on the sick list for further observation and further antiluetic treatment,

which was administered as follows: Arsphenamine: March 22, 0.3 gram; March 29, 0.35 gram; April 5, 12, and 19, 0.45 gram.

April 20. Returned to duty.

May 4 and 11. He received 1-cubic-centimeter injections of bismosol.

On admission to the hospital, early meningovascular syphilis was suspected but the spinal fluid was negative in all respects. As no history of previous epileptiform seizures could be obtained and patient showed no further seizures during the 6 weeks of hospitalization, and further because the period of clouding of consciousness was prolonged beyond that ordinarily seen in epilepsy, the diagnosis of epilepsy did not seem justified. Also, in view of negative spinal fluid, the diagnosis of neurosyphilis was not made. The exact etiology of the seizure could not be determined, and as the patient made an apparent complete recovery, arsenical poisoning seemed to be the most logical cause for his illness.

(4—1934) A patient developed a chancroid on his penis on December 15, 1929. Subsequently, several Kahn blood tests were negative. In December 1931 he had a headache, sore throat, and a skin rash. On January 4, 1932, a Kahn blood test was 3 plus.

From January 4, 1932 to June 20, 1933, he received 4 courses of neoarsphenamine for a total of 41 injections.

On February 23, 1934, he was given a 0.3-gram injection of neoarsphenamine as the beginning of a fifth course of treatment. On March 2 he received 0.6 gram, and on March 9, 0.6 gram, which was dissolved in 10 cubic centimeters of sterile distilled water and injected in 3 minutes. Fifteen minutes later he went to stool and experienced a sharp, gripping pain centered about the umbilicus. Concomitantly a paresthesia of the feet and finger tips developed. Slight dyspnea ensued. Shortly thereafter he had a chill, which was followed by nausea and vomiting. Several hours later oliguria and albuminuria were noted. The abdominal cramps continued at half-hour intervals for 6 hours. The paresthesia gradually abated in the next 2 days. Oliguria and albuminuria decreased and ceased on the 4th day.

The patient gave a history of having a reaction in June 1933 after the last injection of the fourth course. He did not report this reaction.

(5—1934) A patient was given a diagnosis of syphilis in October 1931 because a dark-field examination of a chancre on his penis was positive for *Treponema pallidum* and a Kahn blood test was 4 plus.

From November 3, 1931 to April 7, 1933, he received 27 injections of neoarsphenamine.

On January 16, 23, 30, and February 2, 1934, he received 0.45-gram injections of neoarsphenamine. As concurrent treatment he re-

ceived 1-cubic centimeter injections of bismosol on January 5, 12, 19, and 26. The last injection of neoarsphenamine was dissolved in 10 cubic centimeters of distilled water and injected in approximately 4 minutes. Within 3 minutes the patient developed nausea, dyspnea, abdominal distention, and unconsciousness. He was markedly cyanotic, and respirations had ceased. His temperature was normal, but his pulse rate was 120. Urinalysis and white blood count were normal. Artificial respiration was applied for 2 minutes and epinephrine, 0.5 cubic centimeter, was administered intramuscularly.

Recovery was apparently complete in 15 minutes.

(6—1934) A patient was given a diagnosis of syphilis on April 11, 1933, because of generalized skin eruption and adenopathy, and healing lesions on his penis. On April 18 a Kahn blood test was 4 plus.

From April 11, 1933, to April 20, 1934, he received 31 injections of neoarsphenamine, total dosage 12.225 grams; 27 injections of bismuth, total dosage 34 grains; and 12 injections of mercury succinimide, one-fifth grain each.

On May 12, 1934, he was given a 0.225-gram injection of neoarsphenamine as the first injection of a fifth course of arsenical treatment. Three minutes later he complained of a severe headache. About 1 hour later he developed a few abdominal cramps. About 3 hours later he complained of chills, fever, and a severe headache. He was given ephedrine hydrochloride, sodium bicarbonate, and sodium thiosulphate. Within 5 minutes after the sodium thiosulphate he vomited 200 cubic centimeters of dark amber colored fluid and complained of cramps in his stomach. His temperature was 103.4° F.; pulse, 120; respirations, 20. His skin was flushed and dry and his abdomen was tense. His speech became slow and there was some retardation of thought.

Recovered in 6 days.

(7—1934) A patient was infected on June 17, 1933, and on July 10, a dark-field examination of the primary lesion was positive for *Treponema pallidum*.

He received 2 courses of neoarsphenamine, consisting of 22 injections, between July 14 and November 21, 1933, and 16 injections of bismuth salicylate between July 14 and January 22, 1934.

On March 15, 1934, he received 0.3 gram of neoarsphenamine and on March 22 and 29, 0.45 gram injections.

Immediately after the last injection the patient had nausea which disappeared in a few minutes. Two hours later he reported to the sick bay with chills, general malaise, and nausea. Blood pressure was 110/76, pulse 90, temperature 90° F. Urinalysis negative. Dickins'

test positive. Red blood count, 4,864,000; white blood count, 7,200; polymorphonuclears 57 percent.

Patient recovered within 2 hours.

(8—1934) A patient was infected April 10, 1934. A dark-field examination was positive for *Treponema pallidum* on April 14, and a Kahn blood test was negative on April 17.

He completed a course of 7 injections of neoarsphenamine for a total dosage of 3.0 grams, and 7 injections of bismosol, 1 cubic centimeter each, on May 30, 1934.

On June 27, 1934, treatment was resumed, the patient receiving a 0.45 gram injection of neoarsphenamine. He also received 0.45 gram injections on July 3 and 10. During the same period he received three 1 cubic centimeter injections of bismosol. During the last injection of neoarsphenamine, and after having received about 0.3 gram of the 0.45 gram dose, the patient suddenly became dyspneic, flushed, and nauseated. He was given 1 cubic centimeter of epinephrine subcutaneously and 24 hours later he was considered fully recovered.

(9—1934) A patient was given a diagnosis of syphilis on June 20, 1932, because he had typical penile ulcers, moderate inguinal adenopathy, and a scabies-like rash on trunk and legs with a few lesions on his hands. Dark-field examinations were positive for *Treponema pallidum.*

He completed a first course of treatment which consisted of 7 injections of neoarsphenamine, total dosage 4.75 grams, and 7 injections of bismosol, 0.1 gram each.

On October 27, 1934, treatment was resumed, the patient receiving a 0.225 gram injection of neoarsphenamine. On November 3 and 10 he received 0.45 gram injections.

Three hours after the last injection he developed a severe chill and temperature of 102° F. He had no other physical signs. The next day he was entirely normal. He was retained on the sick list under observation until November 16, when he was discharged to duty.

(10—1934) A patient was infected March 3, 1930. On April 10 and 17 he had 4 plus Kahn blood tests.

From April 7, 1930, to October 23, 1933, he received 6 courses of arsenical treatment which consisted of 38 injections of neoarsphenamine and 6 injections of sulpharsphenamine.

On March 22, 1934, he was given 0.3 gram of neoarsphenamine and about 20 minutes later complained of headache and pain in his chest. Temperature was 97° F.; pulse, 100; respirations, 20. His conjunctivae were injected and he had coarse bronchial rales. Urine: Reaction, acid; specific gravity, 1.024; albumin and sugar, negative.

Microscopical, few pus and few epithelial cells. Autenreith's test positive for arsenic.

Recovery in 48 hours.

(11–1934) This patient, who was exposed on July 30, 1933, developed a chancre on the shaft of his penis on August 16. On August 24 a dark-field examination was positive for *Treponema pallidum*. He had inguinal adenopathy and on September 20, a Kahn blood test was 4 plus.

From August 25, 1933, to January 10, 1934, he received 16 injections of neoarsphenamine, total dosage 9.0 grams, daily mercury inunctions for 53 days, and 53 days of daily treatment with potassium iodide in ascending doses.

On January 9, 1934, he began a course of bismosol therapy which was continued until March 20, during which time he received eleven 1-cubic centimeter intramuscular injections.

February 28, 1934, a third course of neoarsphenamine treatment was instituted, with a 0.3 gram injection. On March 7 and 21 he received 0.6 gram injections. Approximately 5 minutes after the last injection he became nauseated, vomited, and his bowels moved. He had weakness and vertigo. Fifteen minutes later his blood pressure was 90/60, pulse 80, temperature normal. One hour later his blood pressure was 110/64. He then developed a severe chill which lasted about 5 minutes. His temperature rose to 101.4° F.; pulse, 96; respirations, 18. Urine negative. The symptoms cleared up within 7½ hours. The next morning his temperature, pulse, and respirations were normal and his urine was negative. Blood: Polymorphonuclears, 70; small lymphocytes, 15; large lymphocytes, 9; eosinophiles, 1; transitionals, 3; monocytes, 2. He was discharged to duty the following day.

(12—1934) Two reactions occurred in the same individual. The patient was exposed January 8, 1931, and on January 13 a penile lesion appeared. Three dark-field examinations were negative for *Treponema pallidum*, and the lesion readily healed. Because of persistence of induration about the scar, dark-field examinations were made on February 14, 15, and 16. They were negative. Discrete painless inguinal adenopathy was present on February 16, and on February 19 a Kahn blood test was 3 plus.

Treatment was instituted on February 23, with a 0.2 gram injection of neoarsphenamine. On February 26, March 1, 5, 8, 12, 15, 19, 22, 26, April 2, and 5 he received 0.45 gram injections, an average of 123 milligrams per day. As concurrent treatment he was given 0.1 gram intramuscular injections of bismosol on February 20 and 21, and from February 20 to April 5 he received daily mercury rubs.

One hour after the last injection of neoarsphenamine the patient felt nauseated while he was eating his lunch. He had a chill, and 1

hour later registered a temperature of 99.6° F. and a pulse of 110. In the afternoon he vomited and his temperature rose to 100.6° F. The patient stated he felt nauseated following the injection of April 2, which was not reported. Red blood count, 4,200,000; white blood count, 5,000. Differential: band forms, 6; segmented, 65; lymphocytes, 26; monocytes, 1; eosinophiles, 1. His urine was negative for albumin. Eighteen hours later he was considered recovered.

April 6. Spinal fluid test, cells 1; globulin no increase.

April 8. He was given a 0.3-gram injection of neoarsphenamine as a test dose. He was kept in bed and given a small dish of soup for lunch. Three and one-half hours after the injection he became nauseated and vomited. Temperature, 100° F.; pulse, 85; respirations, 18. He was continued on the sick list under observation until April 11, when he was discharged to duty.

(14–1934) A patient developed a primary lesion on the shaft of his penis which was positive for *Treponema pallidum* on June 1, 1933. Generalized glandular adenopathy was also present.

In his first course of treatment he received 4 injections of neoarsphenamine and 2 injections of bismosol.

On July 28, 1934, he began a second course of treatment, with a 0.225-gram injection of neoarsphenamine. On August 4 and 11 he was given 0.45-gram injections.

Ten minutes after the last injection he became nauseated, vomited, and complained of headache and swelling of the face. He had slight chills and the conjunctivae were injected. Temperature, 98° F.; pulse, 112; respirations, 22. Urine: Appearance, clear, light straw; reaction, acid; specific gravity, 1.003; albumin, 2 plus; sugar, negative. Microscopical: Moderate number of pus and epithelial cells and fair number of hyaline and finely granular casts. Blood: White blood count, 12,500. Differential: segmented 81; band forms 14; juveniles 1; lymphocytes 4.

Recovery in 14 hours.

(15–1934) A patient developed two small penile ulcers, one on the frenum and one on the coronal sulcus, in September 1930. On September 24, a dark-field examination of the lesion on coronal sulcus was positive for *Treponema pallidum.*

The patient received three courses of arsenical treatment which consisted of 16 injections of neoarsphenamine and 6 injections of sulpharsphenamine.

On February 19, 1934, a fourth course of treatment was instituted, the patient receiving a 0.3 gram injection of neoarsphenamine. On March 2 he received a 0.3 gram injection; March 17, 0.45 gram; and on March 24, 0.6 gram. Ten minutes after last injection he began to sweat and feel nauseated. He soon began retching and trying to vomit. He became cyanotic, had difficulty in breathing, and his face became edematous. He was given treatment with adrenalin

and sodium thiosulphate and about an hour later he felt better and well enough to walk around.

The next day he was sent to duty.

(16–1934) A patient who was exposed on May 9, 1934, developed a penile lesion which was positive for *Treponema pallidum*. He started treatment on May 17, with a 0.3 gram injection of neoarsphenamine. On May 21 he received a 1 cubic centimeter intramuscular injection of bismuth salicylate and on May 24, a 0.45 gram injection of neoarsphenamine. Eight hours later he developed chilly sensations and a temperature of 102.6° F. He had no other complaints. His urine was negative.

Returned to duty the next day.

(17, 18—1934) Two reactions occurred in the same individual. A patient who was exposed on April 2, 1933, developed two small indurated penile lesions on April 13, which were positive for *Treponema pallidum* on April 15. On June 6 he completed a course of 6 injections of neoarsphenamine and 6 injections of bismuth. He received 16 intramuscular injections of bismuth during the period of August 7 to November 27, 1933.

On April 5, 1934, he was given a 0.3 gram injection of neoarsphenamine. About 3 hours later he stated he felt hot. About an hour later he felt cold but had no definite chill. He was given adrenalin subcutaneously and ephedrine by mouth and in about 1 hour was relieved of all symptoms. Temperature, 100° F.; pulse, 100; respirations, 24. Blood: Red blood count, 4,300,000; white blood count, 8,100; hemoglobin 90 percent; Differential: polymorphonuclears, 62; lymphocytes, 35; monocytes, 3. Blood pressure 118/70. He was retained on the sick list under observation and further treatment until April 11, when he was discharged to duty well.

The patient was continued under bismuth therapy and by August 30, 1934, had received 10 injections of bismuth salicylate.

On November 1, 1934, he was again started on arsenical treatment, receiving 0.3 gram of neoarsphenamine on that date and a 0.3 gram injection on November 8 and 22. About 1 hour and 20 minutes after the last injection the patient complained of feeling hot, headache, and eyes which felt "sore." About 20 minutes later he perspired profusely and felt cold. Temperature, 99.8° F.; pulse, 90, respirations, 20. His face was flushed and his conjunctivae were injected. Blood pressure, 118/70. Urinalysis was normal. His condition progressively improved under treatment and he became symptom free the next day, November 23, at which time a complete blood count was recorded as follows: Red blood count, 5,370,000; white blood count, 8,850; hemoglobin 90 percent; Differential: polymorphonuclears 83; lymphocytes 10; mononuclears 2; eosinophiles 5. He was transferred to a hospital and retained on the sick list under obser-

vation until January 17, 1935, during which time he ran a persistent 2 plus albumin.

(19—1934) A patient, in whom a dark-field examination of the primary lesion was positive for *Treponema pallidum* on November 11, 1933, completed a course of 11 injections of neoarsphenamine on January 11, 1934. He was given eight injections of bismuth salicylate as concurrent treatment.

On February 22 he received a 0.3 gram injection of neoarsphenamine as the first injection of a second course of treatment, and on March 3 he received a 0.45 gram injection. Approximately 9 hours later he reported to the sick bay complaining of weakness and dizziness. His blood pressure was 110/60 and 30 minutes later was 112/60. Temperature, 99° F.; pulse, 96; respirations, 18. Urinalysis was negative. Dickens' test was positive. Blood: Red blood count, 4,850,000; white blood count, 10,600; Differential: polymorphonuclears, 79 percent.

Recovery within 2 hours.

(20—1934) A patient, who was exposed on September 5, 1934, developed a primary lesion of the pubic region which was positive for *Treponema pallidum.* A Kahn blood test was 4 plus on October 1.

From September 26, 1931, to March 31, 1933, he had received 28 injections of neoarsphenamine, 24 injections of bismuth salicylate, and 30 mercury injections.

On January 20, 1934, he was given a 0.3-gram injection of neoarsphenamine as the first dose of a fifth course of treatment. On January 27 he was given a 0.6-gram injection, and about 15 minutes later had chills, was nauseated, and tried to vomit. In the afternoon he felt well, except for general malaise and a dull headache. Urinalysis was negative. Dickens' test was positive.

He was returned to duty the next day feeling well.

(21—1934) A patient who gave no history of exposure developed generalized adenopathy. A Kahn blood test was positive and two recheck Kahn's were positive.

Treatment was started on February 4, 1934, with a 0.3-gram injection of neoarsphenamine. On February 7 he was given a 2-grain intramuscular injection of bismuth salicylate, and on February 11 a 0.45-gram injection of neoarsphenamine. Seven hours later he developed a headache, slight pain over splenic area, and a rise of temperature to 102.8° F. After an injection of sodium thiosulphate, an Autenreith's test showed delayed arsenic elimination. Blood: White blood count, 6,420. Differential: Polymorphonuclears, 84; lymphocytes, 16. His temperature returned to normal and the next day he was returned to duty.

Heavy metal therapy was continued, and on May 14, 22, and 28 he was given 2-grain intramuscular injections of bismuth salicylate.

(22, 23—1934) Two reactions occurred in the same individual. A patient, who was exposed October 5, 1933, developed a chancre on the glans penis which was positive for *Treponema pallidum* on October 20. On October 30 a Kahn blood test was 2 plus.

Treatment was immediately instituted and by December 6 he had completed a course of eight injections of neoarsphenamine, total dosage 4.5 grams. From December 6 to December 20 he received potassium iodide in ascending doses 3 times daily, and from December 6 to December 30 he received 22 mercury inunctions.

On January 2, 9, and 16, 1934, he received 1 cubic centimeter intramuscular injections of bismosol. On January 17 he received a 0.3-gram injection of neoarsphenamine and on January 24 and 31, 0.6-gram injections. About 1 hour later the patient reported to the sick bay complaining of a chill, dizziness, frontal headache, and weakness. Temperature, 97.8° F.; pulse, 88; respirations, 18. Blood pressure, 104/70. The chill lasted about 30 minutes. The temperature made a rapid rise and reached a maximum of 102.2° F. His pulse varied from 88 to 120. He was restless and vomited three times. His skin was flushed and dry. Urinalysis was negative. Blood: White blood count, 5,700. Differential: Polymorphonuclears, 61; large lymphocytes, 16; small lymphocytes, 20; eosinophiles, 1; transitionals, 1; mononuclears, 1. The symptoms cleared up in about 8 hours. He was discharged to duty the following morning at which time a urinalysis was normal.

On February 14, treatment was resumed and a 0.3-gram injection of neoarsphenamine was administered. In 15 minutes he complained of chills, dizziness, frontal headache, weakness, nausea, and vomiting. Temperature, 97.4° F.; pulse, 90; respiration, 20; blood pressure, 110/72. Temperature reached a maximum of 100.8° F. His pulse varied from 90 to 129. His skin was dry and flushed. Urinalysis was normal. Blood: White blood count, 12,200. Differential: Polymorphonuclears, 69; large lymphocytes, 18; small lymphocytes, 9; eosinophiles, 1; transitionals, 2; mononuclears, 1. The symptoms disappeared within 8 hours and he was discharged to duty the following morning.

(24—1934) A patient who was exposed in May 1928 developed a penile lesion on June 14. On June 19 a dark-field examination was positive for *Treponema pallidum*, and on June 20 a Kahn blood test was negative.

Treatment was administered as follows: June 19, 1928, to April 23, 1929, 14 injections of neoarsphenamine and 70 mercury inunctions; October 30, 1930, to April 20, 1931, 24 injections of bismuth salicylate; February 16, 1932, 1 injection of neoarsphenamine. The patient stated that neoarsphenamine had caused him to have a severe reaction and that its use had been discontinued. His health record showed no entry to substantiate this statement.

Because of a 4-plus Kahn blood test on May 22, 1934, heavy-metals therapy was instituted, and during the period June 21 to September 19, 1934, he was given 16 injections of mercury. On November 6, 1934, he was given a 0.1-gram injection of neoarsphenamine. On November 13 he was excused from taking arsenical treatment because of eating a full breakfast, and on November 20 he was on leave. On November 27 he was given a 0.2-gram injection of neoarsphenamine. About 3 minutes later he complained of dyspnea and weakness with pain in his lower back. He had marked erythema of the skin, especially on the chest and face. His conjunctivae were intensely injected. Temperature, 100° F.; pulse, normal. Urinalysis, normal.

Recovery in about 5 hours.

(25–1934) A patient, who was exposed on December 1, 1933, developed a primary lesion which was positive for *Treponema pallidum* on December 27.

Treatment was instituted on December 27, the patient receiving 0.3 gram of neoarsphenamine. Treatment was continued and on December 30, January 4, 6, 11, 13, 18, and 20 he was given 0.45-gram injections, an average of 138 milligrams per day. As concurrent treatment he was given 0.13-gram intramuscular injections of bismuth salicylate on January 2, 8, and 15.

Approximately 8 hours after the last injection of neoarsphenamine the patient complained of cramps in his upper abdomen. The cramps gradually became more severe and he was nauseated but did not vomit. He then had some difficulty in breathing. His skin was cold and clammy. Blood: White blood count, 9,600; Differential: polymorphonuclears, 61; small lymphocytes, 22; large lymphocytes, 4; transitionals, 2; blood pressure, 110/70; pulse, 110; respirations, 28.

Recovery within 4 hours.

(26–1934) A patient developed a primary lesion which was positive for *Treponema pallidum* on March 4, 1932.

From March 4, 1932, to August 8, 1933, he received 22 injections of neoarsphenamine, 8 injections of bismuth salicylate, 19 days' treatment with potassium iodide given three times daily, and 15 days' treatment with specific mixture. He then received specific mixture for 16 days in November and December 1933 and for 15 days in February 1934.

On May 11, 1934, he was given 0.3 gram of neoarsphenamine. About 15 minutes later the patient stated he had a headache and felt dizzy. He then developed a severe headache, dizziness, and muscular pains in the back and legs. Blood pressure, 126/56; temperature, 100° F.; pulse, 92; respirations, 20; white blood count, 2,300; Differential: polymorphonuclears, 65; lymphocytes, 35.

Recovery in 4 hours.

(27—1934) A patient had a penile lesion and inguinal adenopathy on August 24, 1933. Repeated dark-field examinations of the lesion were negative for *Treponema pallidum*. On September 7 he had a generalized macular rash and positive blood serology.

Treatment was immediately instituted, and by November 7 he had completed a course of 12 injections of neoarsphenamine and 16 injections of bismosol.

A second course of arsenical treatment was instituted on January 18, 1934, at which time the patient was given a 0.3-gram injection of neoarsphenamine. On January 25 he was given a 0.45-gram injection. Approximately 2 minutes later he developed shortness of breath, a cough, and generalized flushing of the skin. Pulse was around 120. He had nausea but did not vomit. Later he developed a chill and fever. Temperature, 100.8° F.; pulse, 110; respirations, 22; urine, negative; Dickens' test, normal. Blood: White blood count, 10,200. Differential: band forms, 2; segmented, 87; lymphocytes, 10; eosinophiles, 1.

Recovery in 48 hours.

(28—1934) A patient, who was infected in December 1932, received 10 injections of neoarsphenamine and 10 injections of bismosol between January 4 and April 6, 1933.

From July 17 to July 28, 1933, he received three injections of neoarsphenamine. Following the last injection the patient developed a mild vasomotor reaction with recovery in 3 days. He was then continued on mercury inunctions.

During the period of August 8 to October 17, 1933, he received 10 injections of bismuth salicylate.

From March 20 to May 25, 1934, he was given 10 injections of neoarsphenamine and 8 injections of bismuth salicylate.

On June 29, 1934, he received 0.3 gram of neoarsphenamine as the first injection of a fourth course. About 30 minutes later the patient complained of severe headache, numbness of both legs, and nausea. Temperature, 100.6° F.; pulse, 102; respirations, 22; blood pressure, 130/78; urinalysis, negative; Dickens' test, positive. Blood: White blood count, 8,000; Differential: Polymorphonuclears, 56; eosinophiles, 1; basophiles, 1; monocytes, 4; small lymphocytes, 24; large lymphocytes, 14.

Recovery in 10 hours.

(29—1934) A patient, who was exposed on February 5, 1934, developed a small penile lesion which rapidly healed. Slight inguinal adenopathy developed and on February 26 and March 12 Kahn blood tests were 3 plus.

On March 13 the patient was administered a 0.3 gram injection of neoarsphenamine and on March 20, 23, 27, April 4, 10, and 17 he was administered 0.6 gram injections, an average of 108 milligrams per

day. As concurrent treatment he was given 1 grain intramuscular injections of bismuth salicylate on March 16, 20, 23, 27, April 4, 6, and 13.

Five hours after the last injection of neoarsphenamine the patient felt feverish and developed an occipital headache. One hour later his temperature was 101.4° F.; pulse, 100; respirations, 20. At this time he experienced continuous dull aching pain in the wrist joints and a sensation of pressure over the metacarpals. Two hours later his temperature was 100.6° F.; pulse, 90; respirations, 18. At this time the pain in wrists had decreased. Urine negative.

Recovery in 24 hours.

(30—1934) A patient, whose date of infection was unknown because he had been exposed on numerous occasions, was given a diagnosis of syphilis on November 17, 1933, because of an extensive venereal history, palpable epitrochlear nodes, and a 4-plus Kahn blood test.

By June 23, 1934, he had completed 10 injections of neoarsphenamine for a total dosage of 5.7 grams and five 1 cubic centimeter intramuscular injections of bismuth.

Two and one-half hours after the last injection of neoarsphenamine, administered on June 23, the patient had a severe chill, headache, and vomited about a half ounce of blood. The patient stated he had jaundice while taking previous injections of neoarsphenamine, although no entry was in his health record to that effect. Dickens' test was positive. Blood: Red blood count, 4,800,000; white blood count, 9,800. Differential: polymorphonuclears, 73; lymphocytes, 18; monocytes, 9. Temperature, 102° F.; pulse, 120. He developed pain in his stomach and lumbar region. His temperature, pulse, and renal function returned to normal in 2 days, but he was retained on the sick list under observation until July 3, 10 days after the onset of symptoms.

(31—1934) A patient, who was exposed on June 18, 1932, developed a penile lesion on July 7, the serum from which was positive for *Treponema pallidum* on July 8.

From July 14, 1932, to June 1, 1934, he received 21 injections of neoarsphenamine, total dosage 8.781 grams, 43 injections of bismuth salicylate, and 32 days' treatment with specific mixture.

On July 12, 1934, he received a 0.227-gram injection of neoarsphenamine as the first injection of a third course of arsenical treatment. On July 19 and 26 he was given 0.45-gram injections. About 10 minutes after the last injection the patient vomited and complained of headache and numbness of both legs. The conjunctivae were injected. Temperature, 100.4° F.; pulse, 108; respirations, 24. Urine negative. Dickens' test positive. Blood: Red blood count, 5,250,000; White blood count, 16,000; Differential: Segmented, 82; band forms, 11; lymphocytes, 5; monocytes, 2.

Recovery in 23 hours.

(32—1934) A patient, who was infected in September 1932, completed three courses of neoarsphenamine for a total of 28 injections on September 14, 1933. He also received five injections of mercury salicylate as intercurrent treatment.

On January 18, 1934, a fourth course of neoarsphenamine treatment was instituted, the patient receiving a 0.3-gram injection. On January 25, February 1, 8, and 15 he received 0.45-gram injections.

About 2 minutes after the last injection the patient felt a choking sensation and a tightness in his chest with difficulty in breathing. Temperature, 97.8° F.; pulse, 90; respirations, 26. His face became greatly flushed and the entire body surface was purplish. His conjunctivae were markedly injected and there were musical rales in the chest. He developed a chill which was followed by an elevation in temperature to 101° F. Urine negative. Epinephrine promptly relieved the chest symptoms and the marked flushing of the skin.

Recovery in 24 hours.

(33—1934) A patient who was infected in October 1932, received 15 injections of neoarsphenamine and 6 injections of sulpharsphenamine between the dates of November 3, 1932, and June 27, 1933.

On March 23, 1934, he was given a 0.6-gram injection of neoarsphenamine as the first injection of a fourth course of arsenical treatment. He also received 0.6-gram injections on March 23, 26, 30, April 3, 6, and 14, an average of 157 milligrams per day. As concurrent treatment he was given 4 intramuscular injections of bismuth salicylate and 2 mercury inunctions.

Several hours after the last injection the patient complained of nausea, vomiting, and cramplike abdominal pain. He became cold, perspired profusely, and his skin became cold and clammy. Temperature, 97.8° F.; pulse, 68; respirations, 18. Urine, negative. Dickens' test, positive.

Recovery in 15 hours.

(34—1934) A patient developed a primary lesion which was positive for *Treponema pallidum* on September 17, 1934. He was given 0.3-gram injections of neoarsphenamine on September 17, 20, and 29. Two hours after the last injection he was admitted to the sick list complaining of nausea. He later vomited, had edema of the face, and a macular rash appeared on the flexor surface of his arms. His subjective symptoms were headache and anuria. He had hematuria with the first urination. He also had gonorrhea. Blood: Red blood count, 4,730,000; white blood count, 10,400. Differential: Juveniles, 1; band forms, 21; segmented, 71; lymphocytes, 6; Turck's cell, 1. Urine: On specimens passed at 9 p. m., September 29, and 2 a. m., September 30. Appearance, cloudy, straw; reaction, acid; specific gravity, 1.010; albumin, 3 plus; sugar, negative. Microscopical: Mucus, slight amount; leucocytes, 70 to 90 per field; erythrocytes,

5 to 6 per field; epithelium, few squamous. Dickens' test showed no elimination of arsenic in either specimen.

September 30. Temperature dropped to 99.4° at noon but rose to 102° F. that night.

October 1. Temperature 99.4° F. Some rash still on arms. No subjective symptoms. Urine: Appearance, water-clear; reaction, acid; specific gravity, 1.001; albumin and sugar negative. Microscopical: Leucocytes, loaded, too many to count; epithelium, few squamous.

October 2. Temperature normal, no rash and no subjective symptoms. To duty, 68 hours after onset.

(35, 36, 37–1934) Three reactions occurred in the same individual. A patient, who was exposed on July 24, 1933, developed a primary lesion on coronal sulcus of penis on August 1. On August 21 a dark-field examination was positive for *Treponema pallidum*. A Kahn blood test was 4 plus the same date.

From August 17, 1933, to July 15, 1934, he received 20 injections of neoarsphenamine and 42 injections of bismuth salicylate.

On September 13, 1934, he received 0.3 gram of neoarsphenamine, on September 20, 0.45 gram, and on September 27 and October 4, 0.6 gram injections. Following the injection of October 4, and after supper, the patient complained of a headache which lasted about 3 hours.

On October 18 he was given a 0.45 gram injection and after supper he had the same kind of headache as experienced after the previous injection. Blood: White blood count, 5,000. Differential: Mature neutrophiles, 52; immature neutrophiles, 7; lymphocytes, 41.

On October 25 he was given 0.6 gram of neoarsphenamine and after supper he had a similar headache which lasted about 4 hours.

(120—1934) Two reactions occurred in the same individual. A patient, who was infected on January 24, 1934, developed generalized adenopathy, a 4 plus Kahn blood test on March 26, and a secondary rash on March 27.

On March 28 he received 0.3 gram of neoarsphenamine. About 6 hours later he had slight headache and pain in his joints which lasted for about 2 hours. He slept well all night and felt perfectly well the next morning. He was given a 0.4 gram injection of neoarsphenamine on April 3 which caused a mild reaction, described under Jarisch-Herxheimer reactions, case no. 121—1934.

Sulpharsphenamine.—(38—1934) A patient, who was infected September 13, 1934, developed generalized adenopathy on October 4 and a 4-plus Kahn blood test on October 8.

On October 9 he received a 0.2 gram intramuscular injection of sulpharsphenamine and on October 16, a 0.4 gram injection of the same drug. As concurrent treatment he received 8 days' treatment with specific mixture, administered three times daily.

About 8 hours after the last injection of sulpharsphenamine he complained of feeling hot and weak. Temperature, 105° F.; pulse, 100; respirations, 22. Physical examination was negative except for slight injection of the posterior pharynx. Blood: White blood count, 6,050; Differential: band forms, 8; segmented, 84; lymphocytes, 1; monocytes, 2.

October 17. Blood: Red blood count, 5,990,000; hemoglobin, 116 percent; white blood count, 6,200; Differential: band forms, 31; segmented, 54; lymphocytes, 7; eosinophiles, 2; monocytes, 1; myelocytes, 1; juveniles, 4.

October 18. Temperature, 100.8 to 102.6° F. Patient much improved. Sedimentation index 9. Sedimentation time, 1 hour. Blood: White blood count, 5,200; Differential: myelocytes, 1; juveniles, 6; band forms, 40; two lobed neutrophiles, 32; three lobed neutrophiles, 8; four lobed neutrophiles, 1; lymphocytes, 9; monocytes, 3.

October 19. Blood: White blood count, 5,250; Differential: juveniles, 3; band forms, 31; segmented, 33; lymphocytes, 25; eosinophiles, 3; basophiles, 2; monocytes, 3.

October 20. Blood: White blood count, 5,250; Differential: juveniles, 5; band forms, 31; segmented, 23; lymphocytes, 27; eosinophiles, 8; basophiles, 1; monocytes, 5.

October 21. Temperature, pulse, and respirations, normal. Has slight urethral discharge.

October 22. Blood: White blood count, 5,500; Differential: juveniles, 3; band forms, 7; segmented, 55; lymphocytes, 30; eosinophiles, 5.

October 27. No urethral discharge. Treatment for prostatitis continued.

October 29. Urine: Appearance, straw, clear; reaction, acid; specific gravity, 1.018; albumin and sugar, negative. Microscopical, negative. Blood: Red blood count, 5,200,000; hemoglobin, 80 percent; white blood count, 7,100; Differential: band forms, 5; segmented, 62; lymphocytes, 29; eosinophiles, 4.

October 30. To duty, in 14 days.

(39—1934) A patient, whose time and place of infection was unknown, developed an irregularly distributed papulopustular eruption over the trunk and extremities on February 24, 1933. On March 30 a Kahn blood test was 4 plus.

From April 5, 1933, to April 13, 1934, he received 18 injections of neoarsphenamine, and from April 5, 1933, to October 26, 1934, he received 38 injections of bismuth salicylate. The patient had a history of chills and fever following each of the last four injections of neoarsphenamine administered on March 16, 23, April 6 and 13, 1934.

On October 30, 1934, at 9 a. m., he received a 0.2-gram intramuscular injection of sulpharsphenamine as the first injection of a fourth course of arsenical treatment. About 1½ hours later he had a severe chill. Temperature, 97.2° F.; pulse, 80; respirations, 18. Blood at 10:45 a. m.: White blood count, 4,300. Differential: juveniles, 4; band forms, 26; segmented, 67; lymphocytes, 3. The chill lasted about 1½ hours. He had no complaints other than being cold. Blood at 1:30 p. m.: White blood count, 5,400; Differential: segmented, 36; lymphocytes, 61; eosinophiles, 3. Temperature rose to 103.8° F. at 1:50 p. m. By 8 p. m. the temperature, pulse, and respirations were normal and the patient felt well.

October 31. Urine: Appearance, pale straw, clear; reaction, acid; specific gravity, 1.001; albumin, faint trace, negative 1 to 5; sugar, negative. Microscopical, negative. Blood: Hemoglobin, 96 percent; white blood count, 7,400; Differential: band forms, 5; segmented, 58; lymphocytes, 30; monocytes, 6; eosinophiles, 1. To duty this date.

(40, 41—1934) Two reactions occurred in the same individual. A patient, who was infected in June 1934, was administered a 0.2-gram intramuscular injection of sulpharsphenamine on July 31, 1934. On August 7 he received a 0.4-gram injection of the same drug. About 9 hours later he had loss of appetite and chilly sensations. On August 10 he was admitted to the sick list with a temperature of 102.2° F., complaining of chills and dizziness, and showing two areas of Vincent's infection around the lower third molars. In the morning he had received a prostate massage for gonococcus infection of prostate. Urine: Appearance, amber, cloudy; reaction, acid; specific gravity, 1.025; albumin, 1 plus; sugar, negative. Microscopical: leucocytes, too numerous to count; epithelium, some round cells. Blood: White blood count, 12,750; Differential: band forms, 23; segmented, 65; lymphocytes, 11; basophiles, 1.

August 11. Temperature 104° to 104.2° F. He was nauseated and vomited. No pain or tenderness in abdomen, chest clear, bowels constipated. Urine: Appearance, straw, cloudy; reaction, alkaline; specific gravity, 1.025; albumin, 1 plus; sugar, negative. Microscopical: many bacteria; much mucus; leucocytes, too numerous to count; erythrocytes, 25 to 35 per high dry field; epithelium, some round cells; crystals, amorphous phosphates. Blood: White blood count, 12,700; Differential: juveniles, 5; band forms, 41; segmented, 40; lymphocytes, 11; monocytes, 3. Smear negative for organisms of Vincent's infection.

August 12. Temperature, 99 to 99.6° F.; pulse, 76 to 90; respirations, 20. Bowels open. Patient felt well. Smear from mouth for Vincent's infection showed numerous fusiform bacilli, few spirilla of Vincent's. Icterux index 6. Van den Bergh's, indirect reaction

negative, direct reaction less than 0.1 milligram bilirubin per 100 cubic centimeters of blood.

August 13. Temperature, pulse, and respirations, normal.

August 14. He received a 0.2-gram intramuscular injection of sulpharsphenamine. About 6 hours later he had a chill, which was followed by rise in temperature, slight nausea, and headache. Temperature, 103° F.; pulse, 98; respiration, 18.

August 15. Temperature, 99°; pulse, 86; respiration, 18. The patient felt fine. Urine: Appearance, slightly cloudy, amber; reaction, acid; specific gravity, 1.024; albumin, 1 plus; sugar, negative: Microscopical: Occasional hyaline cast; leucocytes, too many to count; erythrocytes, 4 to 6 per high dry field; epithelial cells, few. A blood culture showed no growth.

On August 16 his temperature, pulse, and respirations were normal and he was symptom free.

(42, 43, 44—1934) Three reactions occurred in the same individual. The first reaction followed an injection of neoarsphenamine, and the second and third reactions followed injections of sulpharsphenamine. The patient was given a diagnosis of syphilis on March 21, 1933, because of a primary lesion on his glans penis, sore throat with mucous patches on tonsils, headache, papular rash on chest, and a 4-plus Kahn blood test.

From March 21 to October 20, 1933, he received 9 injections of neoarsphenamine and 12 injections of bismuth. Following the last two injections of neoarsphenamine the patient had nausea, vomiting, abdominal cramps, and diarrhea.

From February 1 to April 15, 1934, he received 74 days' treatment with specific mixture.

On October 16, 1934, he received a 0.2-gram injection of sulpharsphenamine and on October 23 a second 0.2-gram injection of the same drug. Five minutes later he complained of fullness in his head and severe headache. Blood pressure 104/66. Temperature, 98; pulse, 70; respiration, 20. Fifteen minutes later he had a slight chill, vomited once, and complained of a dull pain in his stomach. His temperature rose to 100° F.; pulse, 84; respirations, 22. He recovered within 12 hours. He was continued on the sick list in order to determine ability to take some other type of arsenical.

November 13. Urine: Appearance, clear, amber; reaction, acid; specific gravity, 1.022; albumin and sugar, negative. Microscopical: Considerable mucus and squamous epithelium.

November 20. Administered sulpharsphenamine, 0.1 gram intramuscularly, at 9 a. m. One hour later temperature rose to 100° F., and conjunctivae were slightly injected. Blood: 10:30 a. m., white blood count, 10,700. Differential, band forms, 11; segmented, 75; lymphocytes, 14. At 1:30 p. m.: White blood count, 15,200; Differ-

ential: juveniles, 2; band forms, 17; segmented, 72; lymphocytes, 9. Urine: Appearance, clear, amber; reaction, neutral; specific gravity, 1.004; albumin and sugar, negative. Microscopical: No abnormalities. At 3 p. m., 6 hours after onset of symptoms, he had apparently recovered. At 10:45 p. m., white blood count, 4,300. Differential: band forms, 3; segmented, 59; lymphocytes, 35; eosinophiles, 1; monocytes, 2.

November 27. Administered sulpharsphenamine, 0.1 gram intramuscularly, at 9 a. m. One hour later the patient felt weak and cold and complained of headache and abdominal discomfort. His conjunctivae were slightly injected. Temperature, 99.2° F.; pulse, 80. At noon, temperature, 100.2° F.; pulse, 84. At 1:45 p. m., white blood count, 14,000. Differential: band forms, 12; segmented, 74; lymphocytes, 8; eosinophiles, 6. Urine negative. By 3 p. m., the temperature, pulse, and respirations were normal and he felt well.

November 28. Van den Bergh's test: Direct reaction negative; indirect reaction, less than 0.5 milligram of bilirubin per 100 cubic centimeters of blood serum.

December 1. Symptom free. To duty on December 4.

(45—1934) A patient, who was infected in April 1934, developed an ulcer at the peno-scrotal junction. A dark-field examination was positive for *Treponema pallidum* on May 22 and a Kahn blood test was 4 plus on May 24. By September 26, 1934, he had received 9 injections of neoarsphenamine and 4 injections of thio-bismol. Treatment with specific mixture was started on September 29, 1934, and continued until October 23, 1934.

On October 2, 1934, the patient was given a 0.2 gram intramuscular injection of sulpharsphenamine. On October 9, a second 0.2 gram was administered and on October 16 and 23, 0.4 gram injections were administered. For 1 hour prior to the last injection the patient had been working in the engine room. He received the injection of sulpharsphenamine at 9 a. m. and returned to his duties. One and one-half hours later he began to feel dizzy, became nauseated, and vomited. His bowels had not moved for 2 days. Temperature, pulse, and respirations were normal. Blood pressure 120/80. White blood count, 6,700; Differential: band forms 2; segmented 52; lymphocytes 43; monocytes 3.

October 24. White blood count, 9,200; Differential: band forms 6; segmented 68; lymphocytes 20; eosinophiles 1; monocytes 5.

October 25. The patient had diarrhea. He felt well except for dizziness when walking.

October 27. No symptoms.

October 28. Returned to duty, under treatment for syphilis and gonococcus infection of urethra.

(46—1934) A patient, who was infected in March 1932, developed a maculopapular rash on May 30 and a 4-plus Kahn blood test on May 31, 1932.

From May 30 to October 9, 1932, he received 21 injections of neoarsphenamine, and from May 30, 1932, to September 28, 1934, 68 injections of bismuth.

On October 9, 1934, he was given a 0.2-gram intramuscular injection of sulpharsphenamine. On October 16 he received a second 0.2-gram injection, and on October 23, a 0.4-gram injection. As concurrent treatment he was given specific mixture.

At 9:30 a. m., 30 minutes after the last injection, the patient felt dizzy, became nauseated, and vomited. He vomited several times and had six watery stools. He felt weak, had a slight headache, and slight injection of conjunctivae. Temperature, pulse, and respirations were normal. Blood: Red blood count, 4,860,000; hemoglobin, 85 percent; white blood count, 4,200; Differential: band forms, 46; juveniles, 13; segmented, 34; lymphocytes, 7. After an injection of sodium thiosulphate he went to sleep, and upon awakening, about 4 p. m., felt well.

The next day he was apparently fit for duty. Blood: White blood count, 10,900; Differential: band forms, 9; segmented, 74; lymphocytes, 14; eosinophiles, 2; monocytes, 1.

TABLE REACTIONS

Neoarsphenamine.—(47—1934) A patient was infected in October 1932.

From November 1, 1932, to August 29, 1933, he received 16 injections of neoarsphenamine, total dosage 7.65 grams.

On February 12 and 17, 1934, he received 0.3 gram injections of neoarsphenamine. Immediately following the last injection the patient's face became flushed, conjunctivae reddened and suffused. Dyspnea was definite. His pulse was rapid, weak, and thready.

The patient felt well in 2 hours, except for a mild headache.

(48—1934) A patient was infected in May 1933.

From June 20 to December 12, 1933, he received an unstated number of injections of neoarsphenamine for a total dosage of 7.2 grams.

On January 30, 1934, he received a 0.3 gram injection of neoarsphenamine and on February 6, 13, 20, 27, and March 6, 0.45 gram injections. Two minutes after the last injection the patient developed swelling of the face and tongue, flushing and giddiness. Recovery within 10 minutes.

(49—1934) A patient was infected in June 1933.

From July 24 to December 5, 1933, he received 19 injections (11.25 grams) of neoarsphenamine and 17 injections of mercury. The pa-

tient gave a history of slight chills, pain over body, and vomiting during the latter part of his treatment.

He received 0.13 gram intramuscular injections of bismuth salicylate on June 7, 15, and 22, 1934. On June 14, 21, and 28 he received 0.3 gram, 0.45 gram, and 0.45 gram injections of neoarsphenamine, respectively. Immediately after the last injection of neoarsphenamine the patient vomited twice, and 15 minutes later had a chill which lasted about 15 minutes. He was given sodium thiosulphate and all symptoms cleared up in about 10 minutes.

50, 51—1934) Two reactions occurred in the same individual. The patient was infected in October 1933.

From November 2, 1933, to January 18, 1934, he received 11 injections (4.65 grams) of neoarsphenamine and 10 injections (1.3 grams) of bismuth salicylate. Following an injection of neoarsphenamine on November 23, 1933, the patient had a table reaction of 30 minutes' duration.

On March 15, 1934, the patient received a 0.3 gram injection of neoarsphenamine, and on March 22, 29, and April 5, 12, and 19, 0.45 gram injections. Immediately after the last injection the patient developed headache, nausea, and a flushed face. He was given adrenalin intramuscularly and ephedrine orally. In about 20 minutes the symptoms cleared.

On April 26 he was given 0.45 gram of neoarsphenamine and immediately complained of headache and nausea. He was given adrenalin intramuscularly. He recovered in 15 minutes.

(52—1934) A patient was infected in September 1925.

From October 28, 1925, to May 30, 1934, he received 104 injections of neoarsphenamine and 159 injections of bismuth. The patient stated he had nausea and vomiting with loss of appetite following injections, when first treated, which he did not report.

On November 15 and 25, 1934, Kahn blood tests were 4 plus. Arsenical treatment was deemed advisable and on November 28, 1934, he received a 0.3 gram injection of neoarsphenamine. Immediately after the injection the patient became nauseated and vomited three or four times. He recovered from vomiting in a few minutes but felt nauseated for 5 hours. The next day he was up and about but had no appetite for 1½ days.

(53—1934) A patient was infected in February 1933.

From February 22, 1933, to January 4, 1934, he received 23 injections of neoarsphenamine and 23 injections of bismuth.

On February 24, 1934, he received a 0.3 gram injection of neoarsphenamine and on March 3, a 0.45 gram injection. Approximately 5 minutes later the patient began to feel weak and dizzy and then became very pale.

Recovery within 30 minutes.

(54—1934) A patient was infected in February 1930.

From March 5, 1930, to February 12, 1934, he received 56 injections of neoarsphenamine.

On April 3, 1934, he was given a one-fifth grain injection of mercury succinimide and a 0.13 gram injection of bismuth salicylate. The next day he was given a 0.3 gram injection of neoarsphenamine. About 10 minutes later he became flushed, his eyes suffused, and he felt nauseated and retched.

He felt fairly well 1 hour later, except for a dull headache.

(55—1934) A patient was infected in April 1931.

From May 19, 1931, to October 26, 1932, he received 70 injections of neoarsphenamine and 36 injections of bismuth salicylate. He then received 8 injections of bismuth salicylate between the dates of November 6, 1933, and January 15, 1934.

On January 25, 1934, he received a 0.3 gram injection of neoarsphenamine and on February 1 a 0.45 gram injection. During the last injection the patient became very pale and weak. He was given sodium thiosulphate and within 30 minutes made a complete recovery.

(56, 57, 58–1934.) Three reactions occurred in the same individual. The patient was infected in July 1933.

From August 18, 1933, to August 15, 1934, he received 28 injections of neoarsphenamine and 36 injections of bismuth salicylate.

On September 13, 20, and 27, 1934, he received 0.3 gram, 0.45 gram, and 0.6 gram injections of neoarsphenamine respectively. Immediately following the last injection the patient became nauseated. Thirty minutes later he recovered.

On October 11 he received a 0.3 gram injection of neoarsphenamine which was immediately followed by slight nausea. Thirty minutes later he recovered.

On October 18 he received a 0.45 gram injection of neoarsphenamine which was immediately followed by nausea and flushing of the face. Temperature, 99.4° F. White blood count, 6,500; Differential: mature neutrophiles, 55; immature neutrophiles, 3; lymphocytes, 42. He was given treatment with adrenalin and 30 minutes later he had recovered.

(59–1934) A patient was infected in December 1930.

During January 1931 he received 3 injections of neoarsphenamine and 3 injections of bismuth. From August to December 1933 he received 10 injections of neoarsphenamine.

On January 20, 1934, he received a 0.3 gram injection of neoarsphenamine and on January 27 and February 3, 0.45 gram injections. Five minutes after the last injection the patient became pale and felt weak and dizzy. Blood: Red blood count, 4,536,000; white blood count, 10,500; Differential: polymorphonuclears, 72 percent. Blood pressure, 110/70. Temperature, 98.8° F.; pulse, 96. Recovery within 1 hour.

Sulpharsphenamine.—(60, 61—1934) Two reactions occurred in the same individual. The patient was infected in 1931. In December 1933 he developed general adenopathy and a faint macular rash over chest and back. Kahn blood tests were 4 plus on December 14 and 18, 1933.

On December 22 and 26, 1933, he received 0.2 gram intramuscular injections of sulpharsphenamine. On January 2 and 9, 1934, he received 0.4 gram injections. Immediately after the last injection the patient began to sneeze and was nauseated. Examination revealed slight congestion of the mucous membranes of the nose and throat. Five minutes later he had no complaints.

On January 16 he received a 0.4 gram intramuscular injection of sulpharsphenamine and immediately he began sneezing, which lasted for about 5 minutes. He was nauseated but did not vomit. His eyes were injected and he had edema of the face, neck, and mucous membranes of the nose and throat.

Recovery was complete within 1 hour.

HERXHEIMER REACTIONS

Neoarsphenamine. (118—1934) A patient had a history of chancroidal infection in 1933. On September 3, 1934, he was admitted to the sick list with inguinal bubo. Kahn blood tests were 4 plus on November 1 and 6.

On November 6 he received a 0.45 gram injection of neoarsphenamine and a 0.1 gram intramuscular injection of bismosol. About 7 hours later he developed headache, nausea, malaise, and a generalized rash.

Recovery in 2 days.

On November 18 and 29 he received injections of neoarsphenamine, 0.3 gram doses, with no untoward symptoms.

(119—1934) A patient was infected on July 16, 1934. A primary lesion developed on the glans penis on August 16, which was positive for *Treponema pallidum* by repeated darkfield examinations. A typical rash developed on the body and face on September 18, and on the same day a Kahn blood test was 4 plus.

On September 21 he received a 0.3 gram injection of neoarsphenamine. Six hours later the patient had a decided chill which was followed by a rise in temperature to 102° F. He had pain in his leg muscles and the rash on his face and body became more pronounced. There was no pruritus, dyspnea, or difficult swallowing.

Twenty-four hours later his temperature was normal and the rash was subsiding.

(121—1934) Two reactions occurred in the same individual. On March 28, 1934, the patient received a 0.3 gram injection of neoarsphenamine as the first injection of a first course of treatment. This

injection caused a mild vasomotor reaction of a few hours duration. (The reaction is described under vasomotor reactions case no. 120—1934.)

On April 3, 1934, he received a 0.4 gram injection of neoarsphenamine as the second injection of a first course of treatment. About 2 hours later he began to feel chilly and his joints began to ache. He went to bed and during the night he slept fairly well. The next morning he had another chill, felt nauseated, and vomited several times. His face was edematous, especially around the eyes. The conjunctivae were acutely inflamed. The skin about the face and neck was erythematous, with a profuse secondary macular rash over the trunk. He had a uriniferous odor and complained of severe headache and nausea. Fauces were reddened. Temperature 102° F.; pulse, 110 and thready. Urine: Appearance, amber, cloudy; reaction, acid; specific gravity, 1.018; albumin, heavy trace; sugar, negative. Microscopical, numerous epithelial cells, many white-blood cells, few red-blood cells, numerous hyaline and finely granular casts. Urine was negative for arsenic.

All symptoms had subsided by April 14, 11 days after the onset of symptoms.

(122—1934) A patient was infected on August 20, 1934. A primary lesion developed on the dorsum of his penis, posterior to the corona, on September 17. Numerous dark-field examinations were negative for *Treponema pallidum*. On October 19 a typical rash on arms and body and mucous patches in mouth were evident. A Kahn blood test was 3 plus on October 24. On October 23 the patient received 0.3 gram of neoarsphenamine. Ten hours later the rash on arms and body became more pronounced. The patient had a chill followed by a rise in temperature to 101° F. Six hours later he had recovered.

(123—1934) A patient (supernumerary, Naval Reserve Force), who was infected on November 25, 1933, developed a penile lesion on December 1, and secondaries on January 9, 1934. Kahn blood test was 4 plus.

He received 0.3 gram of neoarsphenamine on January 11, 0.45 gram on January 15, and 0.6 gram on January 18, 1934. As concurrent treatment he was given 2 grain intramuscular injections of bismuth salicylate on January 12 and 15. Immediately after receiving the last injection of neoarsphenamine the patient exposed himself to cold without properly protecting himself. Three and one-half hours later he had a severe shill which lasted about 1 hour, marked nausea, and some vomiting. His temperature rose to 102.8° F.; pulse, 104. The next morning a marked reddish discoloration of the skin was noted which first appeared on the back and gradually spread over the entire body surface. This discoloration varied considerably and

was best described as an erythema multiforme chiefly of a scarlatiniform nature. Each day the temperature varied from 101° in the morning to 103° F. at night. After 10 days the temperature subsided and with it the erythema. About the seventh day a slight jaundice was noted which lasted for 3 days. The patient was almost constantly nauseated but did little vomiting. At no time was there any noticeable induration of the skin or areas of moisture.

He was considered recovered in 10 days but was retained on the sick list under observation for 11 additional days and was discharged to home on February 8, 1934.

HEALTH OF THE NAVY

Based on statistical returns for diseases and injuries occurring in April, May, and June, 1935, the general admission rate was 438 per 1,000 per annum, as compared with 452, the rate for the corresponding months of 1934. The median rate for the second quarter, as indicated by the records of the preceding 5 years, is 497. The admission rate for disease was 371 per 1,000 per annum. The median or expected rate for the corresponding quarter of the previous 5 years is 443. The admission rate for accidental injuries was 65 per 1,000 per annum, as compared with 62, the 5-year median for the corresponding 3 months. The slightly increased rate was the result of an increase in accidents occurring within command but not connected with work.

A total of 638 cases of acute respiratory diseases were reported from all shore stations, catarrhal fever causing 67 percent of the admissions.

The following table shows the number of admissions for various communicable diseases for the quarter recorded at certain shore stations:

Station	Catarrhal fever	German measles	Tonsillitis, acute	Bronchitis, acute	Chickenpox	Scarlet fever
U. S. Naval Academy, Annapolis, Md. (midshipmen)	52	1	2	0	1	0
Marine Barracks, Quantico, Va	55	7	7	3	3	0
Navy Yard:						
Washington, D. C	5	0	3	0	0	1
Portsmouth, N. H	6	2	5	0	1	0
District headquarters, New York, N. Y	0	0	0	0	1	0
Submarine base, New London, Conn	7	0	2	2	0	2
U. S. Naval Training Station, Norfolk, Va	62	30	16	0	0	0
Marine Corps base, San Diego, Calif	28	11	8	0	0	0
U. S. Naval Training Station, San Diego, Calif	44	27	18	32	0	0
U. S. Naval Air Station, Sunnyvale, Mountain View, Calif	9	0	0	0	0	1
U. S. Naval Radio Station, communication activities, Wailupe, Oahu, T. H	0	0	0	0	1	0
Fleet air base, Canal Zone	18	0	1	1	0	0
Regimental hospital, Fourth Marines, Shanghai, China	24	0	7	4	0	0
Marine detachment, American Legation, Peiping, China	3	0	1	0	2	1

In addition to the above diseases, 1 case of cerebrospinal fever was reported by the United States Naval Training Station, San Diego, Calif. The patient was a recruit with one month's service.

The Navy Yard, Mare Island, reported 1 case of poliomyelitis, anterior, acute, in May.

The admission rate, all causes, for forces afloat was 372 per 1,000, as compared with 327 per 1,000, the rate for April, May, and June, 1934, and 444, the corresponding median rate for the preceding 5 years.

A total of 1,533 cases of acute respiratory diseases was reported by all ships during the second quarter of the year 1935, or a 30 percent decrease from the number of cases notified for the preceding quarter.

Five cases of cerebrospinal fever were reported from forces afloat as follows:

Rate	Age	Place of original admission	Date of admission	Length of service (years)	Disposition
F-3c	19	U. S. S. Pennsylvania	Apr. 9, 1935	1 3/12	Died Apr. 9, 1935.
Sea. 2c	18	U. S. S. New York	do	4/12	Duty May 4, 1935.
Sea. 1c	20	U. S. S. Idaho	May 6, 1935	1 3/12	Duty Aug. 27, 1935.
Sea. 2c	20	do	May 5, 1935	1 3/12	No disposition yet reported.
Do	19	U. S. S. Marblehead	May 15, 1935	1 9/12	Do.

Two cases of typhoid fever were notified by forces afloat during the quarter—1 from the U. S. S. *Tutuila* on May 19 and 1 from the U. S. S. *Augusta* on May 31.

The U. S. S. *Saratoga* reported 1 case of scarlet fever in April and the U. S. S. *Texas* 1 case in June.

Ships recorded admissions for chickenpox, distributed over the quarter, as follows:

Ship	April	May	June
U. S. S. Altair	0	2	0
U. S. S. Arizona	0	1	0
U. S. S. Henderson	2	2	0
U. S. S. Lexington	1	0	0
U. S. S. Pennsylvania	2	3	0
U. S. S. Saratoga (F. A. D.)	1	0	0
U. S. S. Williamson	0	1	0

A case of poliomyelitis, anterior, acute, was admitted on board the U. S. S. *Isabel* on May 28 and died at the United States Naval Hospital, Canacao, P. I., on May 31. Diagnosis was confirmed by autopsy and laboratory findings.

Summary of morbidity in the U. S. Navy for the quarter ended June 30, 1935

	Forces afloat, average strength, 73,963		Forces ashore, average strength, 36,656		Entire navy, average strength, 110,619	
	Admissions	Rate per 1,000	Admissions	Rate per 1,000	Admissions	Rate per 1,000
All causes	6,887	372.46	5,232	570.93	12,119	438.22
Disease only	5,768	311.94	4,499	490.91	10,267	371.26
Injuries	1,081	58.46	719	78.46	1,800	65.09
Poisonings	38	2.06	14	1.53	52	1.88
Communicable diseases transmissible by oral and nasal discharges (Class VIII):						
(A)	297	16.06	491	53.58	788	28.49
(B)	1,332	72.04	1,007	109.89	2,339	84.58
Venereal diseases	1,307	70.68	409	44.63	1,716	62.05

Deaths reported, entire Navy, during the quarter ended June 30, 1935

Cause—Disease		Navy			Marine Corps		Nurse Corps	Total
Primary	Secondary or contributory	Officers	Midshipmen	Men	Officers	Men		
Average strength		9,445	1,537	82,050	1,153	16,104	330	110,619
Abscess, peritonsillar	Meningitis, cerebral.			1				1
Appendicitis, acute	Obstruction, intestinal, from spastic or paralytic causes.			1				1
Cerebrospinal fever	None			1				1
Diabetes mellitus	Thrombosis, coronary.	1						1
Dilatation, cardiac, acute	Dementia praecox.	1						1
Hemorrhage, cerebral	None			1				1
Do	Arterial hypertension.					1		1
Myocarditis, chronic	None			1				1
Do	Embolism, cerebral.				1			1
Do	Thrombosis, coronary.	1		4				5
Leukemia	Nephritis, acute			1				1
Do	Pneumonia, lobar.			1				1
Nephritis, chronic	None			1				1
Obstruction, intestinal, from external causes.	None			1				1
Obstruction, intestinal, from spastic or paralytic causes.	None	1						1
Pylephlebitis	None	1						1
Pneumonia, broncho	None			1				1
Pneumonia, lobar	None			2				2
Pleurisy, suppurative	None	1						1
Poliomyelitis, anterior, acute	None			1				1
Thrombosis, cavernous sinus	None			1				1
Thrombosis, coronary	None			2				2
Syphilis	None			1				1
Tuberculosis, pulmonary, chronic	None			1				1
Do	Hemorrhage, pulmonary.			1				1
Do	Nephritis, chronic.			1				1
Do	Septicemia			1				1
Ulcer, stomach	Peritonitis, general, acute.			2				2
Valvular heart disease, mitral insufficiency	None					1		1
Total for disease		6		27	1	2		36

Deaths reported, entire Navy, during the quarter ended June 30, 1935—Contd.

Cause—Disease		Navy			Marine Corps			
Primary	Secondary or contributory	Officers	Midshipmen	Men	Officers	Men	Nurse Corps	Total
INJURIES AND POISONINGS								
Avulsion, skull	None					1		1
Burn, multiple	None			1				1
Crush, chest	None			2				2
Drowning	None	1		5				6
Do	Intracranial injury.			1				1
Fracture, compound, skull	None			2				2
Fracture, simple, skull	None			1				1
Fracture, simple, vertebrae, cervical.	None			1			1	2
Injuries, multiple, extreme	None	3		7	1			11
Intracranial injury	Meningitis, cerebrospinal, acute.			1				1
Strangulation, neck	Dementia, praecox.			1				1
Wound, gunshot:								
Abdomen	None			1				1
Head	None			1		3		4
Thorax	None					1		1
Poisoning, arsenic, acute	None			1				1
Total for injuries and poisoning.		4		25	1	5	1	36
Grand total		10		52	2	7	1	72
Annual death rate per 1,000:								
All causes		4.24		2.54	6.94	1.74	12.12	2.60
Disease only		2.54		1.32	3.47	.50		1.30
Drowning		.42		.29				.25
Poisoning				.05				.04
Other injuries		1.27		.88	3.47	1.24	12.12	1.01

ADMISSIONS FOR INJURIES AND POISONINGS, SECOND QUARTER, 1935

The following table, indicating the frequency of occurrence of accidental injuries and poisonings in the Navy during the second quarter, 1935, is based upon all form F cards covering admission in those months which have reached the Bureau:

	Admissions, April, May, and June 1935	Annual admission rate per 100,000	
		Second quarter, 1935	Year 1934
Injuries:			
Connected with work or drill	768	2,777	2,397
Occurring within command but not associated with work	512	1,851	2,064
Incurred on leave or liberty or while absent without leave	520	1,880	1,699
All injuries	1,800	6,509	6,160
Poisonings:			
Industrial poisoning	6	22	15
Occurring within command but not connected with work	35	126	244
Associated with leave, liberty, or absence without leave	11	40	15
Poisonings, all forms	52	188	273
Total injuries and poisonings	1,852	6,697	6,433

Percentage relationships

	Occurring within command				Occurring outside command	
	Connected with the performance of work, drill, etc.		Not connected with work or prescribed duty		Leave, liberty, or absent without leave	
	April, May, and June 1935	Year 1934	April, May, and June 1935	Year 1934	April, May, and June 1935	Year 1934
Percent of all injuries	42.7	38.9	28.4	33.5	28.9	27.6
Percent of all poisonings	11.5	5.3	67.3	89.3	21.2	5.3
Percent of total admissions, injury, and poisoning titles	41.8	37.5	29.5	35.9	28.7	26.6

Poisoning by a narcotic drug or by ethyl alcohol is recorded under the title "Drug addiction" or "Alcoholism", as the case may be. Such cases are not included in the above figures.

There were no cases during the second quarter of 1935 worthy of notice from the standpoint of accident prevention.

STATISTICS RELATIVE TO MENTAL AND PHYSICAL QUALIFICATIONS OF RECRUITS

The following statistics were taken from monthly sanitary reports submitted by naval training stations:

April, May, and June 1935	United States Naval Training Station	
	Norfolk, Va.	San Diego, Calif.
Recruits received during the period	1,161	1,503
Recruits appearing before Board of Medical Survey	4	0
Recruits recommended for discharge from the service	4	0
Recruits discharged by reason of Medical Survey	1	0
Recruits held over pending further observation	0	0
Recruits transferred to the hospital for treatment, operation, or further observation for conditions existing prior to enlistment	20	21

The following table was prepared from reports of medical surveys in which disabilities or disease causing the surveys were noted as existing prior to enlistment. With certain diseases, survey followed enlistment so rapidly that it would seem that many might have been eliminated in the recruiting office.

Cause of survey	Number of surveys	Cause of survey	Number of surveys
Absence, acquired, teeth	3	Gonococcus infection, urethra	1
Adhesions, abdominal	1	Gonococcus infection, epidymis	1
Arterial hypertension	3	Hernia, inguinal	2
Arthritis, chronic, both ankles	1	Malocclusion, teeth	2
Color blindness	1	Obesity	4
Constitutional psychopathic inferiority, without psychosis	2	Otitis, media, chronic	2
		Pyorrhea alveolaris	2
Constitutional psychopathic state, emotional instability	2	Stuttering	1
		Synovitis, chronic, both knees	1
Cystitis (nonvenereal), chronic	1	Syphilis	2
Deformity, acquired, right elbow joint	1	Union of fracture faulty	3
Deformity, acquired, prespondylolisthesis	1	Valvular heart disease, aortic insufficiency	1
Dislocation, articular cartilage, right knee	1	Valvular heart disease, aortic stenosis	1
Effort syndrome	1	Valvular heart disease, combined lesions, aortic and mitral	2
Enuresis	9		
Epilepsy	1	Valvular heart disease, mitral insufficiency	1
Flat foot	9	Valvular heart disease, mitral stenosis	3
Foreign body, right knee, gunshot	1	Varicose veins	1

VOLUME XXXIV APRIL 1936 NUMBER 2

United States Naval Medical Bulletin

PUBLISHED *for the* INFORMATION OF
MEDICAL DEPARTMENT *of the* NAVY

THE MISSION OF THE MEDICAL CORPS OF THE NAVY

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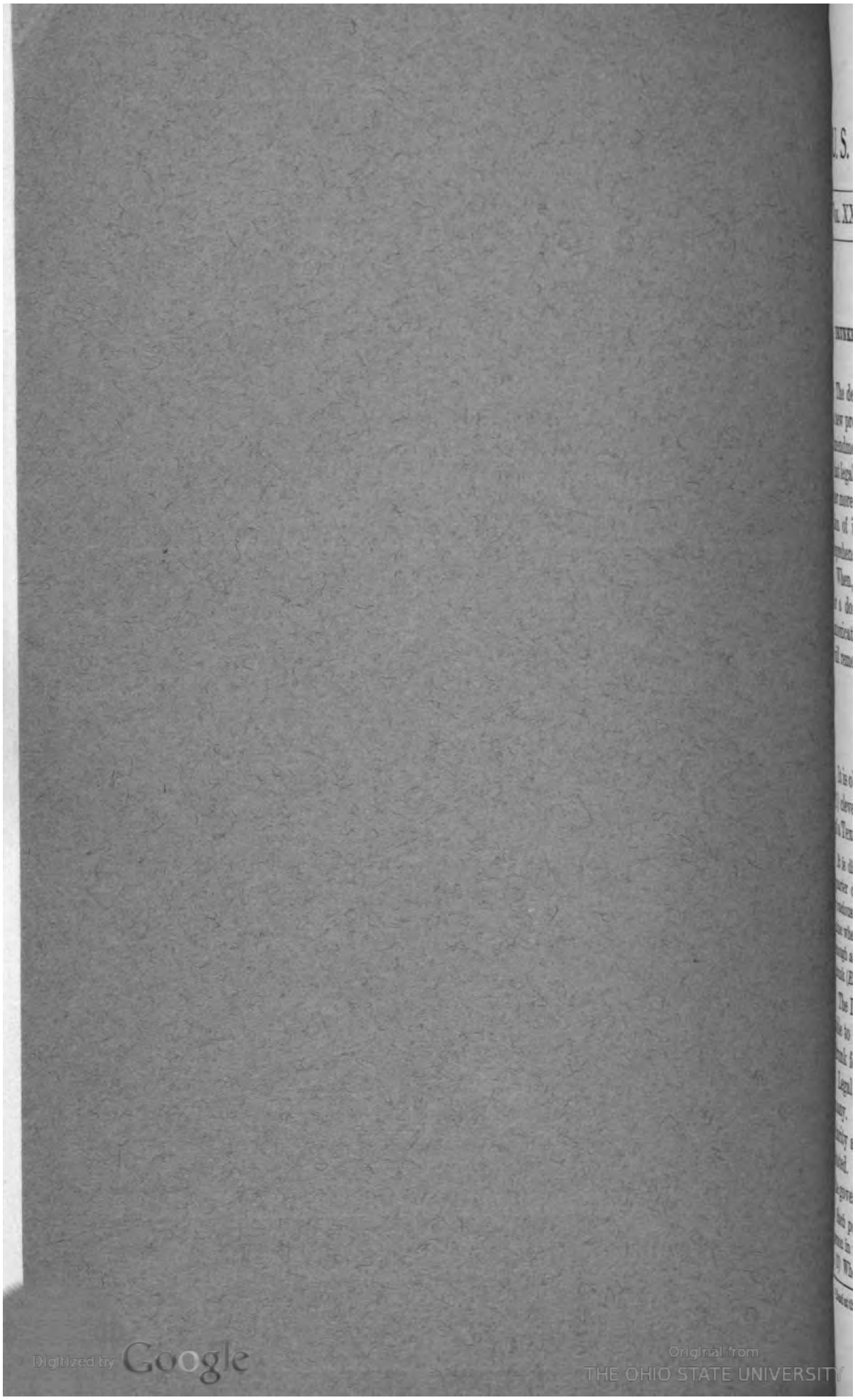
**TO KEEP AS MANY MEN AT AS MANY GUNS
AS MANY DAYS AS POSSIBLE**

**Issued Quarterly by the Bureau of Medicine and Surgery
Washington, D. C.**



VOL. XXXIV APRIL 1936 NO. 2

UNITED STATES NAVAL MEDICAL BULLETIN

PUBLISHED QUARTERLY FOR THE INFORMATION OF THE MEDICAL DEPARTMENT OF THE NAVY

Issued by

THE BUREAU OF MEDICINE AND SURGERY
NAVY DEPARTMENT

DIVISION OF PUBLICATIONS
COMMANDER LOUIS H. RODDIS
MEDICAL CORPS, U. S. NAVY, IN CHARGE

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NAVY DEPARTMENT,
Washington, March 20, 1907.

This UNITED STATES NAVAL MEDICAL BULLETIN is published by direction of the Department for the timely information of the Medical and Hospital Corps of the Navy.

TRUMAN H. NEWBERRY,
Acting Secretary.

Owing to the exhaustion of certain numbers of the BULLETIN and the frequent demands from libraries, etc., for copies to complete their files, the return of any of the following issues will be greatly appreciated

Volume IX, no. 1, January 1915
Volume X, no. 2, April 1916
Volume XI, no. 3, July 1917
Volume XII, no. 1, January 1918
Volume XII, no. 3, July 1918

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PREFACE

The UNITED STATES NAVAL MEDICAL BULLETIN was first issued in April 1907 as a means of supplying medical officers of the United States Navy with information regarding the advances which are continually being made in the medical sciences, and as a medium for the publication of accounts of special researches, observations, or experiences of individual medical officers.

It is the aim of the Bureau of Medicine and Surgery to furnish in each issue special articles relating to naval medicine, descriptions of suggested devices, clinical notes on interesting cases, editorial comment on current medical literature of special professional interest to the naval medical officer, and reports from various sources, notes, and comments on topics of medical interest.

The Bureau extends an invitation to all medical and dental officers to prepare and forward, with a view to publication, contributions on subjects of interest to naval medical officers.

In order that each service contributor may receive due credit for his efforts in preparing matter for the BULLETIN of distinct originality and special merit, the Surgeon General of the Navy will send a letter of commendation to authors of papers of outstanding merit.

The Bureau does not necessarily undertake to endorse all views or opinions which may be expressed in the pages of this publication.

P. S. ROSSITER,
Surgeon General, United States Navy.

NOTICE TO SERVICE CONTRIBUTORS

Contributions to the BULLETIN should be typewritten, *double spaced*, on plain paper, and should have wide margins. Fasteners which will not tear the paper when removed should be used. Nothing should be written in the manuscript which is not intended for publication. For example, addresses, dates, etc., not a part of the article, require deletion by the editor. The BULLETIN endeavors to follow a uniform style in heading and captions, and the editor can be spared much time and trouble, and unnecessary changes in manuscript can be obviated, if authors will follow in these particulars the practice of recent issues.

The greatest accuracy and fullness should be employed in all citations, as it has sometimes been necessary to decline articles otherwise desirable because it was impossible for the editor to understand or verify references, quotations, etc. The frequency of gross errors in orthography in many contributions is conclusive evidence that authors often fail to read over their manuscripts after they have been typewritten.

Contributions must be received at least 3 months prior to the date of the issue for which they are intended.

The editor is not responsible for the safe return of manuscripts and pictures. All materials supplied for illustrations, if not original, should be accompanied by reference to the source and a statement as to whether or not reproduction has been authorized.

The BULLETIN intends to print *only original articles, translations, in whole or in part, reviews, and reports and notices of Government or departmental activities, official announcements, etc. All original contributions are accepted on the assumption that they have not appeared previously and are not to be reprinted elsewhere without an understanding to that effect.*

U. S. NAVAL MEDICAL BULLETIN

VOL. XXXIV APRIL 1936 No. 2

SPECIAL ARTICLES

DRUNKENNESS, NAVAL MEDICO-LEGAL ASPECTS OF THE DIAGNOSIS [1]

W. W. HALL, Lieutenant Commander, Medical Corps, United States Navy

The determination of alcoholic intoxication or drunkenness, is not a new problem, nor did it become less important with the eighteenth amendment, the Volstead Act and high-speed transportation. Now that legal liquor is with us again the problem, while perhaps no greater nor more frequently presented, is in a sense clarified. It is the condition of intoxication not the act of drinking which is illegal and reprehensible.

When, then, is a person intoxicated: When he has taken one drink, or a dozen? Many descriptions and definitions of the state of intoxication have been given. One venerable masterpiece that many will remember is:

> He is *not* drunk who, from the floor,
> Can rise again and drink once more.
> But he *is* drunk who prostrate lies,
> And who can neither drink nor rise.

It is obvious that at some point the line should be drawn. "Judge" (1) cleverly emphasizes this point by quoting the humorous decision of a Texas court:

> It is difficult to draw the line on a drunk: There are various stages, such as quarter drunk, half drunk, and dead drunk. There are the stages of being vivacious, foxy, tipsy, and on a high lonesome. It is about as difficult to determine when a young lady gets to be an old maid as to tell when a man has taken enough alcoholic stimulant to pass the line between jolly sober and gentlemanly drunk (*Ex parte Townsend* (Tex. 1911), 144 S. W. 633).

The Duke of Connaught once said of a certain colonel, "He is just able to walk straight. That is sober enough for a civilian but very drunk for a soldier." So much for the difference in points of view.

Legal decisions in civil courts bearing upon this point have been many. What is most important to a naval medical officer is the clarity and precision with which the legal opinions in naval law are stated. I quote the following, beginning with the eighth article for the government of the Navy, which states in part:

> Such punishment as a court martial may adjudge may be inflicted on any person in the Navy.
>
> (1) Who is guilty of drunkenness.

[1] Read at the Fleet Medical Conference on board the U. S. S. *Relief*, Pearl Harbor, T. H., May 28, 1935.

Paragraph 743 Manual of the Medical Department, United States Navy, 1927:

> Examination of persons suspected of being under the influence of an intoxicant—Medical officers will make a sufficiently comprehensive examination to determine: First, Is the condition and behavior of the individual normal or abnormal? Second, Is he suffering mentally or physically from the results of sickness or injury? Third, Is his condition or behavior due wholly or in part to the use of a drug or alcohol? Fourth, Is the intoxication "sufficient sensibly to impair the rational and full exercise of the mental and physical faculties?" (Definition of legal drunkenness, sec. 229, Naval Courts and Boards.) Fifth, Is there evidence that the individual has used alcoholic liquor for drinking purposes? Sixth, Is he fit to perform duty?

As the medical officer's opinions will probably be accepted as those of an expert, he should be prepared to cite facts to support them. In view of the probability that he will be called as a witness before a court martial it is important that at the time he takes notes recording the time of examination and the detailed history of the case.

Attention is invited to Court Martial Order No. 92, 1905, which states:

> One who is under the influence of liquor in any degree, however slight it may be, is unfit to be intrusted with the important duties incident to the Naval Service.

Medical officers are reminded that under the stimulus of an official examination an individual may assume such self-control that it may be difficult to diagnose his condition. Sufficient time, therefore, should be given to the examination. Disturbances of motor coordination, speech, gait, balance, etc., as well as abnormalities of ideas, have special significance.

Court Martial Order No. 1, 1922, 14 is quoted in full because of its value. It has been referred to again and again in other court martial orders and decisions. *Under the influence of intoxicating liquor.* What constitutes?

In reviewing the summary court martial in the case of ———, it is noted that the accused was found guilty of but one specification, namely, the first, which reads as follows:

> In that ——— ———, hospital apprentice first class, United States Navy, attached to and on duty at the United States Naval Hospital, Chelsea, Mass., was, at or about 9 p. m. October 15, 1921, while on duty in ward 1–1 of said hospital, under the influence of intoxicating liquor, and thereby incapacitated for the proper performance of duty.

The convening authority in commenting on the court's findings on this specification stated, in effect, that in his opinion, the finding was in error for the reason that there was no direct testimony to the effect that the accused was "incapacitated for the proper performance of duty." Notwithstanding the expression of the above opinion, however, the convening authority approved the finding of the court in this specification.

It appears from the record that Lieutenant ———, Medical Corps, United States Navy, was the principal witness for the prosecution in connection with the specification in question. The gist of Lieutenant ———'s testimony in regard to the point at issue is contained in the following questions and answers taken from the record:

4. Q. Was he under the influence of liquor about 9 p. m. October 15, 1921, while in ward 1–1?

A. My opinion is that any man that takes a drink is under the influence of liquor, so I will say "Yes", but I would not say he was drunk.

5. Q. From your examination of the man and your knowledge of the effects of liquor would you swear that this man was under the influence at that time, when you first saw him over in ward 1–1?

A. Yes; according to my definition any man who has been drinking is under the influence of liquor.

6. Q. Could you swear that his breath smelled of some intoxicant?

A. Yes.

7. Q. Did he show any other evidence of being under the influence of liquor?

A. No, sir; he was not noisy or insolent.

It will be noted from the above that Lieutenant ———'s testimony in support of the specification is based upon a distinction which he made in his mind between the meaning of the phrase "under the influence of intoxicating liquor" and the word "drunk." It is clear from a reading of the above-quoted excerpt from his testimony that this witness was under the impression that proof of the specification required only the establishment of the fact that the accused had partaken of intoxicating liquor, that it was immaterial how slight an an amount of such liquor the accused had taken, and that it was also immaterial whether or not the drink had any visible effect upon him. The court apparently accepted this witness' version of this matter and found the specification proved on the strength of his testimony.

It may be true that as a matter of medical theory even the slightest indulgence in intoxicating liquor, or anything else for that matter, leaves some effect upon the human system, medically considered. However, in the opinion of the Department, such theory has no place in naval court-martial procedure. To hold otherwise would be equivalent to holding that in order to find persons in the naval service guilty of drunkenness at any time or place it would only be necessary to show that the person so charged had partaken of even the slightest amount of intoxicating liquor and his guilt would be established irrespective of the fact that such act was unaccompanied by any sensible impairment of the faculties. Such a proposition readily reduces itself to absurdity. However, it may be pointed out that one of the sample specifications contained on page 98 of Naval Courts and Boards in support of the charge of "Drunkenness" alleges merely that the accused was "*under the influence of intoxicating liquor.*" This in itself shows that for the purposes of naval court-

martial trials, the terms "*drunkenness*" *and* "*under the influence of intoxicating liquor*" are synonymous.

The real test in cases of this kind is set forth in Court-Martial Order 18, 1917, in which it is stated that—

> The articles for the government of the Navy do not require any particular degree of drunkenness, and it is considered that such charge is supported by showing *incapacity for the full performance of duty* as a result of indulgence in intoxicating liquor.

The same court-martial order further defines drunkenness as "*any intoxication which is sufficient to sensibly impair the rational and full exercise of the mental and physical faculties.*" It follows from the above that the matter at issue in cases of drunkenness is whether or not the faculties have been sensibly impaired by indulgence in intoxicating liquor and, unless such impairment can be shown, it matters not whether the accused has partaken sparingly or freely.

In connection with the above, however, attention is invited to paragraph 118 (2) Navy Regulations, 1920, which provides:

> The use or introduction for drinking purposes of alcoholic liquors on board any naval vessel, or within any navy yard or station, is strictly prohibited, and commanding officers will be held directly responsible for the enforcement of this order.

Accordingly it follows that in cases where, as in the present case, there appears to have been no impairment of the faculties, however slight, as a result of indulgence in intoxicating liquors, there still remains an offense in violation of Navy Regulations, provided it can be shown that such indulgence took place on board a naval vessel or within any navy yard or station. In order for a court martial to take cognizance of this offense, however, it is of course necessary that the specification referred to the court set forth facts which constitute a violation of this regulation.

In view of the above, the finding on the first specification and, inasmuch as that is the only specification found proved by the court, the sentence in the case of the above-named man was set aside (file 26287–8384, J. A. G. Jan. 10, 1922). Signed: Edwin Denby, Secretary of the Navy.

Naval Courts and Boards, section 229, reads as follows:

> *Drunkenness.*—This is provided for in 8th A. G. N., paragraph 1.
>
> *Charge.*—Drunkenness.
>
> *Elements.*—Any intoxication which is sufficient sensibly to impair the rational and full exercise of the mental and physical faculties is drunkenness (Court-Martial Order 1, 1922, 14). If the accused was incapacitated for the proper performance of any duty for the performance of which a person of the rank or rate of the accused could properly be called, conviction is proper.
>
> If the drunkenness occur on duty this must be alleged as an aggravation, as a greater limit of punishment is provided in such a case.

Court-Martial Order 92, 1905, 3: Degree necessary to constitute "drunkenness." "One who is under the influence of liquor in any degree, however slight it may be, is unfit to be trusted with the important duties incident to the Naval Service."

Court-Martial Order 5, 1913, 3: To support a charge of "drunkenness" it is not necessary for the evidence to show that the degree of intoxication was so great as to occasion a profound stupor on the part of the accused; the universally established practice of the Naval Service, on the contrary, is such as to warrant a finding of guilty of the charge of "drunkenness" when it is proved by reliable witnesses that the accused was *under the influence of intoxicating liquor;* indeed, no other finding would be justifiable.

Court-Martial Order 5, 1915, 2: Manifestly there are different degrees of intoxication. However officers of the Naval Service should not be guilty of *overindulgence* which will in *any way incapacitate them* for *any duties* which *may be required of them.* The fact that an officer apparently performs the duties assigned to him at a particular time does not of itself indicate that he is capable of performing any duties which might have been assigned to him. The degree of intoxication goes to the gravity of the offense, but does not relieve an officer of the consequences of his condition if he has been guilty of such overindulgence as will incapacitate him for the full performance of his duties.

Court-Martial Order 28, 1915, 2: Drunkenness, evidence of; a medical officer who examined the accused testified:

He was further examined by me at the time, and it was soon evident in my opinion that he was suffering from the excessive use of alcoholic liquor. This was evidenced by his general manner and deportment, his unsteadiness, sluggish reaction of both his pupils, his tremulous tongue, pulse of 112, odor of alcohol on his breath, and his own acknowledgment that he had been out drinking the night before.

Q. Doctor, would you say that you considered this accused under the influence of liquor?

A. Yes, and I didn't consider him fit to perform his duty that day, and so reported.

Court-Martial Order 17, 1917, 4: Degree of intoxication ——— in the Army Digest, 1912 (540 X11 A9a), drunkenness within the meaning of the Articles of War is defined as "any intoxication which is sufficient sensibly to impair the rational and full exercise of the mental and physical faculties." Nor do the articles for the government of the Navy require any particular degree of drunkenness, and it is considered that such charge is supported by showing incapacity for the full performance of duty as a result of indulgence in intoxicating liquor.

Court-Martial Order 1, 1929, 30: Drunkenness (2) Incapacity for the proper performance of duty—Court to determine.

NOTE.—This clearly brings out the medical officer's duty as examining and testifying officer to determine only whether or not the man is exhibiting "any intoxication which is sufficient to sensibly impair the rational and full exercise of the mental and physical faculties."

The judge advocate, on direct examination of a witness, asked the following question: "Was the man in condition to perform the duties of his rate at the time"? This is an improper question. It involves two conclusions. One, the nature and character of the duties of his rate. The other the conclusion as to whether a man in his condition is competent to perform those duties. Both questions are to be determined by the court, from evidence adduced before it. With reference to the duties of the rate, the court takes judicial notice of those duties. It is proper to inform the court by the evidence in whatever detail may be necessary as to the physical condition of the accused. From that description, and from its knowledge of the duties of the rate, the court must determine whether or not the ability of the accused to fully and completely perform all the obligations of his position was impaired by the use of alcoholic liquor. *Incapacity for proper performance of duty is not a fact that can be proved by direct testimony as by the conclusion of a medical officer*, or by any other person in authority, but it is a conclusion that the court itself must draw from the evidence before it by taking into account all the testimony as to the condition of the accused at the time in question and the character of the duties to be performed by the accused. In other words, if evidence is offered to satisfactorily prove that the accused was under the influence of intoxicating liquor, the court must then decide as a conclusion of fact whether or not the accused was incapacitated for the proper performance of his duties.

Other court-martial orders covering these or similar points are: Court-Martial Order 22, 1884, 3; Court-Martial Order 3, 1929, 23; Court-Martial Order 7, 1932, 7; and Court-Martial Order 11, 1929, 8.

As indicated in various places where the testifying individual is referred to as a "reliable witness" (C. M. O., 5, 1913, 3), "a witness" and again "any other person in authority" (C. M. O., 1, 1929, 30), the opinion of a medical officer on the degree of intoxication is not by any means indispensable. Any reliable adult should be able to form and express an opinion on the matter. Such witnesses would appear to have been acceptable to the courts. However when called upon to examine a man for intoxication the medical officer's greater knowledge infers greater responsibility in diagnosis and differential diagnosis. Most often the signs of drunkenness are so clear that he who runs may read, but again the question may be involved. "As the medical officers opinions will probably be accepted as those of an expert he should be prepared to cite facts to support them" (par. 743, Manual for the Medical Department).

Thus as has been stated above, the medical officers problem from a medico-legal standpoint is to determine when the intoxication is sufficient sensibly to impair the rational and full exercise of the mental and physical faculties. The medical officer also has a problem to solve in differential diagnosis. He must differentiate many patholog-

ical conditions that may present some or many of the same symptoms commonly considered those of drunkenness such as the flushed face, the staggering gait, mental confusion, or boisterous actions and speech. Bogen (2) mentions such conditions as vasomotor instability with flushed face or tachycardia, stammering, constitutional psychopathic states, as well as acute febrile disturbances, thyrotoxicosis, hypoglycemia, insulin shock and any disease, injury, or accident affecting the central nervous system. Of course, says Bogen:

> The differentiation between all of these conditions and acute alcoholic intoxication may readily be made in a majority of instances by the presence or absence of other signs or symptoms essential for diagnosis, but this is not always the case and it must not be forgotten that a man suffering from one of these other conditions may and frequently does suffer also from the effects of drinking alcoholic liquors.

The determination of the alcoholic content of the body fluids offers a most valuable and definite numerical measurement, as an aid in diagnosis and differential diagnosis of intoxication. I wish to emphasize the word "aid", for this or any diagnosis should be based upon the ensemble of symptoms plus laboratory examinations where needed, and not upon a single test no matter how helpful or valuable the information that that test gives may be.

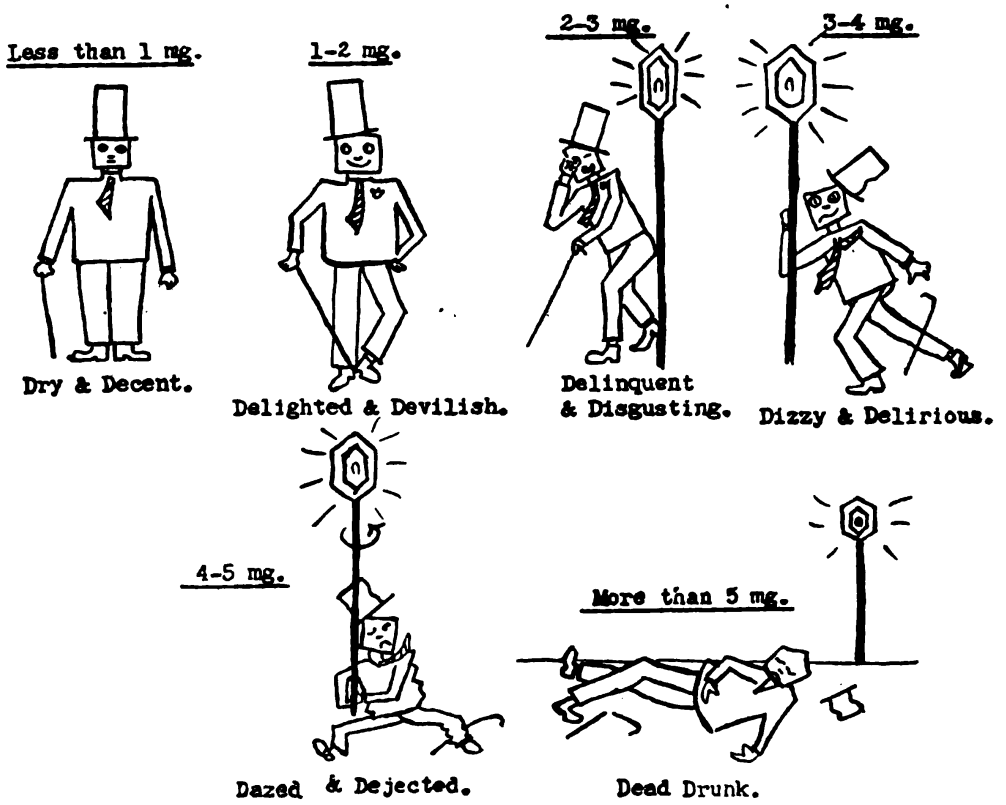
It is an established fact that the intoxicating effect of alcohol, like many other drugs, and substances, is proportional to the amount present in the tissues. In this case the tissue most important in the question of intoxication is the brain. It is obviously impossible to analyze brain tissue routinely and as the concentration in the blood bears a direct relation to that in the brain or vice versa, the examination of the blood offers an accurate index of the brain's alcoholic content. Imbibed alcohol passes through the gastro-intestinal mucosa and becomes rapidly and uniformly diffused through the body. The highest blood concentration is usually reached about 1 hour after a given dose. Urine excreted from blood of a given alcoholic concentration equals that concentration or is slightly higher. The breath also, as everyone can testify, is heavily laden with alcoholic vapor, directly proportional to that of the blood.

Attempts to correlate the level of alcohol in the tissues and body fluids with the degree of intoxication, as noted by Webster (3), have been made by Nicloux (4) in 1900, by Ford (5) in 1906, by Schwarz (6) in 1926, and others. They contributed valuable information and their results compare favorably with those of Bogen (2).

In 1928 Bogen reported a quantitative study of acute alcoholic intoxication. He adopted a solution he designated as Anstie's reagent, essentially the same as that used in the original Nicloux (4) method which has been often modified and more widely used than any other for the estimation of alcohol.

He determined the concentration of alcohol in the urine, blood, spinal fluid, and breath of alcoholics and correlated the results with the clinical degree of intoxication. His work gave the method standing and established its value. His methods were the basis of the work reported by Johnson (7) and were followed in the series herewith reported.

Sheets covering the method somewhat modified by us have been prepared and are reproduced below. The first sheet contains information desired by the clinician, including the method in brief, with a short discussion and figures which graphically represent the correlation of findings in the urine with the clinical condition. These figures,



Alcohol in 1 cubic centimeter of urine

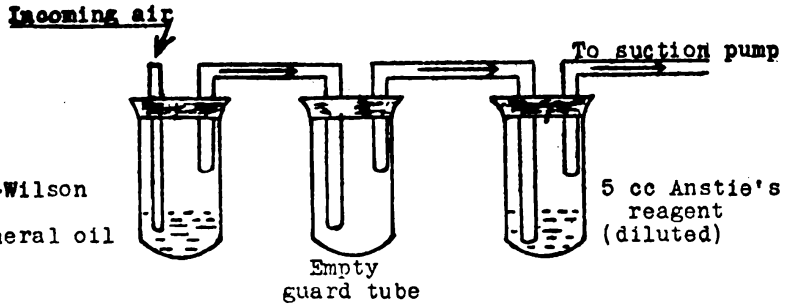
which have not been previously published, were drawn by Dr. Esther Bogen Tietz for use in an oral presentation of the subject and are reproduced by permission of Dr. Emil Bogen. The second sheet gives information in full as to the reagents, apparatus, and method; i. e., information needed by a technician in order to perform the test.

First Sheet

The chemical determination of acute alcoholic intoxication by examination of blood, urine, or spinal fluid.—Method in brief: Draw air through 1 cubic centimeter of specimen to be examined and then through 5 cubic centimeters of Anstie's reagent (dichromate-sulphuric

acid) while both are immersed in a boiling water bath. Measure alcohol present by comparing color of reagent with previously prepared standards containing known amounts of alcohol. Color varies from reddish yellow to greenish blue.

The alcoholic content of urine, blood, spinal fluid, gastric contents, brain tissue, or breath was determined by special chemical methods and correlated with the clinical findings in more than 300 patients who were suspected of drunkenness. No person with less than 1 milligram of alcohol in 1 cubic centimeter of urine gave clinical evidence sufficient to justify a diagnosis of acute alcoholic intoxication. Above this concentration, the frequency of the clinical symptoms of drunkenness increased with the amount of alcohol found. Because of the difficulty in making the diagnosis of acute alcoholic intoxication from the clinical evidence alone, the examination for drunkenness should in every case include a quantitative determination of the concentration of alcohol in the urine, breath, or body fluids (Bogen).



Reaction:-

$$K_2Cr_2O_7 + 4H_2SO_4 + 3C_2H_5OH = 3C_2H_4O + K_2SO_4 + Cr_2(SO_4)_3 + 7H_2O$$

The green color in the completed reaction is Chromic Sulphate.

The values in blood and their correlation with the degree of intoxication correspond closely with those in urine as depicted in the figures above. Due to the fact that the blood concentration always represents the degree of alcoholic saturation at the moment and not at some time past (as is possible in urine which may have been held in the bladder), it has been selected by us as the most desirable test fluid (W. W. H.).

SECOND SHEET

Blood is collected by venipuncture and oxalated as for other blood chemical procedures (½ milligram oxalate per mil of blood). When cleansing arm with alcohol or tincture of iodine prior to puncture allow to dry completely to avoid fouling of needle.

Three tubes are set up in a row and connected for aeration. In the tube farthest from suction pump, place 1 cubic centimeter of blood, urine, or other fluid to be examined, 1 cubic centimeter Scott-Wilson reagent (for fixation of acetone), 3 to 5 cubic centimeters of distilled water, and 5 to 10 drops of liquid petrolatum (optional) to

prevent foaming. The center tube is empty and is placed in series to guard against foaming over directly into reagent tube. The tube nearest the suction pump contains 5 cubic centimeters of Anstie's reagent. Place connected tubes in a boiling water bath and aerate for 10 minutes. Compare reagent tube against known standards.

The method depends upon the fact that, when a solution of potassium bichromate in sulphuric acid is treated with alcohol, the alcohol is oxidized, largely to acetaldehyde, while the bichromate is reduced with the formation of chromic sulphate according to the above equation.

Scott-Wilson reagent.—Dissolve 1 gram of mercuric cyanide in 60 cubic centimeters of distilled water, using a heavy walled glass jar. Add a solution of 18 grams of sodium hydroxide in 60 cubic centimeters of water, stir vigorously with a heavy glass rod, add 0.290 gram of silver nitrate dissolved in 40 cubic centimeters of water. The mixture should be clear and ready for use at once. If turbid, allow to stand 2 or 3 days, decant the clear supernatant fluid. This keeps several months.

Anstie's reagent.—Dissolve with the aid of heat in water bath 0.666 gram of potassium dichromate (CP) in 100 cubic centimeters of concentrated sulphuric acid (CP). This will be labeled "Concentrated Anstie's" and is used in preparing the standards. Dilute with *equal* parts distilled water for use in the test.

Preparation of standards.—Make a 1-percent solution of alcohol (by weight) by pipetting 1.260 cubic centimeters of absolute alcohol at 15° C. into distilled water. Make up volume to 100 cubic centimeters in volumetric flask. Prepare a series of 11 tubes (12 by 150 mm), labeled 0.0, 0.5, 1.0, 1.5, 2.0, 2.5, 3.0, 3.5, 4.0, 4.5, 5.0 milligrams per cubic centimeter, respectively. Pipette the 1-percent alcohol solution into each tube to represent the above figures. Make up volume in each tube to 2.5 cubic centimeters with distilled water. Add to each tube 2.5 cubic centimeters of concentrated Anstie's reagent (⅔ percent potassium dichromate in sulphuric acid). Seal tubes by heating and drawing from top.

Example.—Tube marked "0.5 milligram per cubic centimeter" contains:

	Cubic centimeters
1 percent alcohol solution	0.05
Distilled water	2.45
Concentrated Anstie's reagent	2.50
Total	5.00

These standards are reliable for approximately 3 months, after which time the color in the higher concentrations of alcohol breaks down and begins to fade.

It has been our routine in cases which seem to have 3 milligrams alcohol per cubic centimeter of blood or higher to repeat the test using only 0.5 cubic centimeter of blood and to multiply by 2 for the final reading. This has been considered advisable as matching of color, easy in the lower, is more difficult in the higher standards. One is also approaching the limit of capacity of the reagent at the 4-milligram concentration.

A small color change approximating the 0.5 milligram standard is, in our experience, produced by all normal bloods in the above method. This change is produced by some volatile organic constituent of the blood, probably, at least partially alcohol, for there is known to be alcohol present in minute quantities in the tissues normally (3). Therefore a change corresponding to the 0.5 standard should not be taken to mean that the individual being examined has imbibed alcohol.

Imbibing of ginger ale without alcohol, or the consuming of other carbonated beverages, contrary to opinions sometime expressed, does not cause the body fluids to react as does alcohol in this method.

Nonvolatile organic material, glycerin, phenol, cresol, etc., added directly to the reagent, produces the same color change as alcohol. Ether as well as other volatile organic substances not encountered in the body fluids also react similarly. Therefore, care and cleanliness with instruments and glassware as well as in performance of the test are essential to the integrity and reliability of the procedure.

Because of the reaction, with the reagent, of substances in cork and rubber stoppers, tubed standards, which are to be kept for some time and which may be inverted, should not be so closed but should be sealed as directed above by heating and drawing out the glass.

In the following table which compares clinical observations as made by many different examining officers with the chemical determination of alcohol in 1 mil of blood the same symptoms were reported upon as those selected by Bogen. This was done in order to correlate findings with those obtained by him.

42258—36——2

Clinician's notes

	Milligrams of alcohol per mil of blood				
	0 to 1	1.5 to 2	2.5 to 3	3.5 to 4	4.5 or more
Number examined	79	69	53	21	0
Clinical diagnosis of acute alcoholic intoxication	16	52	46	21	0
Admitted drinking	23	37	20	7	0
Complicating injuries	8	14	5	2	0
Flushed face	10	27	29	8	0
Dilated pupils	5	25	25	10	0
Alcoholic odor	48	54	45	20	0
Gait:					
Fair	6	13	11	3	0
Staggering	8	21	17	5	0
Unable to stand	10	60	8	6	0
Sway when standing	2	3	3	1	0
Coordination normal	67	30	0	0	0
Coordination fair	3	9	2	0	0
Speech:					
Fair	5	14	5	0	0
Slurred	5	18	17	4	0
Confused	1	10	7	8	0
Unable to speak	0	2	4	7	0
Comatose	(4X)	1 (4X)	4 (4X)	7 (1X)	----------

NOTE.—X denotes those in which a complicating head or serious internal injury was sufficient to prevent locomotion, interfere with the examination or account for the symptoms.

It has been our experience that the values in urine run slightly higher in the upper brackets than those in the blood for the same degree of intoxication. We encountered no cases with readings of 4.5 milligrams or higher. However, a fatal case of acute alcoholic intoxication with a reading of but 4 to 4.5 milligrams will bear reporting in slight detail. The level probably would have been higher if taken at the time of death, as there is apparently a slow decomposition or oxidation in the blood and tissues after death. In animal experiments it has been found that death regularly ensues when the concentration of alcohol in the blood exceeds 6 milligrams per mil (8).

Two men, J. S. and M. D., had purchased several pints of so-called gin and gone to a hotel room to stage a drinking bout. They drank drink for drink and rather rapidly consumed each an equal amount of the liquor, according to M. D., when the question of going to a dance came up. After an argument, J. S. lay down on the bed and M. D. fared forth to the dance. When he returned to the hotel room about midnight reasonably sober and in search of another drink he found J. S. dead on the floor where he had apparently rolled from the bed. An autopsy 12 hours later showed no important pathological condition other than those incident to a blood alcohol of between 4 and 4½ milligrams.

In the case of M. D. the alcohol had been oxidized as it was absorbed. He had "worked it off." With J. S. the alcoholic concentration of the blood had mounted to a fatally toxic level as he lay in alcoholic coma "sleeping it off."

The blood concentration is determined by the balance of the rate of absorption on one hand and the rates of oxidation, exhalation, and

excretion on the other. The consumption of low-alcoholic-content liquors results in much less rapid absorption than that of high content beverages. The presence of much food or fluid in the stomach with which the liquor is diluted results in the same slow rate of absorption. The lesser intoxicating effect of imbibing on a full stomach than on an empty stomach is well known. The great variation in apparent susceptibility of various normal individuals to alcoholic intoxication must be explained on the basis of the difference in absorption rate or on the difference in excretion and oxidation rates, for variation in susceptibility, as judged by comparison of blood concentration and the clinical condition, is slight.

Individuals whose blood shows less than 1 milligram of alcohol per cubic centimeter show no clinical evidence of intoxication; between 1 and 2, the exhilarating effect of intoxication is quite apparent. Here the effects of alcohol as a social lubricant are seen. Memory and judgment are slightly, if any, impaired, inhibitions are removed, the tongue is loosened, the limbs move freely, a pleasant glow pervades. "Delighted and devilish" describes the condition, the golden stage of intoxication. In individuals in this stage and in the lower half of the next, with a blood concentration between 1½ and 2½ milligrams of alcohol, the factor of tolerance operates if it operates anywhere. Some persons may appear rather drunk with varying minor degrees of incoordination and impairment of judgment, while others show only slight incoordination or it is only brought out in more difficult movements than walking and pointing. The condition of intoxication progresses through the "delinquent and disgusting" stage, corresponding to concentrations of less than 3 milligrams, to the "dizzy and delirious" stage with over 3 and less than 4 milligrams. At 4, a considerable percentage of our cases were comatose (30 percent); all were unable to stand. This variation from Bogen's observation is probably due, as noted above, to the slightly higher level in the urine than in the blood, as Bogen's traditional "dead drunk" is an individual who has in the urine 5 or more milligrams of alcohol per cubic centimeter.

Johnson (7), reporting on 200 cases in which the blood alcohol was used as an aid in diagnosis, considered the 4-milligram level as that at which coma might regularly be expected. On this basis he divided the cases into four groups, considering those showing 1 milligram of alcohol per cubic centimeter of blood as 25 percent intoxicated, those with 2 milligrams as 50 percent, those with 3 milligrams as 75 percent, and those with 4 milligrams as 100 percent intoxicated. This division is quite satisfactory in dividing cases into the steps toward coma, but not so as to the degree of intoxication. For example, a man with 1 or 1.5 milligrams of alcohol may be (and the vast majority are) fully able to perform all acts requiring fine muscular coordination

satisfactorily, to articulate and reason as clearly as one with no alcohol whatever, and he therefore is not at all intoxicated or under the influence of intoxicating liquor within the meaning of the law. On the other hand, a man showing 2.5 milligrams may present (and the majority do) the picture of a man who has crossed the line and in whom one finds varying degrees of incoordination, poor articulation of test phrases, and unsound judgment, with other signs which compel the examining officer to judge him intoxicated. That is, his faculties are sensibly impaired. In other words, judged from the medico-legal standpoint, a man either is or is not intoxicated, either is or is not able to properly perform his duty; either, therefore, is 0 percent intoxicated or 100 percent so.

SUMMARY

1. Naval legal opinions and decisions with respect to the determination of drunkenness are cited.

2. Drunkenness is "any intoxication which is sufficient to sensibly impair the rational and full exercise of the mental and physical faculties."

3. The Bogen method for the chemical determination of alcoholic content of blood, urine, and other body fluids is given. This method is simple and may be performed with a minimum of reagents and equipment.

4. The determination of the alcoholic content of blood or other body fluid should not be considered a diagnosis, only an aid in the differential diagnosis of alcoholic intoxication from other intoxications, diseases, and conditions, and a valuable and rather accurate aid in measuring the degree.

5. The susceptibility of the brain to alcohol varies little between one normal individual and another. The apparent variation in susceptibility in different individuals is thought to be governed more by the rate of absorption on one hand, and oxidation, exhalation, and excretion on the other, than to any inherent resistance of the cell protoplasm to the toxic effects of alcohol.

REFERENCES

(1) Judge: June 1931, p. 12. Judge Magazine Co., New York.

(2) Bogen, Emil: A Quantitative Study of Acute Alcoholic Intoxication. Am. Jour. Med. Sci., vol. 176, no. 2; Aug. 1928, p. 153.

(3) Webster: Legal Medicine and Toxicology, p. 733. W. B. Saunders Co., 1930.

(4) Nicloux: Thèse de Paris. 1900. As quoted by Webster (3) (ibid).

(5) Ford: Jour. Physiol., 1906, vol. 34, p. 430. As quoted by Webster (3) (ibid).

(6) Schwarz: Schweiz. Med. Wchnschr., 1926, vol. 56, p. 923. As quoted by Webster (3) (ibid).

(7) Johnson, F. S.: Report of Two Hundred Examinations for Acute Alcoholism. U. S. Naval Med. Bul., vol. 28, p. 85; June 1930.

(8) Peterson, Haines, and Webster: Legal Medicine and Toxicology, vol. II, 2d ed., p. 615. W. B. Saunders Co., 1923.

DENTINE DESENSITIZATION

By LEROY L. HARTMAN, Lieutenant Commander, Dental Corps, United States Naval Reserve, Professor of Dentistry at Columbia University

The painful reaction of dentine to operating procedures lead me to believe that it was probably due to the presence of lipoids in this tissue. Working on this assumption, a solution was prepared containing the lipoid solvents, alcohol and ether, and thymol, a substance soluble in lipoids.

The formula which has been designated as "Hartman's Solution" has been found effective in relieving pain during operations on dentine.

The formula by weight is—

	Part
Thymol	1¼
Ethyl alcohol (95 percent)	1
Sulphuric ether	2

Keep tightly corked in brown glass bottle. One-half ounce sufficient for 200 applications.

Use cork or tin lined stoppers only.

Directions for use.—This is a topical application. Pack the cavity with dry cotton and make application on a second pellet of cotton moistened in the liquid and held in contact with the cotton in the cavity for the required time. One minute for children, 1½ minutes for adults. Remove both pellets, use warm air gently to leave a film of thymol on the surface of the cavity. Wait 2 minutes before operating. If applied over caries, a second application may be necessary after the caries has been removed.

Use rubber dam for most efficient results.

If cotton rolls are used, varnish gums surrounding tooth and change cotton rolls immediately after application of the fluid.

TREATMENT OF ACUTE MECHANICAL INTESTINAL OBSTRUCTION [2]

By M. D. WILLCUTTS, Commander, Medical Corps, United States Navy

A century ago the master physicians regarded intestinal obstruction as the gravest disorder of the abdomen. Today this same condition continues to be a major disaster with rising incidence and alarming mortality as surgery of the abdomen extends into thousands of lapa-

[2] Read at the Staff Conference, U. S. Naval Hospital, Washington, D. C. on Oct. 25, 1935.

rotomies performed daily throughout the country. Holden (1) claims every operation in the pelvis, on the appendix, gall bladder, stomach, and kidney to be a potential source for intestinal obstruction.

No field of abdominal surgery challenges our attention more, for unrelieved mechanical intestinal obstruction is hopelessly fatal. The approach of death is swift and cruel in intense pain, vomiting, and thirst.

Except in the functional paralytic or the spastic types, the lesion is surgical, urgent, and absolute. The early recognition of the functional or of the mechanical factors producing the obstruction give life-saving opportunities for the treatment and the management of this catastrophe. The term "ileus" is often confusing and far from definite. The text book expressions of "dynamic ileus" and "adynamic ileus" are unsatisfactory. Of Greek origin, ileus means a twist—dynamic ileus, a twist with force, is defined as spastic obstruction. Adynamic ileus, a twist without force, is defined as paralytic obstruction.

Wangensteen (2) makes a much clearer and more useful classification. He names three main groups:

I. Mechanical obstruction, subdivided into (*a*) simple obstruction and (*b*) strangulation obstruction due to strictures; adhesions and bands of congenital, inflammatory, traumatic, and neoplastic origin; hernia; volvulus; and intussusception.

II. Intestinal obstruction due to neurogenic factors, (*a*) inhibition (paralytic) ileus, and (*b*) spastic or dynamic obstruction.

III. Vascular obstruction due to primary mesenteric occlusion from thrombosis and embolism.

The paralytic and spastic types (neurogenic) are fortunately usually self-limiting and amenable to simple gastric lavage and stimulating measures. Prognosis is usually good. The primary vascular lesions are rare but extremely fatal, recovery depending upon radical intestinal resections, with mortality in most clinics exceeding 90 percent.

Mechanical obstruction constitutes the major group commonly seen which despite a century of effort continues to carry a mortality rate of nearly 50 percent. It is the great group that we wish to discuss today. Surgical measures differ widely in various clinics but all agree that prompt action is urgently necessary. Holden (1) states that "Every recovery from acute mechanical obstruction is due to modern surgery; without surgery all would die."

Etiology.—Acute mechanical obstruction may follow (*a*) congenital or acquired strictures that narrow the intestinal lumen; (*b*) the obstruction is frequently due to adhesions and bands of inflammatory or traumatic origin following abdominal operations; (*c*) hernia; (*d*) volvulus; and (*e*) intussusception are also important etiological factors.

The role of operative surgery is important and constitutes a great field for prophylactic measures. Attention to operative technique, gentle intra-abdominal manipulations, minimum traction with no rough handling of the intestines will curtail greatly post operative adhesions and inflammation.

Pathology.—The mechanism of death in intestinal obstruction has been disputed for years. Failure to restore promptly the continuity of the intestinal canal portends death. All agree that intestinal obstruction is intolerable but many are not in accord as to the death producing factors.

There are theories based upon experimental work with animals that indicate toxic absorption of certain poisons accumulated or produced in the obstructed segment of intestine. The toxic features in intestinal obstruction continue to be accepted in many clinics but are being gradually displaced by later findings that offer a field for definite therapeutic measures.

The work of Hartwell and Hoguet (3), McIver and Gamble (4) established the important clinical facts of dehydration and disturbance of the acid base structure by loss of essential body fluids and digestive secretions from vomiting in high intestinal obstruction. They showed that this loss could be temporarily replaced by abundant saline injections.

Haden, Orr (5) found that high obstruction leads to a violent fall in the blood chlorides and that the administration of water or isotonic glucose solutions in this condition was far less beneficial than the injections of isotonic saline solutions.

The theory that toxic absorption occurred from the bowel is refuted by Wangensteen (6), who believes that very little if any absorption takes place from the obstructed loop and, further, considers the intestinal contents proximal to the obstruction to be no more toxic than the contents distal to the obstruction.

Wilkie (7) believes that the release of the contents of an obstructed loop into the bowel distal to the obstruction to be clinically beneficial. He stresses the importance of a physiological lack of the bowel below the obstruction by deprivation of the secretions, which should normally descend into it from above. High obstruction with immediate drainage of the bowel without release of the obstruction below is as rapidly fatal as an obstruction without drainage. The very fluid which in the bowel above the obstruction may appear to be toxic and threatening to life will prove lifesaving if introduced into the bowel below the obstruction.

Scott and Wangensteen (8) found that strangulation of the obstructed gut produces venous and arterial hemorrhage with infarcts. If the segment of intestine is long the blood loss may be large and deplete the circulation. They believe that the main cause of death

in this type of obstruction is damage to the bowel wall due to distention and strangulation.

The enormous loss of essential intestinal and interstitial fluids by vomiting in high obstruction is not present in low obstruction. The administration of saline injections is not nearly so effectual for the very practical reason that essential fluids and chlorides have not been lost. Distention with gas and fluids progresses slowly but steadily to the point of grave damage to the intestinal wall. Wangensteen observes "The old clinical adage that the high obstructions are the most serious, demands revision to read the low obstructions are the most hazardous."

Obstruction of the small bowel results in early distention of the entire proximal gut up to and including the stomach. Obstruction in the large bowel produces great distention which is confined to the colon, as the ileo-cecal valve and sphincter act in the manner of a check valve to preclude reverse peristalsis and regurgitation into the small intestine. Vomiting may be absent with little or no loss of essential fluids as seen in high obstruction.

McIver and coworkers (9) from extensive experimental work believe that the source of gaseous distention of an obstructed bowel is chiefly swallowed air.

From the above findings the following points appear of fundamental therapeutic value:

1. Simple unrelieved obstruction of the small intestine is fatal. The higher the obstruction the more rapid the death unless delayed by saline injections.

2. Simple unrelieved obstruction of the large intestine kills slower but surely. In the absence of great loss of body fluids the specific saline injections are not indicated. The fate of the patient appears to be dependent almost solely on releasing the distention of the bowel before viability of the obstructed segment is lost or seriously impaired.

3. Intestinal obstruction may be differentiated sharply into simple and strangulating types. The former permits of conservative treatment, the latter, strangulation, demands urgent surgery.

4. The principal death-producing factors are: Loss of essential fluids, dehydration, disturbance of the acid-base structure and damage to the blocked loops by distention from gas and dammed up fluids.

Diagnosis.—The onset of acute mechanical intestinal obstruction is sudden. The victim may be at work, at play, asleep or convalescing uneventfully from an abdominal operation, when like a bolt from the sky he is stricken. Surgical history is important, as old or recent abdominal scars indicate potential sources of obstruction. He may or may not appear acutely ill. The first few hours usually produce

little or no shock. The outstanding symptom is severe recurring abdominal pain. He is seized with intense cramps that are definitely intestinal in origin. The colicy pains are accompanied by gurgling intestinal sounds that are audible with the stethoscope and may be noted to be loudest always at the height of pain. This finding, highly important, establishes the presence of intestinal colic. Wangensteen (10) stresses the point that renal, biliary, or other colic may present audible intestinal noises but never with such time relation between height of pain and intestinal contraction.

Pain is followed by vomiting and complete stoppage of feces and flatus. There is little or no fever. No other acute abdominal disorder gives such definite progressive findings.

The early hours of onset are important. The patient usually recalls the very moment that the cramp or gas pains, as he calls them, began. The ordinary bellyache of dietary indiscretion or enterocolitis is excluded by bedside observations. If no relief is had from gastric lavage, and repeated enemas disclose absolute constipation, then blocked bowel is obvious. Depending on height of obstruction site the vomiting, at first reflex, progresses to almost constant overflow from the stomach in high obstruction. In low obstruction, distal to the ileo-cecal valve, vomiting, after the early gastric reflex, is usually absent. Fecal or sterocoraceous vomiting indicates that obstruction is in the small intestine. Fecal vomiting is rare in colonic obstruction.

Text books stress abdominal extension. This occurs early in paralytic obstruction but is a late sign in acute mechanical obstruction. The abdomen may be rounded out but is compressible. A simple flat X-ray film, patient supine, will give valuable aid in localizing the gas field. Checked by bedside clinical observations, the X-ray film reveals retention of gas in the blocked loops, the lower limit of gas field indicates the site of obstruction. With a series of interval pictures, following the insertion of an indwelling duodenal catheter for suction, progress of the obstruction or release of same may be noted. Wangensteen believes that non-absorbable gas is not formed locally as the result of putrefactive process, but concurs with McIver that the chief source of gaseous distention is swallowed air.

Of great significance is the absence or presence of abdominal rigidity or tenderness. There is little or no local tenderness and no rigidity in simple obstruction. There is marked rigidity and rebound tenderness in strangulation. This is explained by compression and traction of the blood supply with escape of irritating serosanguinous fluid into the peritoneal cavity. These findings are so constant that careful bedside observations should establish the presence of strangulation.

As an aid in early diagnosis the blood picture is not trustworthy. Blood chemistry changes occur only after severe vomiting and loss

of body fluids. The violent fall in blood chlorides rarely happens during the first 48 hours and will not occur even then if saline injections are copiously administered. If the urinary output can be maintained between 7 and 10 hundred cubic centimeters per day there will be no serious fall in the blood chlorides. The blood urea and carbon dioxide combining power, rapidly mounting in high obstruction, may also be kept within normal limits by these saline injections. The leukocyte count is elevated but is not consistently high in the early hours of obstruction, so is of small value in differential diagnosis.

Blocked bowel: The enema is a popular relief agent in early obstruction and may appear effective until the bowel distal to the obstruction is emptied. Then follows absolute constipation with complete stoppage of both feces and gas. Occasionally bloody mucus from the bowel may persist and indicate the violent reaction secondary to intussusception or the presence of colonic malignancy.

Fever: There is usually slight or no elevation in temperature. High temperature is so uncommon that fever of 3° or 4° F. is sufficient to exclude a provisional diagnosis of obstruction. To summarize, the diagnosis of intestinal obstruction rests upon the cardinal symptoms of pain due to intestinal colic accompanied by vomiting and absolute constipation. There is no fever. These are bedside observations requiring only stomach tube, enema can, stethoscope, clinical thermometer and cool judgment.

Treatment.—Despite the above-known factors in pathology and symptomatology, surgical relief for intestinal obstruction continues to carry a heavy mortality. There are two grave causes. First, delayed operation, the patient is not seen until the late phase of obstruction. Second, surgical procedures are too radical.

Patency of the intestinal canal is essential to life. An obstructed gut must be decompressed and, if present, nonviable segments excised. Only in early simple obstruction may the surgeon undertake a one stage exploration and full correction of the obstruction by operation of choice, such as evisceration, stripping, and emptying the bowel by enterostomy. In late simple obstruction such procedures kill. Decompression is urgently needed but must be limited to simple enterostomy. In strangulation obstruction, operative needs are desperately defined. There is no choice, the obstruction must be reduced and nonviable gut excised. Mortality is high but will be absolute without operation.

In former days, patients who had progressed beyond the early phase of simple obstruction died because they were rushed into surgery while in a state of dehydration and disturbed acid balance from excessive vomiting. The master surgeon John B. Deaver held that in the presence of nausea in a suspected obstruction case, "One should lavage stomach and, if washings are foul, open the abdomen."

DaCosta concurred with "Intermittent pain, nausea, inability to pass gas or feces with strong peristalsis warrants operation." These admonitions are still imperative but in delayed cases, as so frequently seen, surgical procedures carry a fearful mortality and must be restricted to simple drainage and decompression.

With the single exception of strangulation obstruction, the patient need not be rushed into surgery. Definite therapeutic agents are available that may restore him to a grade of fair if not good surgical risk.

These are: (1) Suction siphonage decompression by means of an indwelling duodenal catheter; (2) saline solutions; (3) blood transfusions.

Suction siphonage: The nasal catheter suction siphonage method of Wangensteen (11) is life saving and remarkably efficient in decompressing all simple obstructions except neoplastic strictures and occlusions distal to the ileocecal valve. Not only will decompression be accomplished but release of obstruction may follow and surgery be obviated in many types of simple mechanical obstruction. The adhesive type of mechanical obstruction due to band lesions of inflammatory origin will relent to this suction without operation in a majority of instances.

The mechanism consists of a simple water siphon that exerts constant but mild suction through a Levine type of duodenal catheter that is passed through the nose into the stomach and duodenum. Perforations are carried back on the catheter for 10 inches or more so that when the tip enters the duodenum concurrent suction may be exerted on both the stomach and the duodenum. The catheter is attached by rubber tubing to a series of water bottles, one inverted and suspended from an irrigation standard with leads to another water bottle, resting on floor about 75 cm. below the level of the patient's stomach. (See fig. 1.) Vomiting is at once controlled and the abdominal colic lessened. Morphine is not required and is contraindicated because of the danger of masking persisting obstruction. It is important to check the progress of decompression by bedside films taken with the patient in supine position. The films will disclose position of catheter and changes in the gas fields. If the obstruction is being favorably influenced, advance of the blocked gas from the small intestine into the large intestine will be seen. The suction prevents further vomiting and the comfort of the patient is remarkably enhanced. The administration of salines maintains the fluid balance and chemistry of the blood which may give false security unless X-ray films confirm satisfactory decompression and release of the obstruction. Should the films show no evidence of gas in the distal bowel segments and the suction siphonage drainage exceeds the fluid taken by mouth then operation should not be long delayed. The patient however, is in good condition for surgery.

The details of this type of drainage by nasal catheter suction is fully described in current surgical literature.

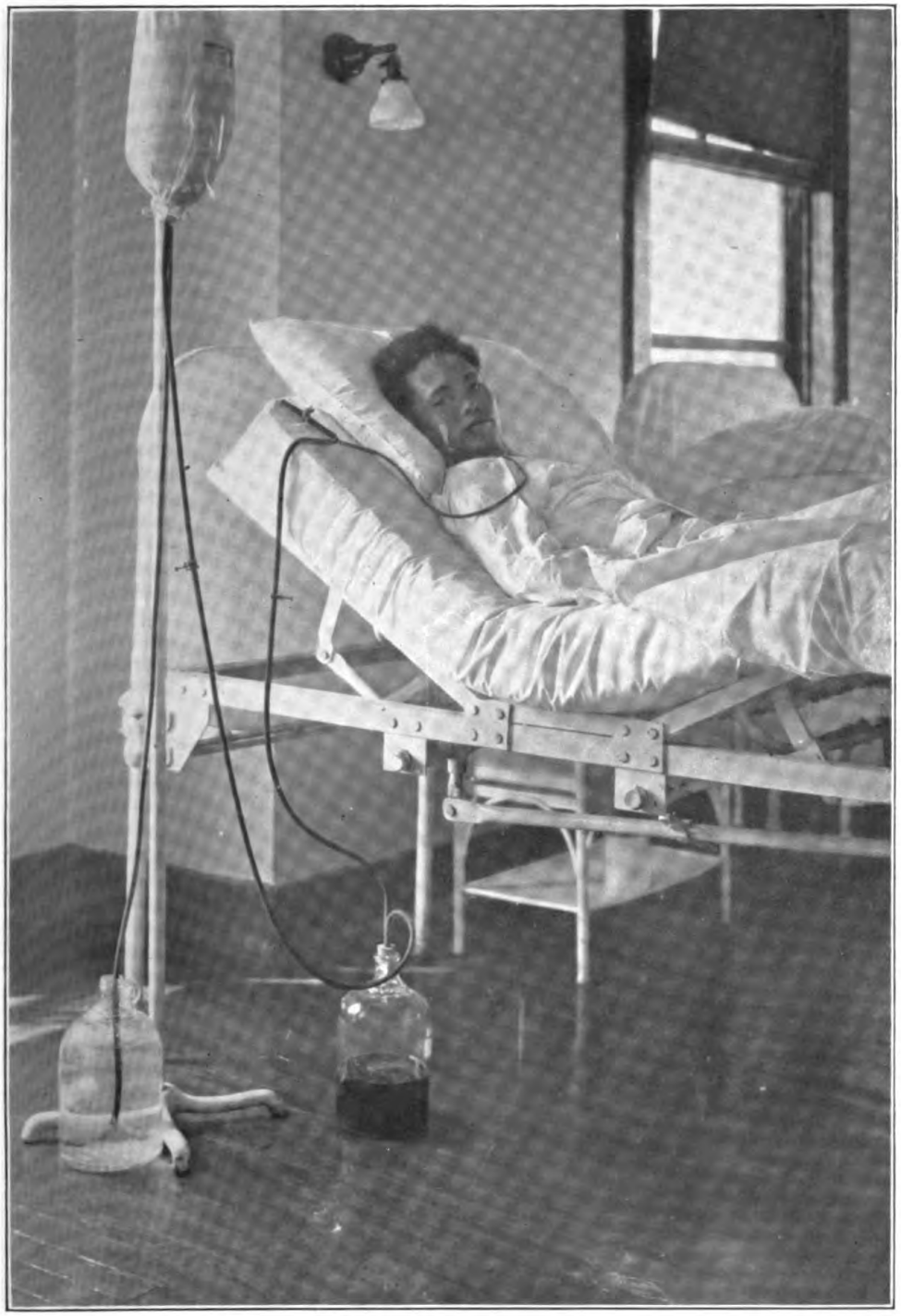
Wangensteen and coworkers by a grant from the graduate school fund, for medical research from the department of surgery of the University of Minnesota, perfected the technique and showed that an ordinary indwelling duodenal tube used as siphon with water sealed drainage is inefficient because of the presence of gas in the alimentary canal which enters the siphonage system and blocks its action.

Saline solutions: The loss of fluids by vomiting in high obstruction must be replaced promptly, otherwise the dangerous factors of alkalosis may threaten life. The copious injection of isotonic and hypertonic salt solutions will prevent dehydration and maintain a normal blood chemistry. The amount of saline used must be great, usually more than 3 to 4 thousand cubic centimeters per day, given by vein and beneath the skin. A good rule is to give enough fluid to maintain a total daily urinary output of not less than 1,000 cubic centimeters. In low obstruction the salines are far less beneficial for the loss of intestinal fluids is rarely serious. Here distention is the main death-dealing factor. Decompression must be had before irreparable damage to the intestinal wall, perforation and peritonitis takes place.

Blood transfusion: In strangulation obstruction with loss of blood into infarcted segments, blood transfusion may prove lifesaving. Transfusions are not indicated in simple obstruction where the blood-loss factor is unimportant and should not be substituted for saline solutions.

Morphine: The pain of intestinal colic is intense and must be differentiated sharply from pain of renal, biliary, or other colic. The cramp is intestinal and is not referred to the scapula, bladder, or external genitalia. The merciful administration of morphine is a serious error for the characteristic rhythm of pain and borborygmus so constant in intestinal colic is lost. The masking of this cardinal symptom may lull both patient and physician to grave delay and fatal error in diagnosis.

Prevention of intestinal obstruction: The prevention of intestinal obstruction of the post-operative mechanical type, by far the greatest group encountered, will be advanced by refinement in surgical technique in all abdominal operations. Wilkie (7) stresses the principle of de-tension in abdominal surgery. He states, "Deal only with the mobile organ if it be immobile, mobilize it. Do it not by force but by strategy based on the anatomical fact that all the abdominal organs were mobile before some became fixed, and therefore all may again be rendered mobile." The prevention of tension means minimum trauma, promotes primary healing, and safeguards against leaky suture lines. Post-operative atony of bowel, adhesions, and inflam-



PHOTOGRAPH OF PATIENT (R. N., OFF. STD. 3/CL., U. S. N.) UNDERGOING DECOMPRESSION FOR ADHESIVE TYPE OF ACUTE MECHANICAL INTESTINAL OBSTRUCTION.

An indwelling Lavine duodenal tube with perforations carried back ten inches from tip has been anchored through the nose. A Y-tube connection is attached to the proximal end of the duodenal tube for the purpose of irrigation and feeding; when not so employed this limb of the Y is tightly clamped. The other limb connects to rubber tubing that leads to long glass tube inserted through a two-hole tightly-fitting rubber stopper into an inverted water bottle suspended in canvas sling above height of patient's bed. A short glass tube is connected to rubber tubing through the second hole in the rubber stopper of this inverted water bottle and runs to second water bottle where it is water sealed at 30 inches below level of patient's stomach. This permits constant suction siphonage. The third bottle is simply connected into first tubing—one leading from patient to the high inverted water bottle—for the purpose of collecting the intestinal siphonage on the floor in a less conspicuous place than in the high inverted bottle.

matory reactions will be sharply curtailed. Now with the Wangensteen suction-siphonage method post-operative tension from within the stomach and intestinal lumen may be effectively controlled. Avoidance of distention following major abdominal operations is a most important prophylactic measure against mechanical obstruction. This is especially true during the early critical period of poor tone and motility of the bowel. Intestinal stasis and a few fibrinous adhesions invite obstruction.

CONCLUSION

Acute mechanical intestinal obstruction is a surgical lesion of exacting demands.

The principal death-producing factors are established as loss of essential fluids, dehydration, disturbance of the acid-base structure, and damage to the bowel wall by distention or strangulation.

These factors respond to definite therapeutic agents, namely saline injections, blood transfusions, and conservative decompression by the Wangensteen method of suction siphonage. Persistent unrelenting obstruction demands surgery, which should be conservative, in most instances a simple enterostomy.

Strangulation demands release of the obstruction and resection of all nonviable intestine.

The preliminary employment of conservative decompression by nasal-catheter suction and free use of salines removes all but the strangulation types of obstruction from the necessity and the dangers of hurried emergency surgery. Thus the desperately obstructed patient may be brought to operation, rated a fair if not safe surgical risk.

REFERENCES

(1) Holden, William B.: Surgical Treatment of Acute Intestinal Obstruction. Surg. Gyn. Obst., vol. L, no. 1A, Jan. 1930.

(2) Paine, Wangensteen: Drainage by Nasal Catheter Suction. Surg. Gyn. Obst., vol. LVII, no. 5, Nov. 1933.

(3) Hartwell, J. A., and Hoguet, G. B.: Experimental Intestinal Obstruction in Dogs with Special Reference to cause of Death and Treatment by Large Amounts of Normal Saline Solutions. J. A. M. A., 59: 82, 1912.

(4) McIver and Gamble: Symposium on Intestinal Obstruction. J. A. M. A., Nov. 17 and 24, 1928.

(5) Haden and Orr: Reducing the Surgical Risk In Acute Intestinal Obstruction. Am. J. Surg. Oct. 1925.

(6) Wangensteen, O. H., and Chunn, J. J.: Studies in Intestinal Obstruction, Comparison of Toxicity of Normal and Obstructed Intestinal Content. Arch. Surg. 1928. 16: 606–614.

(7) Wilkie, D. P. D.: Some Principles in Abdominal Surgery. Surg. Gyn. Obst., vol. L, no. LA, Jan.-1930.

(8) Scott, H. G., and Wangensteen, O. H.: Blood Losses in Experimental Intestinal Strangulations and Their Relationship to Degree of Shock and Death. Proc. Soc. Exper. Biol. and Med., 29: 748, 1932.

(9) McIver, M. A., Benedict, E. B., and Cline, J. W.: Post Operative Gaseous Distention of The Intestine. Arch. Surg., 1926, vol. XIII, 588.

(10) Wangensteen, O. H.: Practical Aspects of The Therapeutic Problem in Intestinal Obstruction. Internat. Clinics, vol. III, series 45, 1935.

(11) Paine G. R., and Wangensteen, O. H.: Nasal Catheter Suction Siphonage; Its Uses and Technique of Its Employment. Minnesota Med. J. 16: 96 (Feb.) 1933.

FRACTURE OF THE CARPAL SCAPHOID[1]

By F. R. Hook, Commander, Medical Corps, and J. D. Boone, Lieutenant, junior grade, Medical Corps, United States Navy

The true importance of fracture of the carpal scaphoid, considered from the standpoint of disability, has only recently been recognized.

Its frequency has been demonstrated as being far greater than was formerly thought. Wilson states that it comprises 86 percent of all carpal injuries. Various authors have placed its incidence at about 1 to 10 as compared with Colles and other fractures in the region of the wrist. In this hospital it has, during the period of this report, outnumbered all other wrist fractures. Out of 1,426 consecutive cases of fractures of the various bones of the hands and wrists, Snodgrass reports 125 in which the scaphoid was involved.

There are, undoubtedly, many cases that remain undiagnosed, but of those that are, in a majority of the cases the diagnosis is made too late for satisfactory treatment. The responsibility for this delay generally lies with the patient, but too frequently his doctor is to blame.

The fact that there is no deformity and that the pain in a recent case is not severe causes both patient and the physician to dismiss it as a "sprained wrist." After the passage of months the patient becomes concerned because the pain, rather than subsiding, actually becomes more severe. He reports the condition, an X-ray picture is taken, and the true cause of the symptoms is revealed. By this time aspetic necrosis, resulting from the disturbed blood supply, is advanced and there remains only slight hope of complete relief from symptoms.

It has been stated by competent authority that the disability resulting from untreated fractures of the carpal scaphoid is greater than that resulting from untreated Colles fractures. Those classed as untreated may well include most of the cases where treatment has been delayed. Although there have been recent improvements in the operative procedures in the treatment of late cases, the greatest hope for reducing the disability lies in the early recognition and the immediate institution of treatment.

Of the various types of fracture of the carpal scaphoid, the first and most important and the one encountered in the great majority

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of cases and which presents the greatest problem in treatment, is the so-called snapped-waist variety. This fracture is entirely intra-articular, occurring at the weakest portion of the bone or through that part which is generally called the neck.

Rarely is it produced by direct violence. Generally it is through a fall on the outstretched hand or from cranking a motor which has backfired. In either case the hand is usually in a position of dorsi-flexion and radial deviation when the violence is applied.

It was formerly thought that the fracture occurred as a result of impingement of the scaphoid between the head of the capitate and the articular surface of the radius; however, Thompson pointed out that the fracture is usually distal to that portion of the scaphoid that lies between these structures. He further demonstrated that there is a twisting violence applied to the bone during the motion of dorsi-flexion. The distal portion of the scaphoid, being attached to the strong collateral ligament and stabilized by the greater multangular, offers resistance to the rotary force that is applied to the mobile proximal portion by the head of the capitate. There results a snapping of the bone near the junction of the fixed and mobile parts.

Another type of fracture is that where the tuberosity has been evulsed. This is extra-articular and is generally incomplete. It results when the wrist is forced into extreme ulnar flexion. The tuberosity is pulled off by the inserted radial collateral ligament. The care of this type offers little difficulty.

A severely comminuted fracture is occasionally seen. There has also been described a compression fracture resulting from a crushing action against the radial styloid. The latter, however, is extremely rare.

It is unfortunate that there is no visible or palpable deformity associated with recent fractures of the scaphoid. There is, of course, pain, and the wrist motions are somewhat restricted, but the symptoms as a whole are such that the injury is likely to be neglected as a minor one. There is, however, particularly painful restriction of the motions of dorsi-flexion and radial deviation. The grip is markedly weakened. Pressure in the anatomical snuffbox elicits a deep tenderness. This is the most important sign. In 1 or 2 days swelling generally appears, at first localized in the anatomical snuffbox obliterating its outlines. As a rule there is no ecchymosis. There are in addition to these signs certain so-called percussion tests that are of value. In one the hand is placed in a position of radial deviation with the fingers flexed and the head of the third metacarpal is tapped. Pain is produced in the region of the fractured scaphoid. By changing to ulnar flexion and repeating the procedure no pain is produced unless the lunate is fractured. In a modification of this, percussion is applied to the tip of the extended thumb.

It may be said that in any case where there is a history of trauma to the wrist and examination reveals tenderness in the anatomical snuffbox, a fracture of the scaphoid should be suspected and an X-ray examination made.

It is not unusual for the X-ray picture to appear normal, even when a fracture exists. In order to lessen the chances for such an error the plates should be made with the hands in the position of ulnar deflexion, palms downward, and thumbs closely approximated. The tube is then centered at a point midway between the radial styloids.

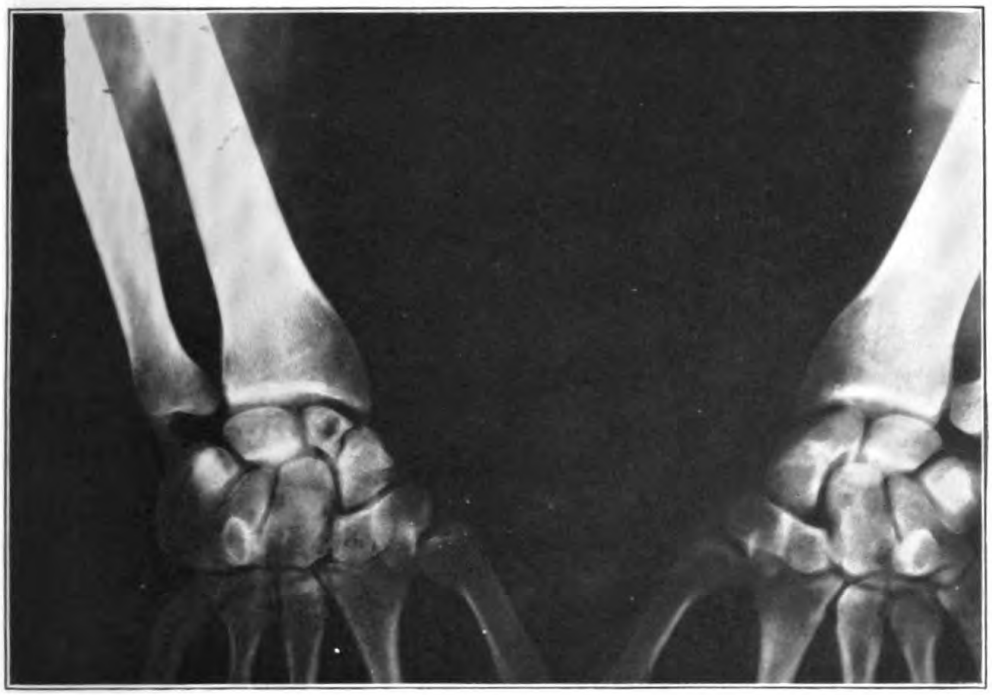
If the X-ray still fails to show a fracture and the symptoms persist, it is advisable to take a second picture after the passage of about 2 weeks. By this time, the slow absorption of bone along the fracture surfaces has probably widened the plane of fracture sufficiently for it to be seen.

The prognosis of fracture of the tuberosity of the scaphoid is generally considered as good. This fracture is extra-articular and its plane lies within the limits of the ligamentous attachment. The scaphoid is dependent for its blood supply upon the small terminal twigs from the radial and ulnar arteries which gain access to the bone by way of this ligament. Consequently when only the tuberosity is involved, many of the arterioles to the main portion of the bone remain uninjured and healing with callus formation proceeds rapidly.

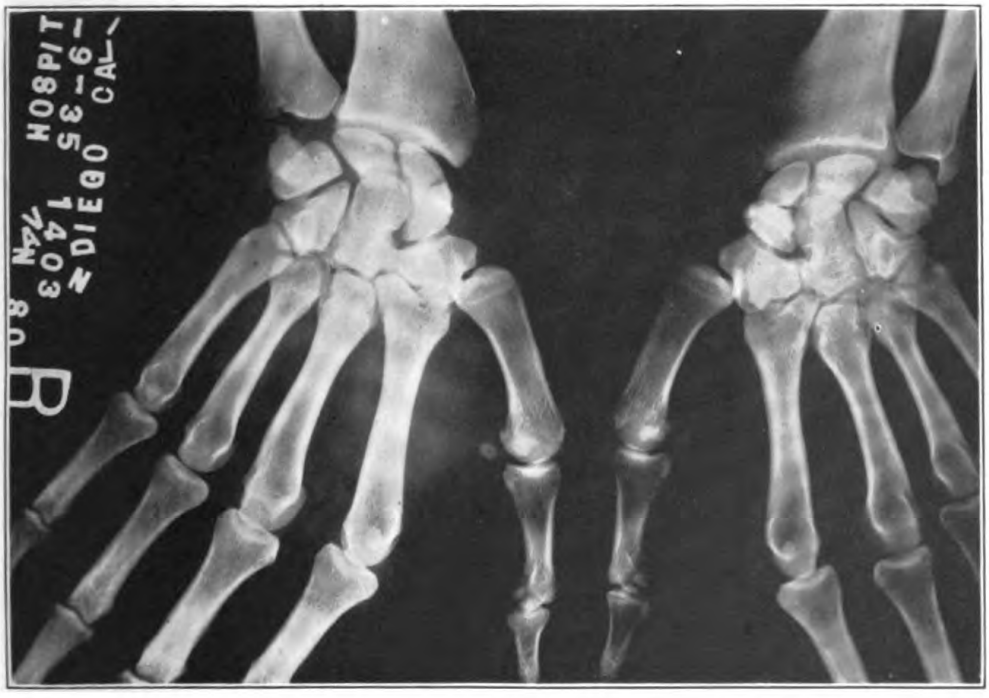
With the usual transverse fracture through the neck of the bone the situation is entirely different. The fracture is now intra-articular and its plane lies proximal to the ligamentous attachment. The proximal fragment of the bone is severed from the chief source of its blood supply. It is now generally agreed that the pseudoarthrosis which so frequently ensues is caused by this interference with the blood supply, plus the failure to immobilize early.

Soon after the fracture the proximal fragment dies and undergoes a slow asceptic necrosis. X-ray examination of an old case shows the distal fragment and the surrounding carpal bones to be lessened in their density as compared with the proximal fragment. This is explained by Speed as due to a loss of calcium salts in the former through the atrophy of disuse, whereas this change does not occur in the dead fragment which is now merely a foreign body and which eventually undergoes cystic degeneration.

After these changes occur the prognosis is extremely bad and operative removal is probably the wisest procedure. This is not done with the anticipation of securing a normally functioning wrist, but merely with the hope of relieving pain and halting the progress of the osteoarthritic changes which extend from the region of the scaphoid to the other parts of the wrist, resulting in varying degrees of limitation of movement, even to complete ankylosis.



CASE NO. 10.—SHOWS A FRACTURE OF 18 MONTHS' DURATION WITH TYPICAL CYSTIC DEGENERATION OF THE PROXIMAL FRAGMENT.



CASE NO. 18.—A RECENT COMMINUTED FRACTURE OF THE RIGHT SCAPHOID WITH AN OLD UNUNITED FRACTURE OF THE LEFT SCAPHOID.

Prior to the time when these changes can be recognized the prognosis becomes progressively poorer in direct ratio to the length of time that has elapsed since the injury. However, at any time during this period, immobilization should be given a trial.

A recent uncomplicated case, properly immobilized for a sufficiently long period of time, offers an excellent prognosis. New blood vessels bridge the gap between the two fragments and bone union results in a high percentage of cases. Böhler, who is a strong advocate of the nonpadded plaster cast, reports bone union in 100 percent of his cases.

The position should, of course, be that which will hold the fragments in the closest approximation. Berlin, by dissecting 60 wrists, demonstrated that a position between 40° and 50° of dorsiflexion with slight radial deviation invariably gives the best results. He showed that extension beyond this point causes a gap between the margins on the palmar aspect and that when there is less than 40° the margins on the dorsal surface are correspondingly separated. Radial deviation is desired in order to release the tension on the radial collateral ligament which stretches between the radial styloid and the tuberosity of the scaphoid.

The cast should be nonpadded and should include the palm, the forearm, and the thumb, holding the thumb in abduction and extension.

There is considerable difference of opinion relative to the period of immobilization, but it is generally conceded that the very minimum should be 6 weeks. Böhler says 6 months if necessary.

A rational procedure is to remove the cast at the end of the eighth week and, if there remains any tenderness over the scaphoid or if the X-ray does not show beginning union, to further immobilize until the desired result is obtained or until after the passage of several weeks it is evident that union will not occur.

In the latter case it is probably advisable to attempt to obtain union by one of the operative procedures that have recently been introduced. Prior to a very few years ago, excision of the bone or one of its fragments was the only surgical procedure considered. Some advocated the removal of only the proximal fragment, but Speed insisted that the entire bone be removed. In many cases excision is still the method of choice, for instance in recent cases where there is severe comminution or in old cases where the degeneration of the bone has progressed to that stage where any attempt to obtain union would be futile. In these cases, according to Speed, "the results are always beneficial no matter how long standing the fracture may be." However, in that large group of cases that are too old for simple immobilization, but which show no evidence of advanced degeneration and where there are no apparent osteoarthritic changes, one of the following methods might well be tried:

First is bone graft. This was successfully performed by Adams of Boston and since then Burnett and others have reported equally good results.

Another method that is proving very successful is the production of multiple drill holes, the drill being introduced at the distal end of the bone, near the point of entry of the arterioles and extending through both fragments. The fracture is then treated as a recent case. Of seven cases so treated by Soto-Hall and Haldeman, five healed with bone union.

During the past 2 years there have been 22 cases seen on the orthopedic service of this hospital. Twelve of these were recent, less than 1 month having elapsed since the injury, in fact less than 1 week in 11 of the cases. Three of the 10 old cases presented intervals of 18 months, 2 years, and 5 years respectively, with the remaining 7 varying from 7 weeks to 9 months and averaging about 6 months between the date of fracture and the beginning of treatment. It is interesting to note that one of the recent cases also showed an old ununited fracture of the opposite scaphoid. Just how long it had existed could not be definitely determined as there was a history of competent injuries 3 months, 5 months, and 15 years previously. The wrist was entirely symptomless during his stay in the hospital and received no treatment.

A review of the old cases well illustrates the cost of neglect. The oldest case in the series is that of a marine officer who sustained an injury to his right wrist when he was thrown from a horse in 1930. He thought that his wrist had been sprained but did not report to his medical officer. Pain continued in the wrist for approximately 1 year, after which there was apparent improvement. Recently, however, the symptoms have been increasing and examination 5 years after injury reveals a wrist that has practically no dorsal or lateral motion and only about 25 percent palmar flexion, marked weakness of the grip, and definite tenderness upon pressure over the anatomical snuffbox. The X-ray shows an old comminuted fracture of the right scaphoid with three fragments. Necrosis and cystic degeneration are apparent in one of the fragments and there are well-advanced osteoarthritic changes in all of the bones of the wrist.

Two patients, one of which is still under treatment at the present time, are of particular interest. These fractures were 6 and 7 months old, respectively, and neither presented any X-ray evidence of necrosis or osteoarthritic changes. They were both operated upon by drilling after the manner of Soto-Hall and Haldeman and then immobilized for a prolonged period. X-ray examination of the first patient 3 months after operation showed the presence of bone union and he was returned to duty with a good functioning wrist. The second patient is still in a cast.

Of the remaining seven old cases, five were invalided from the naval service and two were returned to duty. Of these two, however, both were experiencing rather marked symptoms at the time of their discharge to duty.

Of the 12 recent cases, only 1 was invalided from the service, 1 was discharged because of expiration of enlistment and 10 have returned to duty. It is believed that the one man who was surveyed out exaggerated his symptoms as the X-ray examination made after the usual period of immobilization showed bone union. However, he persistently complained of pain and weakness in the wrist and was finally given the benefit of the doubt.

It might be said that the cases in this group are of too recent origin to be compared with the later cases; that they may yet develop symptoms that will render them unfit for service. The only answer to this is offered by the X-ray. Prior to discharge from the hospital, eight of the recent cases showed the presence of bone union; of the remaining four, three were allowed to return to duty earlier than has more recently been the custom. Symptomatically they all promised good results, although the X-ray did not, at that time, show bone union. These cases were immobilized only for a period of 5 weeks whereas the more recent cases have been maintained in plaster for 8 to 10 weeks. The remaining recent case was one with a marked degree of displacement. After 10 weeks of immobilization there was no union and the functional result was so poor that the bone was excised. Upon return to duty he had no pain and only slight restriction of motion. Although he will not regain complete function, his wrist will, in all probability, remain serviceable.

At the time of discharge from the hospital nonunion was evident in all of the late cases with the exception of the one that was treated by multiple drill holes. The second case so treated is still in the hospital. These two represent the only cases in the series where attempts were made to obtain union by operative procedures. Of the remaining, two were treated by immobilization alone, one by physical therapy alone, four by excision, and in the last case surgery has been advised but not yet performed.

Typical of this group is case no. 13, H. T. L., seaman second class, age 25, admitted to the hospital on July 6, 1934. In December 1933 he fell from his bunk and landed upon his outstretched right hand. He reported to the sick bay the following morning, complaining of pain in the wrist. He was given soap liniment rubs during the next 3 weeks. An X-ray picture was then made and the patient was advised to wear a leather wrist band. Several weeks later he was transferred to another ship and, after the passage of another 2 months during which time the symptoms were becoming progressively more severe, the diagnosis of fractured scaphoid was made. He had been a violinist, but the pain and restricted motion made it impossible for him to continue to play.

When admitted to the hospital, examination revealed tenderness over the scaphoid with restriction of all motions of the wrist, but particularly that of

dorsi-flexion. An old ununited fracture of the right scaphoid with no apparent displacement was demonstrated by the X-ray.

Immobilization was given a trial for a period of 8 weeks. This was followed by physical therapy. There was no improvement and the bone was removed on October 4, 1934. On January17, 1935, he was invalided from the service. At that time he had about 10-percent power in grip, practically no radial or ulnar flexion, very slight dorsal flexion and only about 50-percent palmar flexion. He was again seen on July 19, 1935, and at that time he had almost a normal grip and about 75-percent motion in the wrist. He, however, still complained of pain on use.

In marked contrast is case no. 20, W. E. B., radioman third class, age 22. On March 15, 1935, while working on a plane, he jumped off and tripped, falling to the ground on his outstretched left hand. He thought that his wrist was sprained, being unable to grasp anything without experiencing severe pain. X-ray examination revealed a simple transverse fracture through the neck of the left carpal scaphoid. The part was immobilized in a nonpadded cast for a period of 8 weeks. An X-ray check plate was made after removal of the cast. The fracture that had been previously described could no longer be distinguished. On June 18, 1935, he returned to duty free from pain and with a normally functioning wrist.

CONCLUSIONS

1. The high degree of disability resulting from fracture of the carpal scaphoid is due chiefly to the failure in administering proper treatment immediately after the injury.

2. The majority of recent cases will, if properly immobilized for a sufficiently long period of time, heal with bony union.

3. The prognosis becomes progressively and rapidly poorer in proportion to the time that has elapsed between the date of injury and the institution of treatment.

4. One should keep in mind the fact that frequently the early symptoms are comparatively mild, and any patient who has received an injury of the wrist and presents tenderness in the anatomical snuffbox should have the advantage of an X-ray examination. If this is not available, immobilization should be instituted regardless of the inability to make a positive diagnosis.

5. The position of choice for immobilization is between 40° and 50° of dorsi-flexion with slight radial deviation. The cast should be nonpadded and should include the forearm, the palm and the thumb, holding the thumb in abduction and extension.

6. When simple immobilization has failed or in those older cases where there is no X-ray evidence of necrosis or osteoarthritic changes, an attempt should be made to obtain union by bone grafting or by the production of multiple drill holes through the fragments. These operative procedures are followed by a period of immobilization as in recent cases.

7. In the presence of necrosis or osteoarthritic changes, or when the above procedures have failed to produce union, the bone should be excised.

PHOTOGRAPH OF CASES NO. 18, 17, 14, 16, AND 15. READING FROM LEFT TO RIGHT.

TABLE NO. 1

Case	Date of admission to hospital	Type of fracture	Time elapsing between injury and admission to hospital	Method of treatment	Condition on discharge from hospital	Disposition	Sick days
1. H. M. J., B. M. 2c	7- 2-33	Fracture left scaphoid with posterior lateral displacement of proximal fragment.	1 day	Immobilization, 6 weeks; excision both fragments.	Good strength; no pain; some restriction of motion.	Duty, 11-10-33	131
2. W. B. C., S. 2c	9- 7-33	Comminuted fracture, left scaphoid.	11 months	Immobilization, 4 weeks; surgery refused.	Nonunion; poor function	I. S., 11-2-33	56
3. A. B., S. 2c	9- 7-33	Simple fracture left scaphoid.	2 days	Immobilization, 6 weeks; anterior-posterior plaster splints.	Bone union; normal function	Duty, 1-12-34	127
4. W. W. R., S. 2c	10-18-33	Comminuted fracture right scaphoid.	2 years	Excision of scaphoid	Marked weakness; some pain	I. S., 4-19-34	183
5. C. B. J., S. 1c	11-14-33	Simple fracture right scaphoid.	do	Physiotherapy; proximal fragment excised previous to admission to hospital.	Fairly good motion; some pain	Duty, 2-16-34	94
6. C. C. McC., Lt	11-29-33	do	5 days	Immobilization, 5 weeks; plaster splints.	Good function; bone union not present.	Duty, 1-3-34	35
7. W. E. E., S. 1c	12-21-33	Simple fracture left scaphoid.	7 days	Immobilization, 6 weeks; in plaster splints.	Good function; no union at 8 weeks.	Discharged end of enlistment, 2-15-34.	56
8. A. B. M., F. 1c	1-12-34	do	9 months	Excision of scaphoid	Pain and marked weakness of grip.	I. S., 5-10-34	118
9. G. G. C., S. 2c	2-10-34	Simple fracture right scaphoid.	3 months	Immobilization, 6 weeks; anterior-posterior splints.	Bone union; good function	Duty, 4-27-34	76
10. J. H. S., S. 1c	2-20-34	Simple fracture left scaphoid.	18 months	Physical therapy	Nonunion; fairly good function.	Duty, 3- -34	22
11. A. E. G., S. 1c	3-21-34	do	7 weeks	Immobilization, 10 weeks; plaster splints.	Nonunion; painful wrist	I. S., 9-24-34	187
12. V. D. K., C. R. M.	5-31-34	Comminuted fracture right scaphoid.	1 day	Immobilization, 5 weeks; plaster splints.	Fairly good function; bone union not present.	Duty, 7-10-34	40
13. H. T. L., S. 2c	7- 6-34	Simple fracture right scaphoid.	7 months	Immobilization, 9 weeks; excision of scaphoid.	Painful wrist; poor function	I. S., 1-17-35	225
14. S. A. W., F. 3c	12- 3-34	Comminuted fracture left scaphoid.	1 day	Nonpadded plaster cast for 10 weeks.	Bone union; poor function	I. S., 7-9-35	218
15. H. E. C., S. K. 1c	1-18-35	Simple fracture right scaphoid.	do	Nonpadded plaster cast for 8 weeks.	Bone union normal function	Duty, 6-6-35	139
16. H. E. A., A. S.	2-14-35	Comminuted fracture right scaphoid.	3 weeks	Nonpadded plaster cast for 10 weeks.	Bone union; good function	Duty, 6-4-35	110
17. J. A. S., F. 3c	2-24-35	Simple fracture left scaphoid.	3 days	do	Bone union; normal function	Duty, 8-23-35	180
18. J. E. J., F. 3c	3- 8-35	Simple fracture scaphoid bilateral.	Right, 1 day; left, 5 months (?).	Right, nonpadded cast 9 weeks; left, none.	Right, bone union; left, nonunion; normal function both.	Duty, 6-4-35	88
19. J. P. D., S. 2c	3-12-35	Comminuted fracture right scaphoid.	6 months	Multiple drill holes; nonpadded cast, 8 weeks.	Bone union; good function	Duty, 8-23-35	164

TABLE No. 1—Continued

Case	Date of admission to hospital	Type of fracture	Time elapsing between injury and admission to hospital	Method of treatment	Condition on discharge from hospital	Disposition	Sick days
20. W. E. B., R.M. 3c.	3-18-35	Simple fracture left scaphoid.	4 days	Nonpadded plaster cast for 9 weeks.	Bone union; normal function	Duty, 6-18-35	92
21. F. B., S. 1c	6-10-35	Simple fracture right scaphoid.	7 months	Multiple drill holes; nonpadded cast.	Still in cast		
22. W. K. S., Capt. U. S. M. C.	Not admitted	Comminuted fracture right scaphoid.	5 years	Physiotherapy; hospitalization refused.	Gradually decreasing function		

REFERENCES

Adams, J. D.: Fracture of the Carpal Scaphoid. New Eng. J. of Med., 198: 401, April 1928.

Berlin, David: Position in the Treatment of Fracture of the Carpal Scaphoid. New Eng. J. of Med., 201: 574, Sept. 19, 1929.

Böhler, Lorenz: The Treatment of Fractures. Wm. Maurdrick, 2d ed., 1930.

Burnett, J. H.: Fracture of the Carpal Scaphoid. New Eng. J. of Med., 200: 126, January 1929.

Burnett, J. H.: Fracture of the (Navicular) Carpal Scaphoid. New Eng. J. of Med., 211: 56, July 12, 1934.

Burnett, J. H.: Fracture of the (Navicular) Carpal Scaphoid. Surg. Obs. & Gyn., 60: 529, February 1935.

Hopkins, F. S.: Fractures of the Scaphoid in Athletes. New Eng. J. of Med., 209: 687, Oct. 5, 1933.

Lewis, Dean.: Practice of Surgery, vol. 2, ch. 4, p. 85.

Murray, Gordon: Bone Graft for Nonunion of the Carpal Scaphoid. Surg. Obs. & Gyn., 60: 540, February 1935.

Scudder: The Treatment of Fractures. W. B. Saunders Co., 9th ed.

Snodgrass, L. E.: End Results of Carpal Scaphoid Fractures. Ann. of Surg. 97: 209, February 1933.

Soto-Hall, Ralph, and Halderman, K. O.: Treatment of Fractures of the Carpal Scaphoid. Jour. Bone and Joint Dis., 16: 822, October 1934.

Speed, Kellog: Traumatic Injuries of the Carpus. D. Appleton & Co., 1925.

Thompson, J. E.: Fractures of the Carpal Navicular and Triquetrum Bones. Amer. Jour. of Surg., 21: 214, August 1933.

Wilson and Cochrane: Fractures and Dislocations. J. B. Lippincott Co., Philadelphia, Pa., 1928.

A STUDY OF THE DIET IN RELATION TO DENTAL CARIES ACTIVITY IN 212 ENLISTED MEN AT THE PEARL HARBOR SUBMARINE BASE, HAWAII

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INTRODUCTION

Is dental disease an expression of a metabolic fault? If so, the establishment of the type of diet best suited to health and efficiency under a given set of conditions is of paramount importance to mankind and certainly none the less to the personnel of the American Army and Navy.

Within the last half century diet and teeth in Hawaii have undergone radical changes. The once beautiful teeth of the native people are today ravaged by decay. Native foods have been replaced by imported varieties. Newcomers to the islands living on much the same type of diet they had in temperate climates are said to be peculiarly susceptible to an increase in dental caries. If this claim could be substantiated into an indisputable fact and dietary and other factors associated with the increased susceptibility be deter-

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mined, much progress toward an understanding of the cause of caries would be made.

The recently arrived personnel at the submarine base, Pearl Harbor, afforded an opportunity to make such a study. With the approval of the commanding officer and the enthusiastic cooperation of Commander C. E. Morrow, of the Dental Division, the study of diet and teeth at this naval activity as reported below was made.

PROCEDURE

Examination of teeth.—The dental records of the enlisted personnel who had been examined shortly after their arrival in Hawaii were listed. Among them, 212 of the men represented were available for reexamination. Caries, all fillings, and missing teeth were charted on the standard form used in the Navy. Examinations were made by one of us (G. N. C.) and by Dr. C. E. Morrow, senior dental officer of the base, who had also made most of the examinations on the men on their arrival in Hawaii. In order to get uniformity of results in dental examinations, charts were checked against each other and it was found that interpretations of carious lesions made by the two examiners were identical.

These findings were then compared with those recorded on the first examination. This gave exact information as to the increase in carious teeth, carious lesions, fillings, and missing teeth. In the tabulations, when teeth are spoken of, one tooth is the unit. When defects are spoken of, the unit is one filling, one cavity, or one missing tooth. No effort was made to estimate the extension of a carious lesion. The men were then divided into groups corresponding to the length of their service in Hawaii and according to their ages. The findings in these several groups were tabulated and compared.

Diet analysis.—Quantities and cost of each type of food used in the daily rations for a period of 1 year and the total amount of waste for the same period were furnished by the commissary officer. From the various texts and food tables available (1), (2), (3), (4), the number of calories per pound of each type of food was obtained and the total calories contained in the amounts of foods used were calculated.

From the references cited, values for the various food constituents, as protein, fat, carbohydrate, calcium, phosphorus, etc., were obtained in terms of 100-calorie portions; and from these and the total calories, the total amounts of the various food constituents in the given quantity of food were determined. Meats vary greatly in their fat content and so in caloric values. In these, the amounts of inorganic constituents were calculated from the amounts of protein contained therein.

To determine average values for the various types of vegetables which were grouped under invoice of provisions furnished by the commissary officer as "fresh, canned, and dried", the frequency with which each type of vegetable was used was estimated and average values in terms of 100-calorie portions were determined for the various food constituents. For example, 16 vegetables in different proportions are represented in average values of the various constituents in the fresh vegetable group; 5 in the canned, and 3 in the dried. Likewise average values were obtained for cereals, fruits, cheese, meats, etc.

Table I shows the quantity of each type of foodstuff constituting the yearly ration for the entire personnel in pounds and calories. Table II shows the average value of the various types of foods used in the calculations.

FINDINGS

Grouping of men.—The 212 men examined were divided into 3 groups. The first group consisted of 109 men who had been in Hawaii from 6 to 12 months. The second group of 47 men had been in residence from 1 to 2 years, while the third group of 56 were completing from 2 to 5 years of service. It is felt that the dental findings on the first group are significant. In the other two groups they are regarded as merely suggestive due to the small number of men.

Time factor.—Referring to table III, it is shown that 23.5 percent of all defects in the mouths of the first group of men occurred in 6 to 12 months' time in Hawaii, 30 percent in the second group in 1 to 2 years, and 40 percent in the third group in 2 to 5 years. The average of the ages of the men in the first group is 28.7 years, 27.6 for the second, and 29.7 for the third; the average for all the men being 28.7. The age range for all was from 20 to 40 years. Of the total dental defects found in the mouths of the first group of men, 23.5 percent occurred in 1 year or less, while the remaining 76.5 percent occurred during an average period of 22 years (from sixth birthday to present age). These observations are more significant when it is remembered that after the age of 25 the immunity to dental caries is usually greatly increased.

Table III also shows that 73.4 percent of the men had an increase in carious teeth in 6 months to 1 year and that the average increase per man was 2.74 teeth. This is very high, as it has been estimated that the average increase of dental caries in American children of school age is 1.5 teeth per year. It will be noted that the average increase per man in defects is correspondingly high, being 2.97. Incidence of caries on arrival was 98.15 percent and remained the same. The 2 men of the 109 who had no dental defects on arrival and did not develop any during this period were Filipinos.

Chart I shows graphically the percentages of sound, carious, filled, and missing teeth in the average mouth of the 109 men on arrival in Hawaii and after a residence of 6 to 12 months.

Age factor.—In table IV the men of the first group are divided according to age. It was desired to determine whether the age factor affected our averages. The result failed to show that age was a factor

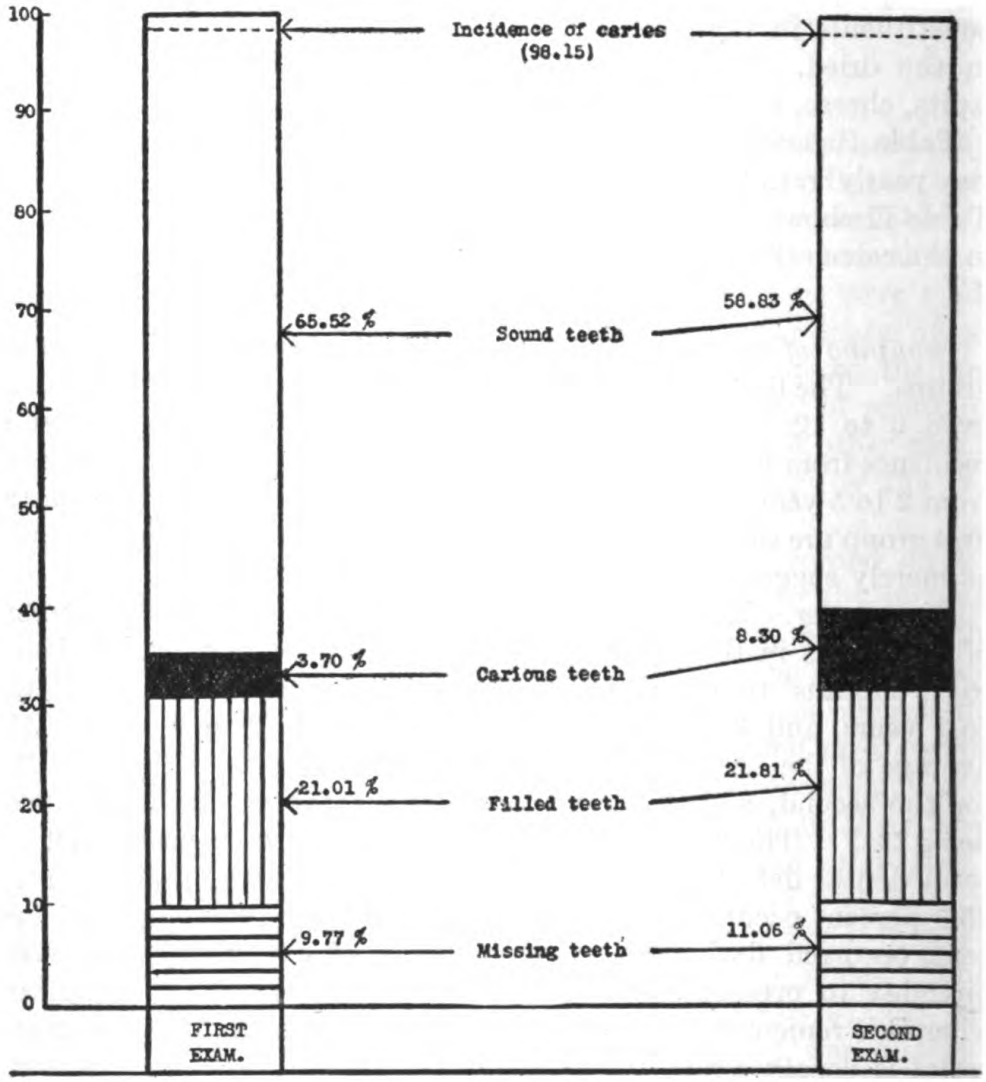


CHART I.—Average dental findings in 109 men on arrival and after 6 to 12 months residence in Hawaii.

in the increase of caries. The increase in total defects per man for each age group was about the same, being 2.91, 2.73, and 3.31 for groups I, II, III, respectively. The percentage increase in defects per man was 23.3, 23.7, and 23.6.

Taking the full complement of 32 teeth per man it was found that 65.52 percent of all the teeth were sound at the first examination, 3.70 percent were carious, 21.01 percent filled and 9.77 percent missing. On the second examination 58.83 percent were sound, 8.30 percent

carious, 21.87 percent filled, and 11.06 percent missing. These figures do not represent a true picture of the increase in carious teeth as in some instances a filled tooth may have developed a new cavity which in turn was filled during the interval between the first and second examinations. Also a tooth may have been both filled and carious. In such a case it was counted as carious and not filled.

It was noted that 68 of the 109 men or 62.3 percent had definite gingivitis in varying degrees, and an additional nine or 8.2 percent had clinical pyorrhea, making a total of 70.5 percent of the men with gingival disease. The ages of 8 of the 9 men with clinical pyorrhea ranged from 30 to 42 years. The other case was a Filipino 24 years of age with 32 sound teeth.

DIETARY FINDINGS

Table V shows the average daily quantity distribution of nutrients per man in the diet. Total food prepared, less waste, furnished 3,613 calories, 136.78 grams of protein, 418.11 grams of carbohydrate, 154.95 grams of fat, 0.844 gram of calcium, 1.943 grams of phosphorus, 0.027 gram of iron, and an excess of alkaline elements equivalent to 18.86 cubic centimeters of normal solution. The vitamin A content was found to be 8074.9 standard units and vitamin C, 219.1.

Table VI shows the average percentage distribution of nutrients and it is noted that 25.24 percent of the total calories, 53.69 percent of the protein, and 57.32 percent of acid elements are derived from meat and fish. Vegetables, including potato, furnish 13.73 percent of the calories, 13.71 percent of the protein and 77.94 percent of the excess alkali.

It is noted that the foods which provide the acid excess are meat and fish, eggs, cheese, and grain products. The alkali excess is derived from milk, molasses, vegetables, and fruits.

The largest amounts of phosphorus and iron are derived from meat and fish, being respectively about 40 and 42 percent of the total. Milk, even in the small quantity supplied by the navy diet, furnishes 36 percent of the available calcium while the vegetables supply 31 percent. A large part of the sodium and potassium (not determined) and iron also came from vegetables. Vitamin A was derived chiefly from vegetables and butter while the vitamin C content came entirely from vegetables and fruit. Grain products, sugar, molasses, and vegetables supplied most of the carbohydrate; flesh foods and butter, the fats.

No quantitative data on vitamin B are available. The principle sources are embryos of grain, yeast, vegetables, and fruits. Vitamin D occurs in small amounts in liver, cream and thin, green leaf vegetables. Egg yolk and cod-liver oil are rated as rich sources of this constituent. The principal source of vitamin D, however, as far as

man is concerned, is the sun. The ergosterol of the skin secretion is converted into this substance by the sun's ultra-violet rays.

DISCUSSION

What constitutes an adequate diet? Unfortunately, so far as we know there are no standards in which the condition of the teeth has been a criterion for the adequacy of the diet. Metabolism studies from which present available standards were deduced were in the most part conducted before the significance of vitamins was appreciated, and on a few cases for short periods of time.

For comparison we have taken the dietary study which was conducted by the United States Bureau of Labor Statistics, the United States Department of Agriculture, and by the New York Association

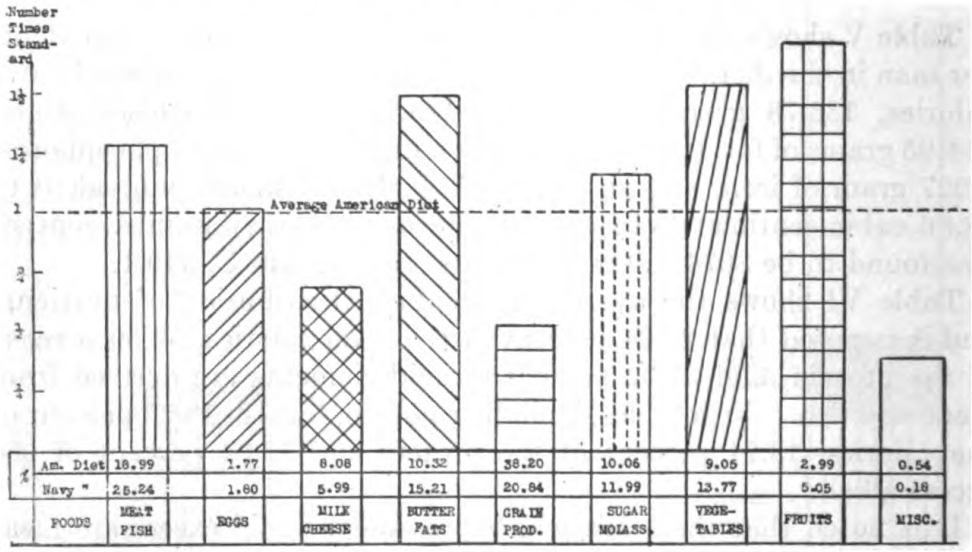


CHART II.—Comparison of Navy (submarine base, Pearl Harbor) and American diets (Sherman) in relation to distribution of calories among the various classes of foods.

for Improving the Poor. Analyses of the diet of the 224 American families were reported by Sherman (1).

Chart II shows the comparison of the two diets from the standpoint of distribution of calories. A rough comparison shows that the Navy ration contains approximately 19 percent more sugar and sweets, 25 percent more meat and fish, 47 percent more butter and fat, 52 percent more vegetables, and 69 percent more fruit than the average American diet. The amounts of egg were approximately the same in the averages of the two diets. The American diet, on the other hand, contains 83 percent more grain and 76 percent more milk products than the Navy ration.

If it is assumed that the optimal diet contains 12½ percent (10–15 percent) of its calories in the form of protein, 37.5 percent in the form of fat, and 50 percent in carbohydrate, the Navy diet closely approximates the ideal in these respects. It would be regarded as relatively

high in protein (15 percent of calories) and low in carbohydrate (46.5 percent of the calories), and normal in fat (38.5 percent).

In regard to the mineral constituents, if we accept the allowances for adult maintenance as reported by Sherman, we find that the Navy ration furnishes 24 percent more calcium, 47 percent more phosphorus, and 80 percent more iron than the above allowances.

The vitamin A and C contents are also relatively high. If we regard 1,500 standard units of vitamin A and 15 of vitamin C as minimum daily protective amounts, the Navy ration furnishes more than 5 times this amount of vitamin A and more than 14 times the amount of vitamin C. It must be remembered that vitamin contents of food are variable due to methods of cooking, handling, and other factors. For these reasons too much emphasis should not be placed upon quantitative estimations of vitamin content of the diet. It would appear, however, that this Navy ration contains generous amounts of vitamin A, B, and C. In Hawaii it is reported that the sun shines on an average of 7½ hours daily. This, certainly, should insure at least a sufficiency of vitamin D.

Potential reaction of the average American diet as reported by Sherman is neutral. The Navy ration contains an excess of approximately 19 cc normal alkali. It was surprising to us that this figure should be so low in view of the large amount of fruit and vegetables consumed.

The Becks-Simmonds standard diet.—In a paper entitled "Importance of an Adequate Diet for Health of Teeth and Paradentium", by H. Becks and Nina Simmonds, appearing in the October 1935 issue of the Journal of the American Dental Association, dietary prescriptions are given quite specifically. In table VII we have compared the composition of the Navy ration with that of Becks-Simmonds diet I, which, they state, "contains adequate amounts of all the minerals and vitamins as well as other dietary essentials." In addition to the values for protein, fat, carbohydrate, calcium, phosphorus, and iron which the authors reported, we have calculated the acid base and vitamin A and C values for comparison with those of the Navy ration.

Becks-Simmonds diet versus Navy ration.—In every factor except calcium the Navy ration is richer than the Becks-Simmonds specification for growing children, even. As it happened, calcium values assigned to some of their foods, particularly cereals and bread, are from two to three times higher than those used in our calculations. If their calcium values for bread and cereals alone were used, the calcium content of the Navy rations would be appreciably increased. However, according to the Becks-Simmonds scale, adult diets containing from 0.3 to 0.4 gram of calcium are appraised as fair; from 0.4 to 0.5 gram as good; and 0.7 gram or more as excellent. The

calcium value of the Navy ration is well within their optimum range. It is disappointing after one has read through pages of tables, menus and case histories to find at the very end of the paper a statement: It should be emphasized that as yet it is not known whether these are caries prevention diets. The authors state that they hope their recommended diets will prevent caries as well as other dental disease, and warn that to get the best result the diets must be faithfully followed and good oral hygiene practiced.

Becks-Simmonds diet versus Mooseheart.—We do not, however, share the same optimism or even hope of Becks and Simmonds that their diet will prevent or arrest caries. The diet of the average sailor at Pearl Harbor is optimum plus, according to their standards, and he lives in a sun-flooded environment. Notwithstanding, his rate of increase in dental caries is almost twice as great as that of the average American child. Perhaps climate makes the difference between Hawaii and the mainland. Turning to the latter (Mooseheart) we find that a diet containing approximately 1 quart of milk (B.-S. diet I contains three-fourths of a quart); 16 ounces of vegetables (B.-S. diet I contains 10 ounces); 8 ounces of fruit (B.-S. diet I contains 3½ ounces); 1 egg (B.-S. diet I contains 1 egg); resulted in 1 year, in active tooth decay in 83 percent of a group of 264 children, each of whom developed an average of 1.9 new cavities. The Mooseheart diet furnished 1.4 grams of calcium (B.-S. requirement is 1 gram per child), and 2 grams of phosphorus (B.-S. requirement is 1.227 grams). The addition of a pint of orange juice and the juice of one lemon retarded and arrested caries in most of the children, only 33.7 percent, showing an increase. The total amount of milk the children drank while taking the orange and lemon juice was less than they previously took. The net result as far as calcium, phosphorus and vitamins are concerned was a slight increase in calcium and phosphorus, an appreciable increase in vitamin A, and a tremendous increase in vitamin C. The orange and lemon alone furnished approximately 392 units of vitamin A and 294 units of C. The amounts of these vitamins are about one-fourth of the daily protective dose (1,500 standard units) of vitamin A, and more than 19 times the vitamin C requirement. There was no increase in vitamin D.

The more than 50 percent reduction in the rate of increase in tooth decay in these children while on the diet supplemented with the increased amounts of orange and lemon juice is convincing evidence of the presence of a caries-inhibiting factor or factors in these foods. Is this factor vitamin C? Unfortunately, Hanke and associates did not run a control group which would have proven this point beyond question. The fact, however, that outbreaks of scurvy, the disease that results from a deficiency of vitamin C, is not associated with an increase in dental caries, is irrefutable evidence that this food factor

does not control the body mechanism which determines immunity or susceptibility to tooth decay. According to the Becks-Simmonds specification for calcium and phosphorus, the Mooseheart diets were par excellence, the ratio of these elements in the three diets being well within the so-called optimum range. Likewise, all other food factors graded by the Becks-Simmonds standard rated more than optimum. What, then, is to be the criterion for measuring the adequacy of a diet consisting of ordinary food-stuffs and containing all of the known essential food constituents in adequate, if not generous amounts?

Acid-base balance.—As was shown by one of us (M. R. J.) (7) analyses of the 3-diet periods of 1 year each on the 264 children studied at Mooseheart furnish a promising clue. If the percentage of children showing an increase in caries during the 3-diet periods is plotted against the excess of alkali in the diets, an inverse relationship between these values is apparent. In other words, as the potential alkalinity of the various diets increased, the susceptibility to dental caries decreased. All three diets contain an excess of alkaline elements. The amounts were found to be 25 cc of normal solution during the control period; 55 cc during the test period (16 ounces of orange juice and the juice of one lemon added) and 22 cc during the recheck. The corresponding percentages of children who developed new cavities during these periods were 78, 34, and 83. The almost true diagonal line resulting, if extended, crossed the base line at a point near 80, which indicated that, if these diets, containing, as they did, amounts considered generous of the various known essential nutrients, had also yielded an excess of alkali equivalent to 80 cc normal solution, the decay process would have been completely arrested.

Analyses of diets reported by other investigators show tremendous differences in acid-base values, while the majority yield comparable values for the known essential constituents. It was interesting to find that all of the diets analyzed were either neutral in potential reaction, or alkaline. The inverse relationship between the incidence of caries and potential alkalinity of the diet that was observed in the Mooseheart diets was also found.

In Hawaii, so far as is known, every type of vegetation (including seaweeds) which grew here before the arrival of visitors 100 years ago yielded an alkaline ash. Fish was the main source of the acid elements. The diet as a whole was high in potential alkalinity and although it undoubtedly contained adequate amounts of all essential food constituents (the large stature, fine physique, and beautiful teeth of the native people attesting), the absolute amounts of calcium and phosphorus were probably low if measured by present-day standards. These observations have been duplicated in Samoa and other Pacific islands.

Relative values of foods.—The potential reaction of any diet depends upon the relative proportions of its various constituents. For instance, a strictly vegetarian diet may be either acid or alkaline in character depending not only upon the relative proportions of grains and "vegetables", but upon the *type* of vegetables composing it. It is perfectly possible for a diet containing a large amount of grain to be high in potential alkalinity, if, among the vegetables, is included a certain percentage of the leafy variety. Grains and greens supplement one another in many ways. Likewise, a diet consisting essentially of flesh may be potentially alkaline if it contains a certain percentage of blood and fat. The first choice of animal tissues of wild carnivora is blood which is potentially alkaline in reaction. Muscle, which contains an excess of acid elements, is the least desired. The Eskimo supplies a large percent of his caloric needs with blubber. For this reason the absolute amount of ash in his diet is probably low if measured by accepted standards. He drinks blood and eats the partially digested roots, shrubs, etc., which he obtains from the stomach of deer. The potential reaction of his diet is unknown.

Becks and Simmonds list certain vegetables as interchangeable in their diets. One such group contains corn, peas, carrots, greens, cauliflower, string beans, turnips and onions. Servings of these foods (approximately one-half cup) vary in excess acid-alkali content from 2.7 cc acid (corn) to 27 cc of alkali (spinach). If the potential reaction of a diet is a factor in maintaining the mineral balance in the body, a "workaday" knowledge of the acid-base balance of foods is of prime importance to the civilized world. Whether it is *the prime* factor in controlling that body mechanism which determines susceptibility and immunity to tooth decay is not proven. That it *may be* cannot be gainsaid; and *if it is*, corn and spinach cannot be interchanged successfully in any diet.

The civilized world is confronted with a nutritional problem of first magnitude. Tooth decay is rampant in many places and on the increase the world over. We may be certain that the nutritional fault which expresses itself in decaying teeth is leaving its imprint elsewhere in the body. It works in a subtle and insidious way slowly undermining resistance to diseases of all kinds. Among the 212 men examined for this study 208 had active tooth decay. The average number of defects (cavities, fillings, and missing teeth) per man was 14.78. The rate of increase in caries is alarming. Can it be stopped, and if so, how?

Suggested changes.—As has been shown, the Navy ration rates as par excellence according to generally accepted standards. It is low in potential alkalinity, a factor which is disregarded entirely by some (8) (9), or considered unimportant by others (10) (11).

Chart III shows graphically the magnitude of changes in the various constituents of the diet resulting from the replacement of certain acid ash with alkaline ash foods of comparable caloric values. Milk and meat are protein rich foods. Milk is potentially alkaline and meat acid. By reducing the meat in the diet 300 calories (about one-third pound) and increasing the milk 300 calories (about 14 ounces) the potential reaction of the diet is appreciably increased and other changes which appear to be desirable introduced. Similar changes result from the substitution of raw vegetables for 100 calories in the form of bread (1 medium slice); and molasses or cane sirup for 100 calories of refined sugar (2 T). The net results of such substi-

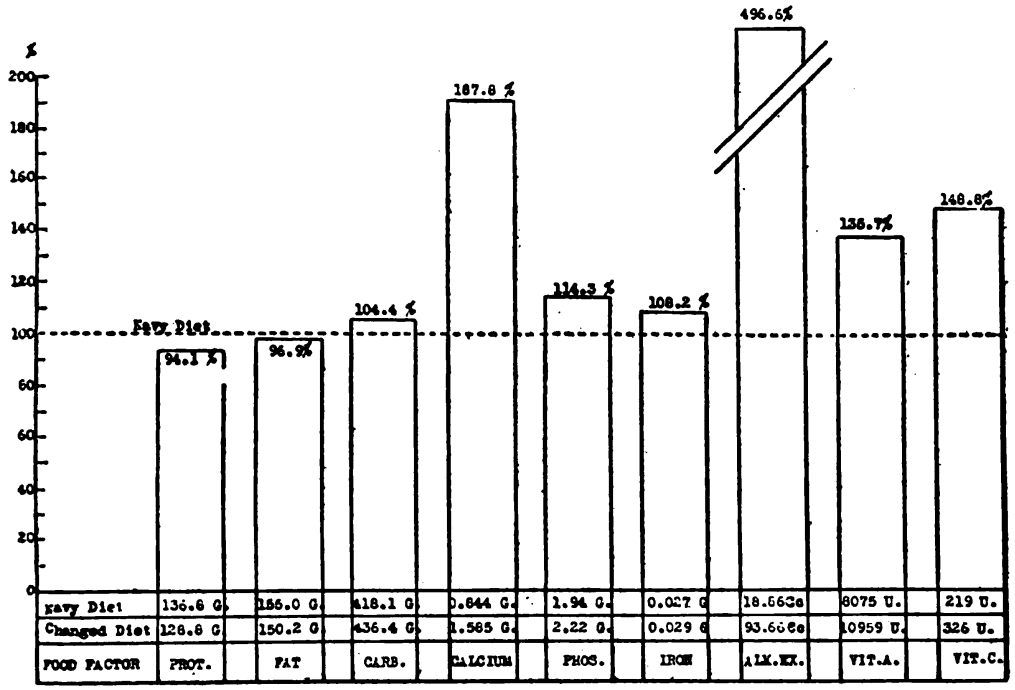


CHART III.—Percentage changes in food factors obtained by replacing 300 meat calories with 300 from milk; 100 bread calories with 100 from raw vegetables; 100 sugar calories with 100 from molasses.

tution is a slight reduction in protein and fat; a slight increase in carbohydrate; an appreciable increase in phosphorus and iron; a large increase in vitamins and calcium; and a net increase of 396.6 percent in potential alkalinity, or from 18.86 cc normal solution to 93.66 cc. Whether such changes in the above diet would prevent and arrest dental caries in the men is unknown.

Diets in Hawaii consisting essentially of fruits, roots, tubers, and other vegetables with moderate amounts of fish, meat, and egg with or without milk are associated with sound teeth. The dietary factor or factors that inhibit the decay process is apparently in fruits and vegetables. The almost universal freedom from enamel caries and extensive atrophy of the alveolar process observed in the adult Samoans whose diet consisted of fruits and vegetables with insignif-

icant amounts of fish, indicate a certain optimal limit of the decay inhibiting factors. In excess, this factor results in deterioration of the bone structure of the human body. The alveolar process is a transitory tissue. It is apparently the first to succumb to the caries inhibiting factor. Sound teeth become loose and fall out. It may be that fruits and vegetables contain a hitherto unknown substance which is specific for dental health. Until that substance is discovered, the potential reaction of an otherwise "adequate" diet appears to be not only *our best*, but *only* guide for the control of dental disease.

Cost.—Table VIII shows the composition and cost of the Navy ration and suggested changes. The milk used to replace meat may be either fresh, evaporated, or dried. In calculating the cost we have used evaporated, estimating the cost at the price listed on the invoice of provisions furnished by the commissary officer. It will be noted that the total cost of the ration with the suggested changes introduced is decreased 1.82 cents.

SUMMARY

A group of 212 enlisted men from the submarine base at Pearl Harbor, Hawaii, was examined and the differences in their dental condition on arrival and after 6 months to 5 years of residence in Hawaii were recorded. Examination showed progressive increase in dental defects paralleling time of service in Hawaii, the percentage being 23.50, 30.06, and 39.97 for the men who had been in residence 6–12 months; 1–2 years, and 2–5 years, respectively. The percentage increase in total defects per man for the 6 months' to 5 years' period was 28.80.

The incidence of caries among the men on arrival was 97.6 percent and on the second examination, 98.1 percent. Age did not appear to be a factor in the increase in caries. In three groups of men of different ages who had been in residence from 6–12 months the average number of new defects per man was 2.9, 2.7, and 3.3, for the 20- to 25-, 26- to 30-, and 31- to 40-year-old groups, respectively. In the same order of age groups the percentage increases in defects per man were 23.30, 23.70, and 23.57.

The diet consisted of approximately 17.2 ounces of meat; 1 egg; 9 ounces of whole milk; 1⅓ ounces of butter; 11 ounces of bread and other grain products; 2½ pounds of potato and other vegetables; 1 pound of fruit; 4 ounces sugar and sirup, and adjuncts. The diet furnished generous amounts of protein, calcium, phosphorus, iron, and vitamins if measured by accepted standards, and yielded an excess of 18.86 cc normal alkali. The excellence of the Navy ration according to accepted standards, with extensive decay suffered by the men during their short residence in Hawaii, suggests the existence of other factors. In Hawaii diets which contain no milk or grains but are rich in fruits, roots, tubers, and other vegetables are univer-

sally associated with sound teeth. It appears that the need for fruits and vegetables in the human diet parallels warmth and exposure to sunshine and the 3½ pounds of these foods in the Navy ration were insufficient under local conditions. There appears to be a factor (or factors) in fruits and vegetables, which, if present in insufficient amounts, results in enamel decay (caries and odontoclasia); and in excess, in atrophy of the alveolar bone. This cannot be attributed to any of the factors which are generally associated with tooth decay (calcium, phosphorus, vitamins). Fruits and vegetables as types of food contain an excess of alkaline elements over the acid, but the amounts of alkali excess vary tremendously in different varieties. Thus, the potential alkalinity of the diet depends not only on the quantity therein, but kinds. Until this unknown factor which inhibits tooth decay, on the one side, and in excess results in bone atrophy, on the other, is discovered, it appears that the potential alkalinity of an otherwise adequate diet is not only our *best* but *the only* guide for the control of dental disease.

Changes planned to increase the potential alkalinity of the diet were suggested and the various alterations occurring simultaneously in other food factors were discussed.

TABLE I.—*Total yearly ration Pearl Harbor submarine base*

Food	Total pounds	Total calories	Food	Total pounds	Total calories
Meat:			Vegetables:		
Preserved	10,485	9,436,500	Dried	14,557	22,781,705
Salted and smoked	43,109	32,762,840	Canned	68,991	16,419,858
Fresh	311,896	280,706,400	Fresh	287,735	49,202,685
			White potatoes	287,735	86,895,970
Total	365,490	322,905,740			
			Total	659,018	175,300,218
Eggs (dozen)	29,330	23,229,360			
			Fruit:		
Milk products:			Dried	2,437	3,021,880
Evaporated milk	32,055	18,271,350	Canned	20,080	9,636,400
Fresh milk	15,176	38,022,112	Preserved	9,663	14,252,925
Powdered milk	347	867,500	Fresh	188,685	37,737,000
Cheese	7,832	13,752,992			
			Total	220,865	64,648,205
Total	55,410	70,913,954			
			Sweets:		
Fats:			Sugar	81,892	148,633,980
Butter	28,744	100,345,304	Sirup (gallons)	229	4,220,928
Lard	23,109	94,484,720			
			Total	82,121	152,854,908
Total	51,853	194,830,024			
			Miscellaneous:		
Grain products:			Cocoa	956	2,158,648
Biscuit	12,931	24,245,625	Pickles	4,040	282,800
Bread	65,095	76,942,290			
Flour	85,051	136,932,110	Total	4,996	2,441,448
Cereal	19,609	32,099,933			
Total	182,686	270,219,958			

Table II.—*Average values of various types of foods used in calculations*

Food	Calories per pound	Values per 100-calorie portions									
		Protein (grams)	Carbohydrate (grams)	Fat (grams)	Calcium (grams)	Phosphorus (grams)	Iron (grams)	Acid excess (cubic centimeters normal)	Alkali excess (cubic centimeters normal)	Vitamin A (standard union)	Vitamin C (standard union)
Meat:											
Preserved	900	15.0	0	4.4	[1] 0.058	[1] 1.078	[1] 0.015	4.0	----------	----------	----------
Salted and smoked	760							1.4	----------	----------	----------
Fresh	900	7.0	0	8.0				4.0	----------	10	0
Eggs (per dozen)	792	9.05	0	7.09	.045	.122	.00205	7.0	----------	1,320	0
Milk:											
Evaporated	750	4.75	7.22	5.78	.174	.134	.00035	----------	3.3	295	0
Fresh (gallons)	2,512									330	0
Powdered	2,500									350	0
Cheese	1,756	6.05	.89	8.03	.212	.156	.00030	.49	----------	648	0
Fats:											
Butter	3,491	.13	0	11.05	.002	.002	.00003	0	0	700	0
Lard	4,080	0	0	11.11	0	0	0	0	0	0	0
Grain products:											
Biscuit	1,875	2.37	17.68	2.20	.006	.025	.00036	2.0	----------	5	0
Bread	1,182	3.60	20.40	.46	.011	.035	.00035	1.6	----------	5	0
Flour	1,610	3.20	21.20	.28	.006	.026	.00023	2.7	----------	0	0
Cereals	1,637	3.10	20.90	.50	.006	.053	.00038	3.2	----------	0	0
Vegetables:											
Dried	1,565	6.36	17.59	.46	.039	.128	.0022	----------	5.5	40	0
Canned	238	4.51	18.53	.81	.034	.115	.0015	----------	5.5	325	59
Fresh	171	4.28	19.03	.79	.136	.116	.0036	----------	23.0	2,637	65
White potato	302	2.69	21.97	.15	.016	.069	.0011	----------	8.6	40	15
Fruit:											
Dried	1,240	.71	23.74	.25	.018	.036	.001	----------	0	----------	0
Canned	480	.603	23.62	.44	.011	.019	.0003	----------	4.4	----------	40
Preserved	1,475	0	25.00	0	.005	.003	.0002	----------	0	[2] 208	0
Fresh	200	1.304	22.00	.74	.031	.033	.0007	----------	8.4	----------	50
Sweets:											
Sugar	1,815	0	25.00	0	0	0	0	----------	0	0	0
Sirup (gallons)	18,432	0	24.70	0	.034	.017	.00104	----------	5.8	0	0
Miscellaneous:											
Cocoa	2,258	4.35	7.00	5.90	.023	.143	.00054	.1	----------	0	0
Pickles	0	3.23	17.42	1.94	.095	.210	----------	0	----------	0	0

[1] Per 100 grams protein.
[2] Average.

TABLE III.—*Caries susceptibility in relation to time of service in Hawaii*

	Time of service			
	6–12 months	1–2 years	2–5 years	6 months–5 years
Number of men	109	47	56	212
First examination:				
Teeth missing, filled and carious	1,200	417	504	2,121
Average per man	11	8.8	9	10
Fillings and missing teeth	1,234	427	525	2,186
Carious lesions	143	32	73	248
Total defects	1,377	459	598	2,434
Average per man	12.63	9.77	10.68	11.48
Second examination:				
Teeth missing, filled and carious	1,435	519	706	2,660
Average per man	13.15	11.04	12.6	12.54
Fillings and missing teeth	1,404	491	659	2,554
Carious lesions	297	106	178	581
Total defects	1,701	597	837	3,135
Average per man	15.6	12.7	14.94	14.79
Increases:				
Men showing increase in carious teeth	80	33	51	164
Percent of men showing increase in carious teeth	73.4	70.2	91.0	77.3
Total number of new carious teeth	299	117	236	652
Average per man	2.74	2.49	4.21	3.07
Increase in defects	324	138	239	701
Average per man	2.97	2.93	4.26	3.31
Percent increase in filled, missing, and carious teeth, per man	19.58	24.46	40.07	25.41
Percent increase in total defects, per man	23.52	30.06	39.97	28.8

TABLE IV.—*Caries susceptibility in relation to age, in 109 men resident in Hawaii from 6 to 12 months*

	Age				Age		
	20–25 years	26–30 years	31–40 years		20–25 years	26–30 years	31–40 years
Number of men	33	41	35	Increases:			
First examination:				Number men showing increase in carious teeth	25	27	27
Carious lesions	54	42	47	Percent of men showing increase in carious teeth	75.7	65.8	74.5
Fillings and missing teeth	358	431	445	Total number new carious teeth	92	110	98
Total defects	412	473	492	Average per man	2.78	2.68	2.80
Average per man	12.48	11.51	14.05	Total increase in defects	96	112	116
Second examination:				Average per man	2.91	2.73	3.31
Carious lesions	91	109	97	Percent increase in defects	23.30	23.70	23.57
Fillings and missing teeth	417	476	511				
Total defects	508	585	608				
Average per man	15.39	14.25	17.37				

TABLE V.—*Quantity distribution of nutrients per man per day*

Food	Calories	Protein (grams)	Carbohydrate (grams)	Fat (grams)	Calcium (grams)	Phosphorus (grams)	Iron (grams)	Acid excess (cubic centimeter normal)	Alkali excess (cubic centimeter normal)	Vitamin A (standard units)	Vitamin C (standard units)
Meat:											
Preserved	28	19.00						1.12		0	0
Salted and smoked	98		0	72.68	0.045	0.837	0.0120	1.40		0	0
Fresh	838	58.63						33.52		83.80	0
Total	964	77.63	0	72.68	.045	.837	.0120	36.04		83.80	0
Eggs	69	6.24	0	4.89	.031	.084	.0014	4.83		910.80	0
Milk:											
Evaporated	71									209.45	0
Fresh (pasteurized)	114									372.90	0
Powdered	3									10.50	0
Total	188	8.93	13.57	10.87	.327	.252	.0006		6.20	592.85	0
Cheese	41	2.48	.36	3.29	.087	.064	.0001	.49		265.00	0
Fats:											
Butter	300	.75	0	64.20	.012	.012	.0002			2,100.00	0
Lard	281									0	0
Total	581	.75	0	64.20	.012	.012	.0002			2,100.00	0
Grain products:											
Biscuit	73	1.73	12.90	1.60	.004	.018	.0003	1.46		3.65	0
Bread	221	7.96	45.08	1.02	.024	.077	.0008	3.54		11.05	0
Flour	406	12.99	86.07	1.14	.024	.106	.0009	10.96		0	0
Cereal	96	3.00	20.06	.48	.006	.051	.0004	3.07		?	0
Total	796	25.68	164.11	4.24	.058	.252	.0024	19.03		14.70	0
Vegetables:											
Dried	68	4.32	11.96	.31	.027	.087	.0015		3.74	27.20	0
Canned	49	2.21	9.08	.40	.015	.056	.0007		2.77	159.00	28.91
Fresh	147	6.29	27.97	1.16	.200	.171	.0053		33.81	3,876.00	95.55
White potato	260	6.99	57.12	.39	.042	.179	.0031		22.36	104.00	39.00
Total	524	19.81	106.13	2.26	.284	.493	.0106		62.61	4,166.20	163.46

Fruit:											
Preserved	42	1.10	9.40	0	.002	.001	.0001	----------	----------	----------	0
Dried	9	.06	2.14	.02	.002	.003	.0001	----------	----------	----------	0
Canned	29	.17	6.85	.13	.003	.006	.0001	----------	1.28	----------	11.60
Fresh	113	1.45	24.86	.84	.035	.037	.0008	----------	9.49	----------	56.50
Total	193	2.78	43.25	.99	.042	.047	.0011	----------	10.77	401.44	68.10
Sweets:											
Sugar	443	0	110.75	0	0	0	0	----------	0	0	0
Sirup	13	0	3.21	0	.004	.002	.0001	----------	.75	0	0
Total	456	0	113.96	0	.004	.002	.0001	----------	.75	0	0
Miscellaneous:											
Cocoa	6	.26	.42	.35	.001	.009	.0000	----------	----------	0	0
Pickles	1	.03	.17	.02	.001	.002	.0000	----------	----------	?	?
Total	7	.29	.59	.37	.002	.011	.0000	----------	----------	0	0
Grand total	3,819	144.59	441.97	163.79	.892	2.054	.0285	60.39	80.33	8,534.79	231.56
Less waste	206	7.81	23.86	8.84	.048	.111	.0015	3.26	4.34	459.88	12.50
Total consumed	3,613	136.78	418.11	154.95	.844	1.943	.0270	57.13	75.99	8,074.91	219.06

TABLE VI.—*Average percentage distribution of nutrients*

Food	Calories	Protein	Carbohydrate	Fat	Calcium	Phosphorus	Iron	Acid excess	Alkali excess	Vitamin A	Vitamin C
Meat, Fish	25.24	53.69	-------	44.37	5.04	40.75	42.11	57.37	-------	0.98	0
Eggs	1.80	4.32	-------	2.99	3.47	4.09	4.91	7.68	-------	10.67	0
Milk	4.92	6.18	3.07	6.64	36.65	12.26	2.11	.78	7.71	6.94	0
Cheese	1.07	1.72	.08	2.01	9.75	3.11	.35	(1)	(2)	3.10	0
Butterfats	15.21	.52	-------	39.19	1.46	.58	.70	-------	-------	24.60	0
Grain products	20.84	17.76	37.13	2.59	6.50	12.27	8.42	34.16	-------	0.17	0
Sugar, Molasses	11.94	.07	25.78	-------	.45	.10	.35	-------	.93	0	0
Vegetables	13.72	13.71	24.11	1.38	31.84	24.00	37.20	-------	77.94	48.81	70.59
Fruit	5.05	1.92	9.78	.60	4.71	2.29	3.86	-------	13.40	4.70	29.41
Food adjuncts	.18	.02	.13	.23	.22	.53	(±)	-------	(±)	(±)	0

[1] Cheese.
[2] Milk.

TABLE VII.—*Comparison of nutritive values of the Navy ration and the Becks-Simmonds standard diet*

	Navy ration total	Becks-Simmonds basal diet no. 1 [1]		Navy ration total	Becks-Simmonds basal diet no. 1 [1]
Calories	3,613	1,526	Alkali excess	75.99	35.4
Protein	136.8	67.4	Net alkali excess	18.8	14.6
Fat	155.0	74.8	Vitamin A units	8,074.9	6,818.9
Carbohydrate	418.1	146.1	Vitamin C units	219.1	120.5
Calcium	.844	1.016	Number times requirement:		
Phosphorus	1.943	1.227	Vitamin A	5.4	4.7
Iron	.027	.012	Vitamin C	14.5	8.0
Acid excess	57.13	20.8			

[1] Gravy, salads, desserts, and "extra foods" not included.

TABLE VIII.—*Composition and cost of the Navy ration and suggested changes*

Food	Navy ration			Suggested ration		
	Calories per man per day	Approximate equivalent	Cost	Calories	Approximate equivalent	Cost
Meat and fish	964	17.20 ounces	0.1638	664	12 ounces	0.1170
Eggs	69	1 egg	.0237	69	1 egg	.0237
Milk	188	9.00 ounces	.0344	488	23.20 ounces	[1].0560
Cheese	41	0.08 ounce	.0046	41	0.08 ounce	.0046
Butter and fats	581	1.33 ounces	.0258	581	1.33 ounces	.0258
Grain products	796	11 ounces (bread)	.0324	696	9.66 ounces	.0265
Potato	260	13 ounces	.0521	260	13 ounces	.0651
Other vegetables	264	27 ounces		364	40 ounces	
Fruit	193	16 ounces	.0257	193	16 ounces	.0257
Sugar	443	3.90 ounces (7.8 T)	.0107	343	3 ounces (6.0 T)	.0081
Sirup	13	0.09 ounce (0.54 T) (mixed sirup).	.0002	113	0.92 ounce (5.6 T) (cane sirup).	.0027
Beverage	-------	----------	.0097	------	----------	.0097
Adjuncts	-------	----------	.0139	------	----------	.0139
Total	-------	----------	.3970	------	----------	.3788

[1] Evaporated milk.

REFERENCES

(1) Sherman, H. C., Food Products, The McMillan Company, 1914.

(2) Sherman, H. C., Chemistry of Food and Nutrition, The McMillan Company, 1923.

(3) Rose, Mary Swartz, A Laboratory Hand-Book for Dietetics, The McMillan Company, 1917.

(4) Bradley, Alice V., Tables of Food Values, The Manual Arts Press, 1931.

(5) Becks, Herman and Simmonds, Nina J. A. D. A. 1935 October 1724.

(6) Hanke, M. T. and the Chicago Dental Research Club, Dental Cosmos, 1933, 75, 933.

(7) Jones, M. R., Dental Cosmos, June 1935, 77, 535.

(8) Cowgill, G. R., Dental Cosmos, 1934, 76, 223.

(9) Mellanby, May, Diet and Teeth, Medical Research Council Special Report Series No. 191.

(10) Boyd, J. D., Drain, C. L., and Sterns, Genevieve, J. Biol. Chem. 1933, 103, 327.

(11) Koehne, Martha, Bunting, R. W., and Morrell, Elsie, Amer. Jour. Dis. Child., 1934, 48, 6.

THE DIFFERENTIAL DIAGNOSIS OF CORONARY ARTERY DISEASE[4]

By ELLIS A. STEPHENS, Lieutenant Commander, Medical Corps, United States Navy, Retired

The differential diagnosis of disease of the coronary arteries may or may not be a difficult matter. In acute occlusion of a main artery or of one of the larger branches the clinical picture is sufficiently dramatic and characteristic to require no particular emphasis. When occlusion of the coronary arteries has been gradual there is often less evidence of myocardial insufficiency than when a slighter injury has followed the sudden occlusion of only one branch. The separation of coronary thrombosis with acute myocardial infarction from general coronary disease is artificial and may even be misleading unless one keeps clearly in mind all the variations and gradations that may be encountered, from slight encroachment on the lumen to complete blocking of a main vessel. Moreover, even in acute occlusion the time that has elapsed since the onset of the attack colors the symptomatology. Coronary embolism, a much rarer occurrence, is indistinguishable clinically from thrombosis. Symptoms and signs of coronary disease depend on the rate of development of the myocardial change, the amount of damage resulting, the efficiency of the undamaged coronary circulation, the coronary anastomoses, congenital variations, the strain on the heart and the sensitivity of the individual. Because acute occlusion is of such grave import and because the symptomatology differs from gradual closure it will be considered first.

The possibility of this cardiac accident being confused with upper abdominal disease, especially perforating gastric or duodenal ulcer, gall-stone colic or acute pancreatitis, has been noted by many observers

[4] Read at the weekly staff conference, Santa Monica Hospital, Santa Monica, Calif., Jan. 16, 1935.

and there remains very little to be added except to recall to mind the frequent coexistence of coronary artery and gall-bladder disease and that the presence of either one does not exclude the other. A history of long standing digestive disturbance does not necessarily exclude a coronary accident. Likewise one should not become too coronary minded lest important disease of the gastrointestinal tract be overlooked. In an elderly patient, or whenever the diagnosis is obscure, an electrocardiogram should be obtained before laprotomy is done.

Acute coronary closure with early congestive failure may, if the pain is atypical or absent, simulate pneumonia, in the aged. Dyspnea, fever, leucocytosis, basal râles and vasi-motor paresis are common to both, early in their courses. Examination of the heart with the finding of a weakened first sound at the apex, precordial friction rub, gallop rhythm or evidence of dilatation at the left ventricle usually dispels all doubt but occasionally when pneumonia is superimposed on an already weakened myocardium, signs of consolidation developing in a few days may compel one to change the diagnosis from acute coronary occlusion to pneumonia.

Another source of error is spontaneous pneuomthorax. The sudden onset with severe chest pain, shock and concomitant fall in blood pressure may suggest a cardiac accident and the displacement of the heart may simulate enlargement, but a careful examination of the lungs with the presence of unimpaired heart tones is usually sufficient to establish the correct diagnosis.

In considering the diagnosis of acute coronary occlusion it is advisable to keep in mind and to exclude rarer events such as diaphragmatic hernia and pulmonary embolism. In the former a careful history, revealing as it does antecedent stress on intra-abdominal structures, a careful physical examination which discloses thoracic organs displaced to right or left and lack of signs referable to the heart are usually sufficient to avoid error. In the latter the diagnostic problem is much more difficult. To be sure, pulmonary embolism is more often a postoperative accident, but it does occur often enough under other circumstances to cause confusion. Moreover, the electrocardiogram found associated with pulmonary embolism is not infrequently suggestive of a coronary accident so that one must depend on the clinical evidence to reach a decision. Haemoptysis is usual in pulmonary infarction but rare with coronary occlusion. Here it should be recalled that these pathological states may be found associated especially after coronary occlusion which chances to involve an artery in the wall of the right ventricle. Should the infarction extend to the endocardium of the chamber a mural thrombus may result and pieces of the thrombus may be ejected into the pulmonary circuit. This accident does not occur until several days after the initial closure in the coronary tree. Failure to recognize this

possibility may lead one to conclude that a second coronary accident has occurred when in reality the infarction is in the pulmonary circuit.

There are many causes of sudden severe pain in the chest but the foregoing include those most likely to cause confusion with the exception of functional angina pectoris which will be considered later.

It is the lesser grades of coronary disease—coronary stenosis or gradual closure of one of the small branches—that present the greater diagnostic difficulties.

Of the cardinal symptoms of heart disease, namely dyspnea, pain, that is the anginal syndrome, and palpitation, pain is the most important in the diagnosis of coronary stenosis. The heart may be able to maintain the general circulation and yet be unable properly to support the coronary circulation. For that reason dyspnea may be insignificant, while pain will usually be present although careful and prolonged inquiry may on occasion be necessary to uncover this symptom since many elderly individuals believe it is part of the aging process. While pain is the most important symptom of coronary disease it should not be forgotten that certain substitution symptoms such as the sudden onset of weakness, sweating, and breathlessness may be a more or less atypical expression of general myocardial weakness or occlusion of one of the small vessels. Palpitation is common to many heart derangements and is unreliable as a symptom. A sallow, unhealthy tint to the skin in an elderly person often suggests coronary disease.

Coronary disease, whether on an arteriosclerotic, atherosclerotic or inflammatory basis with insidious loss of myocardial reserve due to progressive obliteration of the arterial lumen resulting in myocardial fibrous replacement, must be differentiated from the following noncardiac conditions:

1. Poor physical condition.
2. Neuro-circulatory asthenia.
3. Cardiac neurosis.
4. Pulmonary tuberculosis.
5. Obscure pleurisy.
6. Fibrositis of the intercostal muscles.
7. Certain neurological states such as brachial neuritis, tumor of the cord or tabes dorsalis.
8. Upper abdominal disease.

First of all poor physical condition with vague chest pains due to fatigued respiratory muscles is frequently mistaken for heart disease usually erroneously termed myocarditis. The differentiation may be difficult. It may not be easy to disentangle the muscular and cardiovascular factors. The recognition that such an entity exists, the finding of muscular tenderness and tonic contractions, the negative

evidence on cardiovascular examination, on X-ray study and in electrocardiograms is usually sufficient to exclude coronary disease.

Neuro-circulatory asthenia, so frequently accompanied by precordial aching, associated with consciousness of the respiratory act often mistaken for dyspnea, and by palpitation, is a common source of error. The history of nervous strain, fatigue or recent infectious disease in a person of sensitive make-up is frequently the clue to the true condition. Morover, the precordial distress in neuro-circulatory asthenia is often relieved by pleasant exercise, the respiratory act is usually interrupted by sighing, a very important sign, and the palpitation is usually traceable to forceful heart action or minor irregularity such as premature beats. A normal electrocardiogram is valuable in excluding true coronary disease. The fact that neuro-circulatory asthenia may complicate coronary disease should not be forgotten. Such an association is very baffling and exact separation of the role played by each factor may not be possible.

The recognition of a cardiac neurosis usually presents no difficulties if a careful history is taken. An individual who has recently lost a relative or friend by heart disease or who may have witnessed sudden death due to heart disease, may exhibit many of the symptoms of coronary disease. The condition is allied to neuro-circulatory asthenia and the same principles apply in the differentiation.

Fibrositis in the intercoastal muscles may cause pain in the chest. It may take the form of submammary panniculitis. The pain thus occasioned may bear a superficial resemblance to the pain of coronary disease. Fibrositic areas are found in the muscles. The normal heart action and electrocardiogram exclude coronary disease.

Obscure chronic recurring pleurisy should cause no confusion if the close relationship of the symptoms to phases of respiration are observed.

The foregoing conditions are commonly diagnosed erroneously for coronary disease when none exists. The following represent conditions that are frequently wrongly diagnosed when coronary disease does exist:

Pulmonary tuberculosis is probably a rare source of error but that it has been diagnosed in the presence and instead of coronary disease is a matter of record. Chiefly for that reason is it mentioned. The radiation of pain of cardiac origin may often follow unsuspected pathways so that the offending organ may for a long time be overlooked. Pain in the back along the scapula with asthenia and hypotension has been attributed to pulmonary tuberculosis and the real condition—myocardial degeneration—missed. The differentiation should not be difficult; whatever errors have been made were probably due to inadequate examination.

Brachial neuritis, tumors of the cord and tabes dorsalis, because of the presence of pain in the thorax or upper extremities, have been diagnosed instead of coronary disease. Careful physical examination with the recognition that coronary disease may masquerade as a neurological derangement is important if one is to avoid these pitfalls of diagnosis.

Finally, pain in the upper abdomen, indigestion, gaseous eructations, nausea, and vomiting should never be attributed to disease of the abdominal viscera until one has thoroughly and carefully estimated the ability of the myocardium, which in turn is dependent on the efficiency of the coronary circulation. The gastrointestinal tract is often the first system to send out a warning that its blood supply is insufficient because of a weakened heart.

Aside from numerous extra-cardiac conditions, coronary disease in which pain is a prominent symptom is to be differentiated from functional angina pectoris due to (I) marked aortic regurgitation in which the low diastolic pressure prevents proper nourishment of the myocardium, (2) severe secondary or pernicious anemia, producing local anoxemia of the myocardium, (3) blocking of the mouths of the coronary arteries by vegetations as in bacterial endocarditis, rheumatic endocarditis, inflammatory swelling and puckering in luetic aortitis or by arterio-sclerotic plaques even though the coronary arteries are patent beyond, (4) excessive demand on the myocardium as in hypertension and thyrotoxicosis and (5) flabbiness of the heart muscle as in myxedema.

It should be remarked here that the common view that functional angina is of short duration is not necessarily true. I have observed a patient in whom angina pectoris lasted for 60 minutes or more in which serial electrocardiograms and thorough clinical study did not reveal coronary occlusion or important sclerosis. However, coronary artery disease, usual sclerosis and narrowing, less often inflammatory, is found in perhaps ninety percent of patients with the anginal syndrome. Inasmuch as structural changes in the coronary arteries may complicate every other type of heart disease it may be exceedingly difficult to separate the various factors contributing to cardiac inefficiency. The electrocardiograph is our most valuable aid in the differentiation and should be resorted to whenever the myocardium comes under suspicion. Even when the electrocardiogram is normal or equivocal it may prove to be a valuable record for comparison later on. Finally, the X-ray may occasionally reveal evidence of arteriosclerosis of the coronary arterial tree or the presence of a calcified infarct.

TRANSPORTATION OF INSANE PATIENTS FROM MARE ISLAND, CALIF., TO WASHINGTON, D. C.

By ALMA C. SMITH, Commander, and ALLAN S. CHRISMAN, Lieutenant (jr. gr.), Medical Corps, United States Navy

"You will proceed to San Francisco, Calif., and report to the Commandant of the twelfth Naval District for duty in connection with the transfer of a draft of Navy insane patients, from the Naval Hospital, Mare Island, to Washington, D. C."

This kind of order is likely to fall on any medical officer being transferred from west coast to east coast duty, and did, in fact, fall upon us this last year. There is not a great deal of specific rules of guidance to follow, and yet even given a thoroughly adequate staff, the consideration of certain points previous to taking the trip is necessary if one is to handle a draft of insane patients with smoothness. This article is written with the hope that it may aid other medical officers in future trips of this kind. It is merely a statement of the problems we met, the solutions we found, plus a few recommendations for improvement which were suggested to us in thinking the matter over.

Each year, sometimes twice a year, when sufficient patients from the mental divisions of the various west coast and Far East naval hospitals have been admitted to Mare Island, permission is asked of the Bureau of Medicine and Surgery to transport them to Washington, D. C., where they are examined and classified for permanent care. Some are dismissed, others are treated in the Washington Hospital, and still others are sent to St. Elizabeths.

When the Bureau of Medicine and Surgery receives the request for transportation it notifies the Bureau of Navigation, which requests the Military Railway Bureau to arrange for the necessary railway schedule, at the same time directing the Commandant of the Twelfth Naval District to cooperate in working out a schedule of railway equipment and movement.

The Bureau of Medicine and Surgery detail a medical officer and junior medical officer who are being transferred to the east coast to convoy the draft of patients. Also those hospital corpsmen who are short timers and due for a discharge on the east coast are gathered together at Mare Island and held for transfer as attendants with the draft. During their stay at Mare Island they are assigned duty in the psychopathic wards, where they become acquainted with the patients to be transported east. Also, one chief pharmacist's mate is usually assigned to the care of the health records, service records, and survey records of the insane patients; another to the care of the baggage, clothing, and property of the patients; and a third, to the supply of drugs, dressings, and other items of the medical and surgical kit.

The medical officer in charge of the draft and his assistant are generally ordered to report to the commandant of the Twelfth Naval District and to the commanding officer of the hospital a few days prior to the date set for the departure of the draft, and immediately upon reporting, the officer in charge will find that his duties and worries commence, for the general smoothness of the whole trip across the continent will depend upon how thoroughly, at this time, he organizes his various assistants, and makes other necessary plans and arrangements.

The following details should be considered and appropriate action taken.

1. Consult with the commanding and executive officers of the hospital to learn the number of patients in the draft, number of attendants in the draft, the day, hour, and place of departure, the railway schedule, number and kind of cars arranged for, and any other pertinent information these officers may be able to give.

For example, on the last transfer there were 31 patients, 36 Hospital Corps men, and 2 medical officers. The railway equipment consisted of 1 standard pullman, 2 tourist pullmans, 1 dining car, and 1 baggage car. The time of departure was the seventh of September at 3 p. m., the place Benecia, Calif. (a railway terminal station about 15 miles from the Mare Island Hospital).

2. Consult with the medical officer in charge of the psychopathic ward, and make sick call with him to learn as much as possible about the characteristics of each patient. Ascertain which ones have exhibited tendencies toward suicide, homicide, drug addiction, or other abnormalities which might cause trouble during the trip.

3. Organize the attendants and make out detailed plans for embarkation and for watch standing during the trip. Place one chief pharmacist's mate in charge of all records, another in charge of the bags and baggage, and a third in charge of the medical and surgical kit.

4. Inspect all records. Inspect patients' clothing to make sure that each patient has clothing which will be presentable for the trip.

5. Confer with the supply officer and obtain from him the necessary official request forms for railway tickets and meal tickets for each member of the entire party. Also, obtain from him all valuables belonging to the patients which he may have been holding for safekeeping.

A recital of the details entailed during the day of departure may be of value to one embarked upon this project. On this day the railway company places the necessary cars on a siding at Benecia. As has been suggested in the foregoing plan, the Hospital Corps men and attendants have been previously divided into details shown in the attached watch list. Watch no. 2 reported at 8 a. m. to the

first lieutenant of the hospital to handle baggage. This baggage watch also accompanied the baggage to the train and remained on watch in the baggage car. Watch no. 1 accompanied the first ambulance load of patients to the train, and remained on the train on watch in the patients' car. Watch nos. 3 and 4 accompanied the succeeding ambulance loads of patients to the train until all patients were aboard. Each patient had been previously assigned to a specified seat in the patients' car, and as each ambulance load arrived at the train, the patients were mustered and taken immediately to their preassigned seat.

Prior to the arrival of the patients at the train the officer in charge must inspect the train to make sure the cars are in the proper order and to ascertain that the equipment and fittings are satisfactory. The most satisfactory arrangement of the cars from forward aft is as follows: (*a*) Baggage car, (*b*) Pullman car for Hospital Corps men attendants, (*c*) dining car, (*d*) Pullman car for patients, and (*e*) Pullman car for officer in charge, assistant medical officer, and chief petty officers. (This car should have a stateroom available for use as a hospital bed in case of serious illness of any patient.)

The patients' car must have all outside windows securely blocked to make it impossible for them to be raised more than 5 inches. Such a precaution is necessary to prevent any patient from jumping out of the car. A medical officer in charge of a previous trip confided to us that while they were crossing a particularly dry and uninviting stretch of desert country in Colorado, one patient, without warning, suddenly raised both the inner and outer windows, and though the train was in motion, jumped out. The train then had to be stopped in order that the escaped patient might be returned. This, however, was not accomplished without a considerable chase. When questioned concerning his reason for jumping from the moving train the patient's only reply was: "This country looks good to me."

Not only must precautions be taken to prevent the patient from getting out, but he must also be prevented from locking himself in a room. For this reason Hospital Corps men should be stationed at each door of the patients' car, and the toilet and washroom doors must be securely bolted or fastened in the open position. The locks on the toilet doors should be removed.

Arrangements must be made with the representative of the dining car for meals. Patients may be marched into the dining car for their meals, but it causes much less confusion and stress, both for the patients and those in charge, to have their meals served in their own car. Under such an arrangement collapsible tables are set up between each seat and the food served on them. Because the Pullman waiters are more accustomed to the motions of the train than the untrained Navy personnel it is advisable that they do the serving

of the food. It is necessary that the menu be inspected and only foods which can be eaten with a spoon allowed, for no knives or forks are permissible. Hospital Corps attendants and medical officers eat their meals in the dining car. However, a medical officer should be present at each meal in the patients' car to inspect the food and the service. The fact that someone takes an interest in their welfare seems to have a good effect upon the patients, thus increasing their confidence and cooperation.

Each patient has a small ditty bag containing shaving gear and toilet articles. These ditty bags are kept in the baggage car, and at regular intervals are brought out to the washroom for the patients' use. One patient at a time is permitted to use his shaving and toilet articles while a Hospital Corps man stands guard; this corps man also checks the razor and blades to ascertain that they are returned to the bag after use.

Safety, food, and sanitation arrangements should be followed with plans for the patient's contentment and feeling of well-being. In order to keep the patients interested and occupied various "breaks" in the day can be scheduled. About midmorning and again about midafternoon the patients enjoy the distribution of candy, cigarettes, chewing gum, and picture magazines.

Another important daily event is the exercise period. Arrangements can be made with the railway representative to stop 30 minutes each day so that patients, in charge of the attendants, can be exercised. These stops should be made at some isolated place where there is sufficient space in which to exercise the whole group. There should be no crowds, for crowds often confuse and excite the insane, causing them to become unruly and obstreperous.

Sufficient drugs, medical and surgical equipment, and restraint apparatus must be carried in the medical kit to care for any routine illness or emergencies. (This medical kit box is turned over to the Naval Hospital at Washington for shipment back to Mare Island. Standing orders and the daily routine are outlined in the attached sheet A.)

Routine morning and evening sick call should be made and constipation, headache, and colds prevented as far as it is possible.

Much can be done by encouraging all attendants and medical officers to gain the confidence of the patients, and to keep them occupied with magazines, card games, puzzles and the like. It is encouraging to be able to report that most of the patients react to the trip in a very satisfactory manner adopting a holiday spirit. This holiday excursion attitude was exemplified in the incident of a young man recently paroled from the State feeble-minded institution, who was on the station platform of a small town in West Virginia as our

train came in. During our stop there he spent the time talking to some of the patients and attendants. As we pulled out he expressed the desire that he might accompany our excursion as he was sure that we were all going to have a grand time "up there."

Twenty-four hours before arriving in Washington, D. C., a telegram must be sent to the commanding officer of the naval hospital giving the exact time of arrival, the station, the number of patients and attendants, and the number of stretcher cases.

On arriving in Washington the train is met by ambulances from the naval hospital. Each watch section takes charge of its patients, and now they are marched out of the car, mustered and placed in ambulances for transfer to the Naval Hospital or to St. Elizabeths Hospital. The baggage watch unloads the baggage into trucks. The officer in charge proceeds to the naval hospital to turn over to the commanding officer all orders, records, and valuables belonging to the draft.

The officer in charge's final duty is that of making a written report of the trip to the commanding officer of the naval hospital at Mare Island. This report should include a general résumé of the trip, any difficulties encountered, and any recommendations for improvements which he may consider advisable.

STANDING ORDERS

Watches.—The hospital corpsmen will be divided into four sections. Eight men to a section, one chief pharmacist's mate in charge. The section watches will be of 8 hours.

Heads.—Patients going to a head, or washroom, must be accompanied by a hospital corpsman. The hospital corpsman will remain close to the patient and prevent the patient from closing the door to the head and locking himself inside. If a patient should succeed in locking himself in a toilet or washroom, the medical officer will be immediately notified.

Muster of patients.—Hourly muster of patients will be made by the chief pharmacist's mate of section, and an immediate report made to medical officer of any absentees. The chief pharmacist's mate of the section on duty will also make a sight muster of patients before the train starts after any stop and will make an immediate report of any absentees.

Muster of hospital corpsmen.—Hospital corpsmen will be mustered at 0800 and 2000 daily.

Berthing.—Each patient will be assigned to a berth. Hospital corpsmen on watch will use every effort to keep each patient in his berth at night and in his seat during the day if it can be done without unduly antagonizing or irritating the patients.

Shaving.—Only one patient will be permitted to shave at a time. The senior chief pharmacist's mate will have charge of razors and

blades and will be held responsible for them. All razors will be tagged with the name of their owner and kept in a car not occupied by patients. The senior chief pharmacist's mate will issue the razors one at a time to the Hospital Corps man on duty in the washroom. A Hospital Corps man will be detailed to stand by in each washroom during the hours of shaving. Only one patient will be allowed to shave at a time in each washroom. It will be the duty of the Hospital Corps man to watch the patient closely while he is shaving to make sure that he does not remove the blade from the razor and will take the razor from the patient when he finishes. He will note the presence of the blade in the razor and return the razor to the senior chief pharmacist's mate, who has custody of the razors. He also will note the presence of the blade in the razor before restowing it.

Meals.—The senior chief pharmacist's mate on watch will supervise the feeding of the patients. They will be fed in their own car by messmen detailed for this duty assisted by the pullman waiters. Such patients will not be issued any silverware except spoons. The chief pharmacist's mate of the section on duty will note the number of items of silverware brought into the car, reporting immediately to the senior medical officer any missing items.

Smoking.—Patients will be permitted to smoke from 0800 to 2000 daily. They will not be permitted to smoke in bunks. Patients will not be permitted to have matches.

Windows and doors.—Outside windows are secured so that they cannot be raised more than 5 inches. They will be inspected at least once every hour by the senior Hospital Corps man on watch. Patients will be carefully watched by corpsmen on guard for any evidence of intention to escape through a window. Hospital Corps men will be detailed to guard the end doors of cars occupied by patients. It will be their duty to prevent patients leaving the cars and to keep out of the cars all persons except members of the accompanying party and railroad employees. They will not leave their post until properly relieved.

Leaving train.—No Hospital Corps men or patients will be permitted to leave the train without permission of the medical officer in charge.

Daily routine patients.—All patients will be called at 0700 daily. Breakfast at 0800; lunch at 1200; supper 1730.

Hospital Corps men.—All Hospital Corps men will be called at 0630 daily. Breakfast at 0730; lunch at 1200; and supper at 1730.

Restraint.—The chief pharmacist's mate of the section will report, promptly, to senior medical officer any patient who has become unruly or difficult to handle. He is authorized to use restraint apparatus if this becomes necessary. Patients with suicidal intent

will be kept under close observation by chief pharmacist's mate in charge of each section on duty.

Mail.—The Hospital Corps men will not mail any letters which may be written by the patients, but will accept them unsealed and deliver them to the senior medical officer for censoring.

Liquors.—As many of the patients have a history of alcoholism all Hospital Corps men will exercise special vigilance to make sure no beer or liquors are introduced into the patients' car.

BLOOD TRANSFUSION—A MODIFICATION OF EXISTING DEVICES

By JAMES E. REEVES, Lieutenant, Medical Corps, United States Navy

Before describing a modification of existing types of blood-transfusion devices, a brief summary of the history of blood transfusion might be of interest.

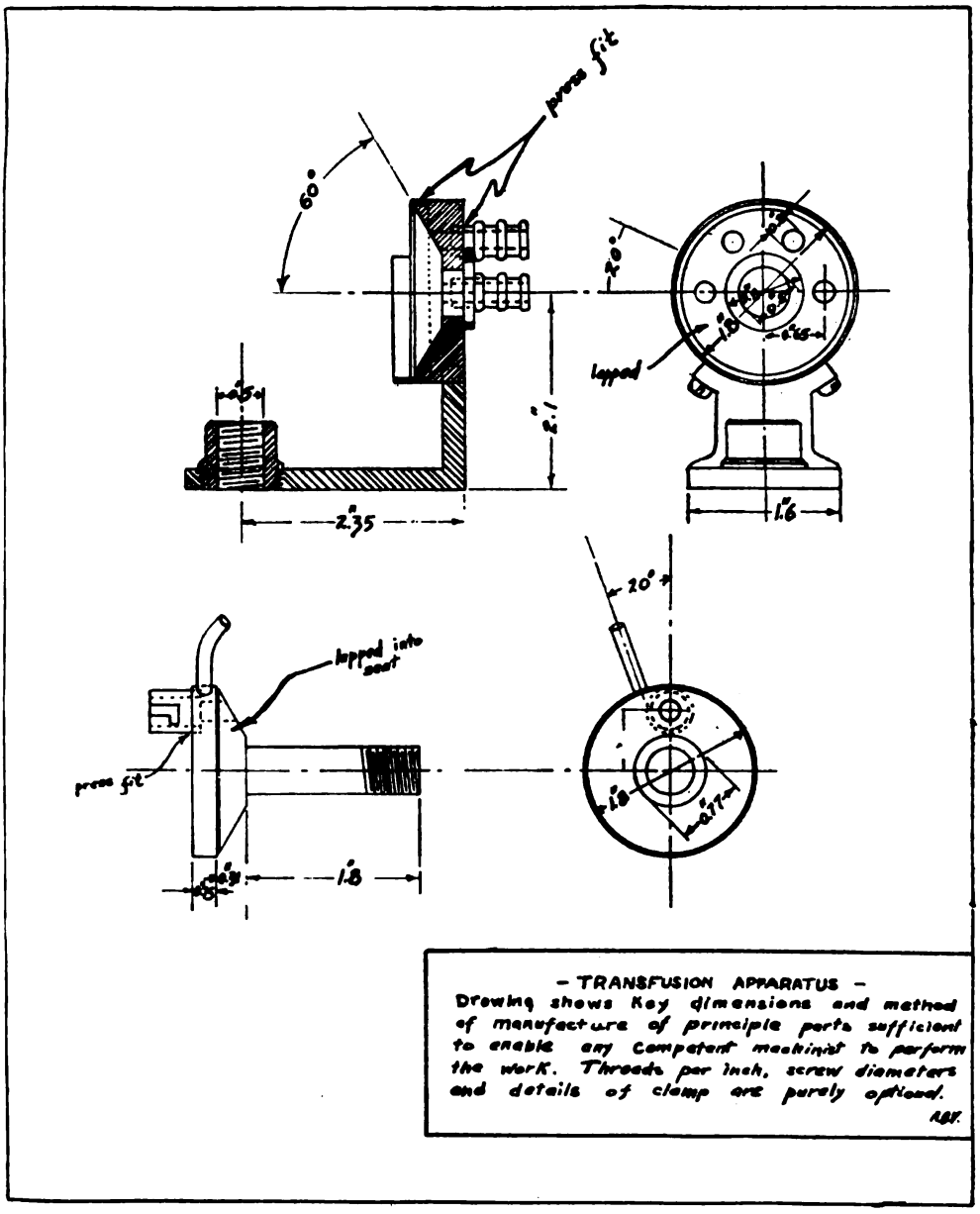
Vague and confusing references to a transfusion said to have been done in the case of Pope Innocent VIII about 1490 occur in almost all works dealing with the history of transfusion but since the practice of drinking the blood of young males was much more prevalent at that time and since no further mention of transfusion occurs for almost 150 years, it is more than likely that an actual transfer of blood did not take place.

The discovery of the circulation of the blood in 1616 by Harvey, really laid the foundation for the work to follow, and it was only 12 years later that Giovanni Colle, professor of medicine at the University of Padua, reported a blood transfusion of lambs' blood, according to Slocker (1). This was soon followed by Potter's suggestion in 1638 to replace the blood of an exsanguinated animal with that of another of the same species (quoted by Landois (2)). An interesting account of this operation is described in his inimitable way by Samuel Pepys (3) under date of 1666. The transfer of lambs' blood to humans was advanced in 1667 and McClure (4), Kerr (5), and Zimmerman and Howell (6), credit one Jean Baptiste Denis with two transfusions of lambs' blood to two patients, both of whom recovered. Like many another development in medicine, interest in transfusions lagged and little of interest occurred until 1796 when Erasmus Darwin advocated transfusion for the malnutrition of esophageal and gastric malignancies.

Probably the first to use human blood in transfusions was James Blundell (7), (8), (9), an English physiologist who began his work in 1824 and was successful in 7 of his 11 cases of postpartum hemorrhage in which transfusion was used. The formidable operation of arteriovenous anastomosis, as well as lack of asepsis and antisepsis, prohibited its use except in grave emergencies. The severe reactions

which must have occurred in many cases, since no matching was done, did little to further interest.

As late as 1874 lambs' blood was seriously offered for transfusion and the German, Haase (10), favorably reported 31 cases of various diseases treated in this manner.



– TRANSFUSION APPARATUS –
Drawing shows Key dimensions and method of manufacture of principle parts sufficient to enable any Competent machinist to perform the work. Threads per inch, screw diameters and details of clamp are purely optional.
ABR.

The discovery in 1900 of the iso-agglutinins by Landsteiner (11), (12), put blood transfusion on a more scientific basis and the workmanlike blood vessel anastomosis of Carrel presented at this time furthered the cause of blood transfusion. When Jansky (13), and Moss (14), published their blood group findings in 1906 and 1910, one of the last real obstacles to transfusion was removed.

From the time of Wren and Denis with their animal experiments, almost every conceivable type of apparatus was suggested and tried

in an effort to convey the blood from donor to recipient, but that of Unger (15), who developed a satisfactory three-way syringe in 1915, was probably the first real transfusion machine.

The device presented claims little real change from existing types but since it has proved satisfactory and was made on board ship by average machinists, it is believed that a few comments may be of interest:

1. In addition to the usual two openings for donor and recipient, an inlet and exit for sterile saline is provided by means of which the system may be cleaned and kept free of clots. Should occasion arise, sterile warmed saline may also be administered to donor or recipient with no outside aid for the operator.

2. The valve openings are 0.22 inch (5.688 mm) in diameter, lessening blood cell trauma and resultant coagulation.

3. The valve face holding the syringe is self-seating and will improve with use and as there is no blind space present, no opportunity is offered for coagulation to occur.

4. The simplicity of the device is such that it can be manufactured aboard ship by a machinist of average ability.

Credit is due to Capt. F. F. Rudder, Medical Department Reserve, United States Army, for permission to use a similar idea and to Lt. A. B. Vosseller, United States Navy, for the mechanical drawing.

REFERENCES

(1) Slocker, E.: Contribution to the History of Blood Transfusion, Siglo Med., 83:75, January 5, 1929.

(2) Laudois, L.: Blood Transfusion, Wiln. Med. Wchnschr., 17:465, 484 and 500, 1867.

(3) Pepys, Samuel: Diary. November 14, 1666.

(4) McClure, R. D.: History of Transfusion of Blood. Report of 150 transfusions. Jour. Mich. M. Sac. 16:178, April 1917.

(5) Kerr, W. M.: A History of Blood Transfusion. U. S. Naval Med. Bull., 16:465, March 1922.

(6) Zimmerman, Leo M., and Howell, K. M.: History of Blood Transfusion. Annals Med. Hist. 4:5, 415, September 1932.

(7) Blundell, J.: The Principles and Practices of Obstetricy. Wash., 1824, p. 263.

(8) Blundell, J.: Researches Physiological and Pathological. London. 1825.

(9) Blundell, J.: Observations on transfusion of blood. Laucet 2:321, 1828.

(10) Haase, O.: Die Lambluttrausfusion beim Meuschen. Leipzig, 1874.

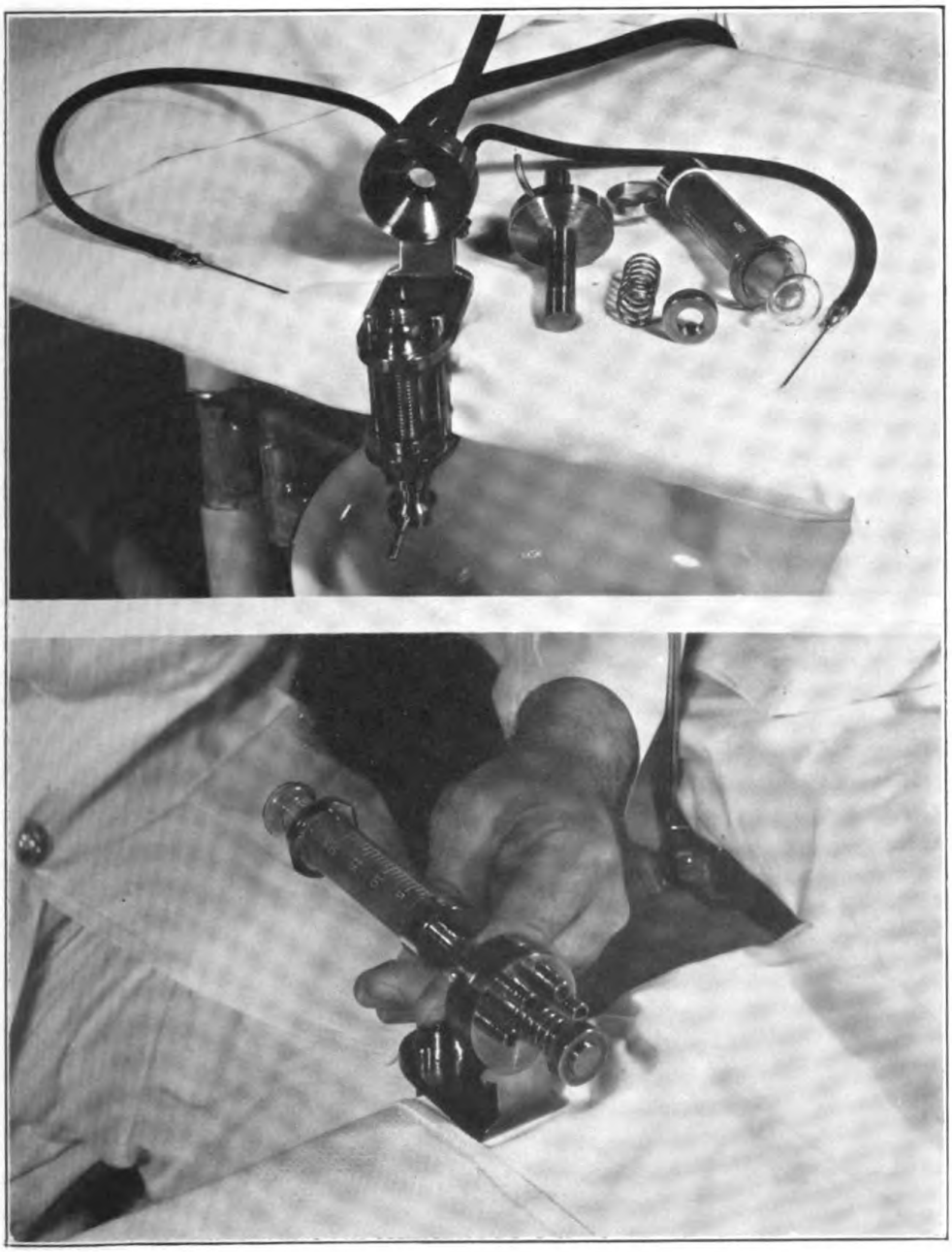
(11) Laudsteiner, C.: Zur Kenntniss der autifermentativen lgtischen, etc. Zeutralbl. f. Bakt. und Parsitiuk., 27:361, 1900.

(12) Laudsteiner, C.: Ueber Agglutinationsercheinungen des normal menschlichen Blutes. Wien. klin. Wchuschr., 1901, p. 1132.

(13) Jausky, J.: Haematologicke, Studie u psychotiku. Sbarn. Klin. 8:85, 1906–7.

(14) Mass, W. L.: Studies in iso-agglutinins. Bull. Johns Hopk. Hosp., 21:63, 1910.

(15) Unger, L. J.: A new method of syringe transfusion. J. A. M. A. 64:582, 1915.



STUDIES OF ACTIVE PNEUMOCOCCUS IMMUNITY—IV. THE DURATION OF TYPES I AND II PNEUMOCOCCUS IMMUNITY[5]

By DAVID FERGUSON, Commander, Medical Corps, United States Navy

In previously reported studies[6] active immunity of high titre was found to follow the subcutaneous injection of a specially prepared and tested penumococcus vaccine. This immunity was measured at intervals during 1 year in three men and was found to remain at satisfactorily high levels. The studies were made only with type I pneumococcus.

The Bureau of Medicine and Surgery then authorized this study which reports:

(1) The measurement of the type I immunity titre at intervals during a further period of 10 months—a total of 22 months in three men.

(2) The measurement of type I immunity in seven additional men at intervals during a period of 1 year.

(3) The measurement of immunity against type II pneumococcus in nine men at intervals during a period of 1 year.

METHODS

Blood was drawn by venupuncture from the volunteers before, and at intervals after, the injection of vaccine.

The serum was separated.

In the first group of three men, 0.2 cubic centimeters of serum was injected into the peritoneal cavity of the experimental animal and followed immediately by the injection of the prescribed number of lethal doses of type I pneumococcus.

Certain irregular results were obtained which were thought due in some instances to the serum and lethal doses being absorbed separately; in other instances the separate components may have come together in the peritoneal cavity and have been absorbed after some neutralization occurred.

Accordingly, in the second and third series, the prescribed number of lethal doses of pneumococcus were mixed with 0.2 cubic centimeters of blood serum and the mixture was injected intraperitoneally into the experimental animals.

Two or three animals were used for each dilution of lethal doses for every sera. Irregular tests which could not be evaluated were repeated as the supply of animals and blood serum permitted.

White rats and mice were the experimental animals used.

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[5] Received for publication Oct. 1, 1935.

[6] U. S. Naval Medical Bulletins, 30, 409, 1932; 32, 155, 1934; and 33, 219, 1935.

THE VACCINE

As described in previous reports, the vaccine was obtained from a culture from the heart blood of a moribund mouse which had been inoculated intraperitoneally with from 3 to 10 pneumococci from a highly virulent culture.

The heart blood was seeded in a broth culture enriched with 2 percent of washed rabbit blood cells.

In previously reported studies a beef broth was used in which the sugar, chlorides, total proteids, urea nitrogen and nonprotein nitrogen content and the pH approximated that of the human blood serum. The rationale was to provide for the growth and reproduction of the organisms an environment as similar as possible to that of the living animal.

In the present study the broth used was made by the method of Douglas and Hartley. The day before using, it was enriched with 2 percent of washed rabbit blood cells.

The organisms were recovered by centrifugalization; killed with acetone; dried in vacuum; and, immediately preceding injection, suspended in saline.

The vaccine before being used was tested in experimental animals for antigenic power, and vaccines that were found deficient in antigenic power were discarded.

DESCRIPTION OF TABLES AND CHARTS

Table 1 is a record of the protection power of 0.2 cubic centimeters of blood serum of three men against type I pneumococcus. The measurements were made before and at intervals after immunization over a period of 22 months.

Chart 1 is a graphic record of the immunity titre throughout the 22 months.

A satisfactory augmentation of the protection power of the serum was found to occur and to persist.

The number of type I pneumococci injected in the vaccine is shown.

Table 2 records the measurements of the protection power of the blood serum of seven men before and at intervals after immunization with type I pneumococcus over a period of 12 months.

Chart 2 is a graphic record of the immunity titre.

The smaller dosage of type I pneumococci is shown.

Table 3 records the measurements of the protection power of the blood serum of nine men against type II pneumococcus before, and at intervals after immunization with type II pneumococcus vaccine over a period of 12 months.

Chart 3 is a graphic record of the curve of the immunity titre.

It will be noted in chart 2 that there is an irregular curve in the protection power of the sera of volunteers nos. 4, 6, and 10 and

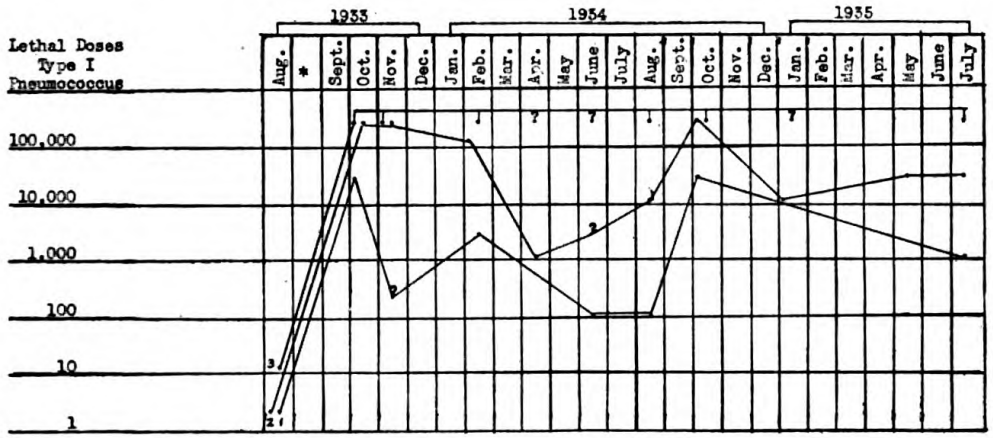


CHART 1.—The curve of the immunity titre of the blood serum of three men (nos. 1, 2, and 3) before and after immunization with type I pneumococcus vaccine.

*Immunization by 4 and 5 subcutaneous injections of vaccine between September 23, 1933, and October 13, 1933.

NOTES.—(1) A question mark at a point in the graph shows an experimental result which was indefinite but justified an estimation of the immunity titre.

(2) Volunteer no. 1 was ordered to sea duty in September 1934 and his blood serum could not be obtained regularly after that date.

probably in volunteers nos. 5 and 7. This is due to the fact that in the experiments of January 15, 1935, and May 22, 1935, the esti-

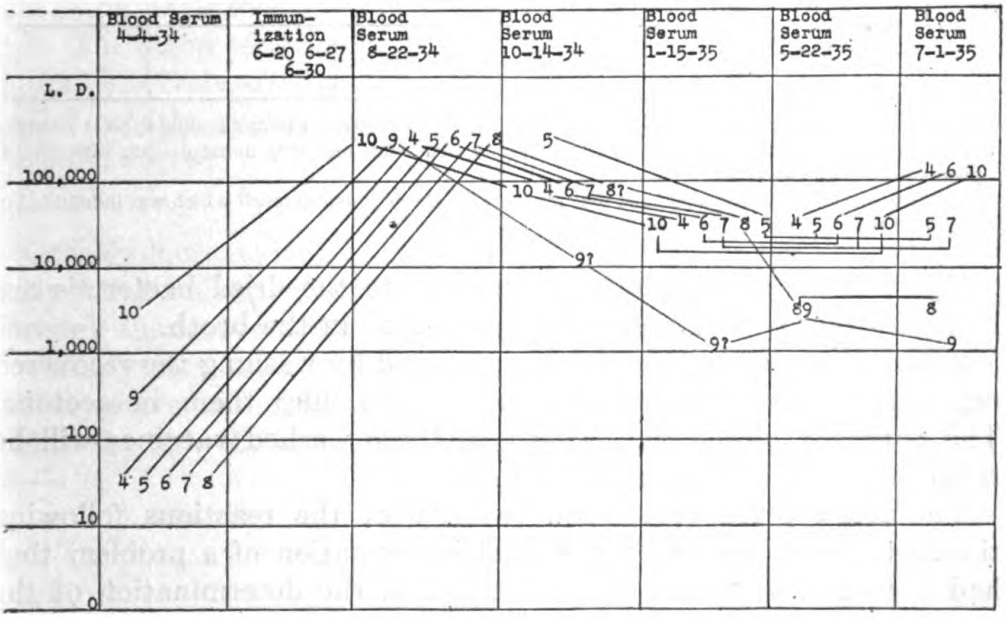


CHART 2.—Type I immunization. Showing the curve of the protection power against type I pneumococcus of 0.2 cubic centimeter of blood serum of seven men before and after immunization with type I pneumococcus vaccine.

NOTE.—A question mark at a point in the graph shows an experimental result which was indefinite but justified an estimation of the immunity titre.

mated lethal doses were not attained; i. e., the decimal dilutions gave 1,000 and 10,000 lethal doses instead of 10,000 and 100,000

on those dates, and there were insufficient experimental animals to repeat the tests.

REACTIONS

In the first group of three men immunized in 1933, there were no reactions severe enough to incapacitate.

In the second group of eight men immunized in 1934, local and general reactions necessitating absence from duty occurred in four men following the injection of approximately 50 million each of types I, II, and III pneumococci, but no reactions followed the second dose of 50 thousand of each type or the third dose of 25 million of each type.

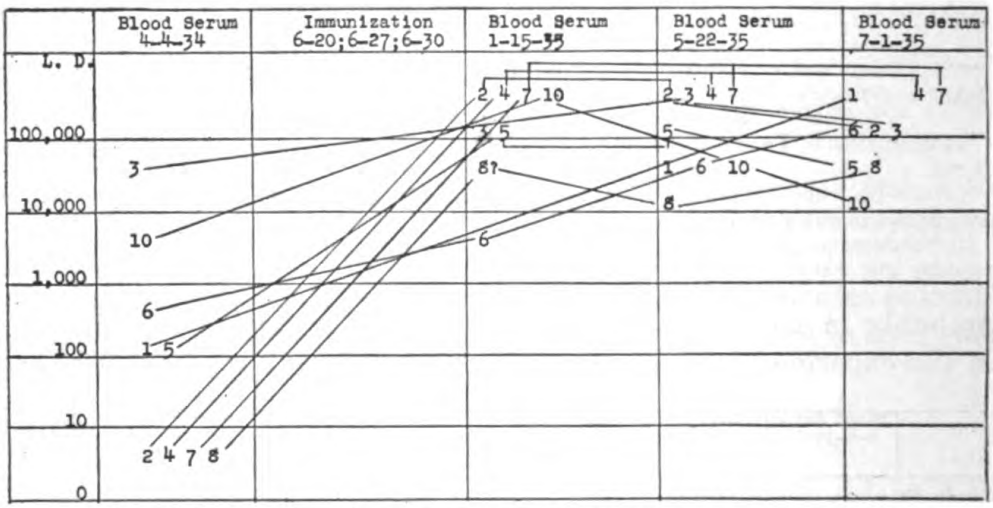


CHART 3.—Type II immunization. Showing the curve of the protection power against type II pneumococcus of 0.2 cubic centimeter of blood serum of nine men before and after immunization with type II pneumococcus vaccine.

NOTE.—A question mark at a point in the graph shows an experimental result which was indefinite but justified an estimation of the immunity titre.

The reactions undoubtedly were due to the dried bacteria's not being entirely free of the proteid contained in the broth.

Subsequent vaccines have been prepared by washing the recovered organisms once in normal saline before killing them in acetone. The antigenic power and toxicity of these washed vaccines will be reported in a later communication.

The relative frequency and severity of the reactions following the first dose permitted the partial investigation of a problem that had been reserved for a later study, i. e. the determination of the minimal optimal dosage of the vaccine. The second dose, a desensitizing dose of only 50 thousand each of types I, II, and III, was given 7 days after the first dose to 9 of the 10 volunteers; the tenth volunteer (no. 3) receiving the scheduled dose of 200 million of type II and III cells. The third dose of 25 million of types I, II, and III were then administered to all except volunteer no. 3 who received

250 million each of type II and III cells and suffered a severe nitritoid reaction.

A preliminary titration of the blood serum of the volunteers showed a satisfactory immunity response, so the observations were continued and form the material for this report.

This series of measurements of the immunity response to pneumococcus vaccine is very small.

The question naturally arises whether the series is too small to draw definite conclusions.

Statisticians agree that the proper size of a series necessary to answer a question depends not upon the number constituting the series, but upon the uniformity of the results obtained.

Whether or not the augmented resistance of the blood serum would prevent the development of pneumonia of homologous types could only be shown by the vaccination of a large group of men in which the average incidence of pneumonia is known.

SUMMARY

1. The blood serum of three men was examined over a period of 22 months, and of seven men over a period of 12 months before and after immunization with type I pneumococcus and a high titre of protection was found to develop and to persist in slowly diminishing and variable degree.

2. The blood serum of nine men was measured for protection before and at intervals after immunization with type II vaccine and found to be greatly augmented and to persist in slowly diminishing and variable degree.

CONCLUSIONS

Greatly increased resistance against type I pneumococcus followed active immunization and persisted for 22 months. It continues through the last recorded measurement.

Greatly increased resistance against type II pneumococcus followed active immunization and persisted for 12 months. It continues through the last recorded measurement.

TABLE 1

Before immunization			Immunization # type I pneumococci in millions					After immunization											
Date	Aug. 23, 1933		Aug. 23	Aug. 29	Sept. 3	Sept. 7	Sept. 13	Oct. 11, 1933		Nov. 22, 1933		Feb. 6, 1934		Apr. 4, 1934		June 5, 1934		Aug. 22, 1934	
Volunteer	L. D.	Result						L. D.	Result	L. D.	Result	L. D.	Result	L. D.	Result	L. D.	Result	L. D.	Result
1	10	2D	0	50	200	100	200	1,000	2S	1,000	2D	1,000	2S			100	1S 1D	100	1S 1D
	100	2D						10,000	2S	10,000	2D	10,000	2D			1,000	2D	1,000	2D
								100,000	2D			100,000	2S			10,000	2D	10,000	2D
2	10	2D	0	50	200	100	200	10,000	2S	10,000	2S	10,000	2S	100	2S	100	2S	1,000	2S
	100	2D						100,000	2S	100,000	2S	100,000	1S 1D	1,000	1S 1D	1,000	1S 1D	10,000	1S 1D
3	10	1S 1D	50	200	200	100	200	10,000	2S	10,000	2S	10,000	2S	1,000	2S	1,000	2S	10,000	2S
	100	2D						100,000	2S	100,000	2S	100,000	2S	10,000	2S	10,000	2S	100,000	2S
Date												Oct. 14, 1934		Jan. 15, 1935		May 22, 1935		July 1, 1935	
Volunteer												L. D.	Result	L. D.	Result	L. D.	Result	L. D.	Result
1												1,000	2S					1,000	2S 1D
												10,000	2S					10,000	3D
												100,000	2D						
2												10,000	2S	1,000	2S	1,000	3S	10,000	3S
												100,000	2S	10,000	1S 1D	10,000	3S	100,000	3D
3												10,000	2S	1,000	2S	10,000	3S	10,000	3S
												100,000	2S	10,000	2S	100,000	3S	100,000	3S

L. D.=# Lethal doses of type I pneumococcus injected immediately after the intraperitoneal injection of 0.2 cc of blood serum; the latter collected on the date shown. D and S preceded by a numeral show the number of animals dying or surviving the given number of lethal doses of type I pneumococcus.

TABLE 2

Before immunization			Immunization # type I pneumococci			After immunization									
Date	Apr. 4, 1934		June 20, 1934	June 27, 1934	June 30, 1934	Aug. 22, 1934		Oct. 14, 1934		Jan. 15, 1935		May 22, 1935		July 1, 1935	
Volunteer	L. D.	Result	Millions	Thousands	Millions	L. D.	Result	L. D.	Result	L. D.	Result	L. D.	Result	L. D.	Result
4	100	2D	60	50	25	100,000	2S	10,000	2S	1,000	{1D 1S}	1,000	3S	10,000	2S
								100,000	{1D 1S}	10,000	2S	10,000	3S	100,000	{1D 2S
5	100	2D	50	50	25	100,000	2S	10,000	2S	1,000	2S	1,000	3S	10,000	3S
								100,000	2S	10,000	2S	10,000	3S	100,000	{2D 1S
6	100	2D	60	50	25	100,000	2S	100,000	{1S 1D}	1,000	2S	1,000	3S	10,000	3S
								1,000,000	2S	10,000	2S	10,000	3S	100,000	{1D 2S
7	100	2D	50	50	25	100,000	2S	100,000	{1S 1D}	1,000	2S	1,000	3S	10,000	3S
								1,000,000	2D	10,000	2S	10,000	3S	100,000	3D
8	100	2D	45	50	25	100,000	2S	----------	----	100	2S	1,000	3S	1,000	3S
										1,000	2S				
										10,000	2S	10,000	{1S 2D}	10,000	{2D 1S
10	100	2S	50	50	25	100,000	2S	100,000	{1D 1S}	1,000	2S	1,000	3S	10,000	3S
	1,000	2S	------	-------	------	-------	----	1,000,000	2S	10,000	2S	10,000	3S	100,000	{1D 2S
	10,000	2D	------	-------	------	-------	----	----------	----	-------	----	-------	----	-------	----
9	100	2S	50	50	25	100,000	2S	100,000	2D	100	2S	1,000	3S	1,000	{1D 2S
										1,000	{1D 1S				
	1,000	2D	------	-------	------	-------	----	1,000,000	2D	1,000	{1D 1S}	10,000	{1S 2D}	10,000	{2D 1S
	10,000	2D	------	-------	------	-------	----	----------	----	10,000	{1D 1S}	------	----	-------	----

L. D.= #Lethal doses of type I pneumococcus mixed with 0.2 cc of blood serum, collected on date shown. S and D preceded by a numeral show the number of animals surviving or dying from the given number of lethal doses.

TABLE 3

Before immunization			Immunization # type II pneumococci			After immunization—									
Date	Apr. 4, 1934		June 20	June 27	June 30	Aug. 22		Oct. 14		Jan. 15, 1935		May 22 1935		July 1, 1935	
Sera no.	L. D.	Result	Millions	Thousands	Millions	L. D.	Result	L. D.	Result	L. D.	Result	L. D.	Result	L. D.	Result
1	10	2S	80	50	25	-------	----	10,000	1D 2S	------	----	1,000	2S	100,000	3S
	100	1D 1S	-----	-------	------	-------	----	100,000	1D 1S	------	----	10,000	2S	1,000,000	3D
2	10	2D	70	50	25	-------	----	-------	----	1,000	2S	10,000	2S	100,000	1D 2S
										100,000	2S	100,000	2S	1,000,000	3D
3	10,000	1D 3S	80	200,000	250	-------	----	-------	----	1,000	2S	10,000	2S	100,000	1D 2S
	100,000	2D	------	-------	------	-------	----	-------	----	100,000	1D 1S	100,000	2S	1,000,000	3D
4	10	2D	80	50	25	1,000	2S	-------	----	1,000	2S	10,000	2S	100,000	3S
						100,000	2S	-------	----	100,000	2S	100,000	2S	1,000,000	1S 2D
5	10	2S	70	50	25	1,000	1D 1S	10,000	3S	1,000	1D 1S	10,000	2S	10,000	3S
	100	1D 1S	-----	-------	------	100,000	2S	-------	----	100,000	1D 1S	100,000	1D 1S	100,000	3D
6	100	2S	80	50	25	1,000	2S	1,000	2S	1,000	2S	1,000	2S	10,000	3S
	1,000	2D	------	-------	------	100,000	2S	100,000	2D	10,000	2D 1S	10,000	2S	100,000	1D 2S
										100,000	2D	-------	----	1,000,000	3D
7	10	2D	70	50	25	-------	----	-------	----	1,000	1D 1S	10,000	2S	100,000	3S
										100,000	2S	100,000	2S	1,000,000	3D
8	10	2D	60	50	25	-------	----	-------	----	1,000	1D 1S	1,000	2S	10,000	3S
										100,000	1D 1S	10,000	1D 1S	100,000	1S 2D
10	1,000	5S	70	50	25	1,000	2S	-------	----	1,000	2S	1,000	2S	10,000	2S 2D
	10,000	1S 3D	-----	-------	------	100,000	2D	-------	----	100,000	2S	10,000	2S	100,000	1S 2D

L. D. = # Lethal doses of type II pneumococcus mixed with 0.2 cc blood serum, collected on date shown. S and D preceded by a numeral show the number of animals surviving or dying from the given number of lethal doses.

BREATHING RESISTANCE OF THE NEW SUBMARINE ESCAPE APPARATUS COMPARED WITH THAT OF PREVIOUS MODELS [7]

By R. A. HANSEN, Lieutenant, A. R. BEHNKE, Lieutenant, Medical Corps, and C. W. SHILLING, Lieutenant, Medical Corps, United States Navy

Resistance to breathing has been considered one of the chief difficulties experienced by men learning to use the submarine escape appliance. Recently an improved model of the apparatus has been developed which it is claimed offers considerably less resistance than previous types issued to the service. Changes incorporated in the latest model are:

(*a*) The mouthpiece assembly has been redesigned with a view toward increasing the area of air passages and eliminating the number and degree of changes of direction of air flow.

(*b*) The mouthpiece is recessed into the top of the bag so that the bag is worn higher on the body.

[7] Received for publication Sept. 30, 1935.

(*c*) The bag is shortened vertically, the capacity being retained by making it wider and with deeper bellows.

(*d*) The flutter valve fitting is larger in sectional area and is attached to the bag so that its opening is even with the bottom edge of the bag.

The last three modifications have resulted in raising the flutter valve to a distance of only 11 inches below the mouthpiece when the bag is fully inflated compared with 15 inches in the old "lung." This reduces the hydrostatic pressure which must be overcome by the wearer's lungs before the valve opens for the release of gas. The average pressure in the system is likewise reduced.

Measurements of breathing resistance offered by the old model were made in 1932 at the submarine escape training tank at the Submarine Base, New London, Conn. Resistances encountered in the use of the new type "lung" were measured recently at the Experimental Diving Unit.

Method.—A mercury sphygmomanometer, connected by a fitting to the metal mouthpiece of the "lung", was installed in the bell or tank. The subject wearing the "lung" adjusted his position to maintain the mouthpiece at the surface of the water. The manometer indicated, therefore, the pressure existing momentarily in the gas system consisting of the bag and the wearer's lungs relative to the pressure existing at the level of the mouthpiece. Minimum and maximum readings were recorded under the following conditions:

(*a*) Preliminary runs at the surface with oxygen being supplied slowly at the charging valve to simulate the expansion of gas during an ascent.

(*b*) A series of ascents from 90 feet at the rate of 48 feet per minute with normal quiet breathing.

(*c*) The same as (*b*) except with forced or irregular breathing.

(*d*) Rapid ascent at the rate of 90 feet per minute.

Results.—During the control runs at the surface (see table 1) the average pressure on inspiration was 23 millimeters of mercury with the old "lung", that of the new one 13 millimeters. The averages on expiration were 30 and 24, respectively, the difference being approximately equal to the difference between the depth of submergence of the flutter valves.

On the normal ascents (see table 2) the averages during inspiration were 23 and 8 millimeters of mercury; during expiration pressures were 34 and 24 millimeters. The average pressure in the bag of the old "lung" was 28.5, in that of the new one only 16 millimeters.

With forced breathing (see table 3) the old "lung" showed high resistances, reaching in some cases 66 millimeters pressure, due to the restricted area of the flutter valve. The new "lung" showed a maximum reading of only 32 millimeters. The averages during expiration were 43 and 25 millimeters respectively. The average

pressure in the bag of the old "lung" was 33 millimeters, compared to an average of 14.5 millimeters in the new "lung."

On rapid ascent with normal breathing (see table 4) pressures reached a maximum of 42 millimeters on expiration and the average was 36 in the old "lung." The new one gave a maximum of 28 and an average of 25 millimeters.

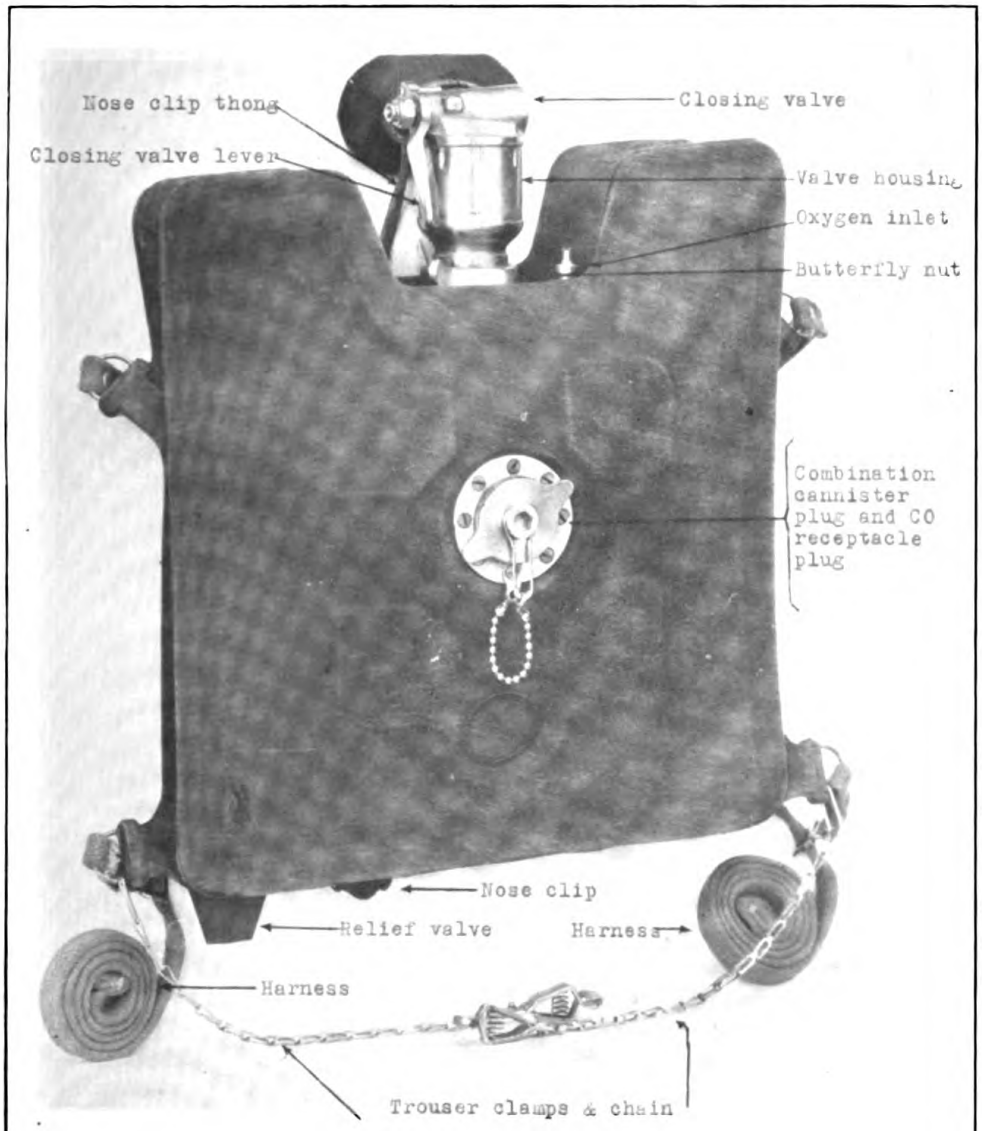
Interpretation of results.—It is not maintained that the manometer readings indicated the true resistance to breathing which involves physiological factors difficult to evaluate. The actual resistance is the increase of muscular effort necessary to inhale or exhale, and this is affected to a varying degree by the hydrostatic pressure on the body. At the end of exhalation, the bag is full and the lungs deflated. The hydrostatic pressure has the least effect and the resistance to exhalation is a maximum. At the end of inhalation, the pressure in the bag is reduced while the respiratory mechanism is opposed by the maximum hydrostatic pressure. The manometer readings do indicate the resistance to be overcome by the jaw, lip, and cheek muscles. These stresses are important in training for their effect upon the individual's mental attitude and may lead to casualties. The manometer readings are accurate for comparing the resistances offered by different models of the apparatus, for in this study the uncertain physiological factors balance.

SUMMARY

Pressures in the gas systems of the old and new types of submarine escape apparatus were measured by a mercury sphygmomanometer in order to ascertain their relative resistances to breathing. The changes in construction of the new type have resulted in a marked reduction in pressures encountered in using the device. It is impossible to build up, even under adverse conditions, the high resistances sometimes developed in the previous model.

TABLE 1.—(*a*) *At surface; normal breathing*

Subject	Old lung						Subject	New lung					
	Inspiration			Expiration				Inspiration			Expiration		
	Minimum	Maximum	Average	Minimum	Maximum	Average		Minimum	Maximum	Average	Minimum	Maximum	Average
A	22	22	22	30	32	30	J	10	12	11	24	26	24
B	24	26	25	28	34	30	K	4	12	8	22	24	24
E	24	26	25	34	34	34	L	12	18	15	23	24	24
F	20	22	21	26	28	27	M	16	19	17	23	26	24
G	18	18	18	26	32	29	N	13	15	13	24	26	24
H	24	26	25	28	33	31	O	13	15	14	23	24	24
Average	------	------	23	------	------	30	Average	------	------	13	------	------	24



Front view - Improved Type Submarine Escape Appliance "Lung".

TABLE 2.—(*b*) *Ascent from 90 feet at 48 feet per minute—normal breathing*

Subject	Old lung						Subject	New lung					
	Inspiration			Expiration				Inspiration			Expiration		
	Minimum	Maximum	Average	Minimum	Maximum	Average		Minimum	Maximum	Average	Minimum	Maximum	Average
A	24	28	27	28	40	36	J	2	10	8	22	24	24
B	22	26	24	28	36	32	K	0	8	6	22	24	23
C	20	24	22	28	36	34	L	10	14	12	22	25	24
E	18	26	23	26	36	32	M	2	10	6	26	30	28
F	22	28	25	32	38	36	N	6	8	7	20	28	24
G	18	22	20	30	38	36	O	10	12	11	22	24	23
H	20	28	25	24	36	31							
P	20	21	20	33	36	34							
Average			23			34	Average			8			24

TABLE 3.—(*c*) *Ascent from 90 feet at 45 feet per minute—forced breathing*

Subject	Old lung						Subject	New lung					
	Inspiration			Expiration				Inspiration			Expiration		
	Minimum	Maximum	Average	Minimum	Maximum	Average		Minimum	Maximum	Average	Minimum	Maximum	Average
A	24	30	27	42	66	53	J	−4	7	0	22	27	26
B	20	26	25	28	44	39	L	0	8	4	22	29	25
E	10	20	15	34	42	38	L	6	12	8	24	26	25
A	26	26	26	34	66	54	M	0	6	3	24	26	25
B	22	26	24	32	48	37	N	4	12	10	20	26	24
P	10	12	11	34	36	35	O	−8	4	0	26	32	28
Average			21			43	Average			4			25

TABLE 4.—(*d*) *Ascent from 90 feet at 90 feet per minute*

Subject	Old lung						Subject	New lung					
	Inspiration			Expiration				Inspiration			Expiration		
	Minimum	Maximum	Average	Minimum	Maximum	Average		Minimum	Maximum	Average	Minimum	Maximum	Average
							K	2	10	6	22	26	24
							L	10	14	13	22	24	23
							M	8	11	9	24	25	25
P	16	21	19	32	34	33	N	7	9	8	22	26	25
P	10	14	13	36	42	39	O	4	8	7	26	28	27
Average			16			36	Average			9			25

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SOME ASPECTS OF THE TREATMENT OF NEUROSYPHILIS IN THE NAVY

By LESTER E. McDONALD, Lieutenant, junior grade, Medical Corps, United States Navy

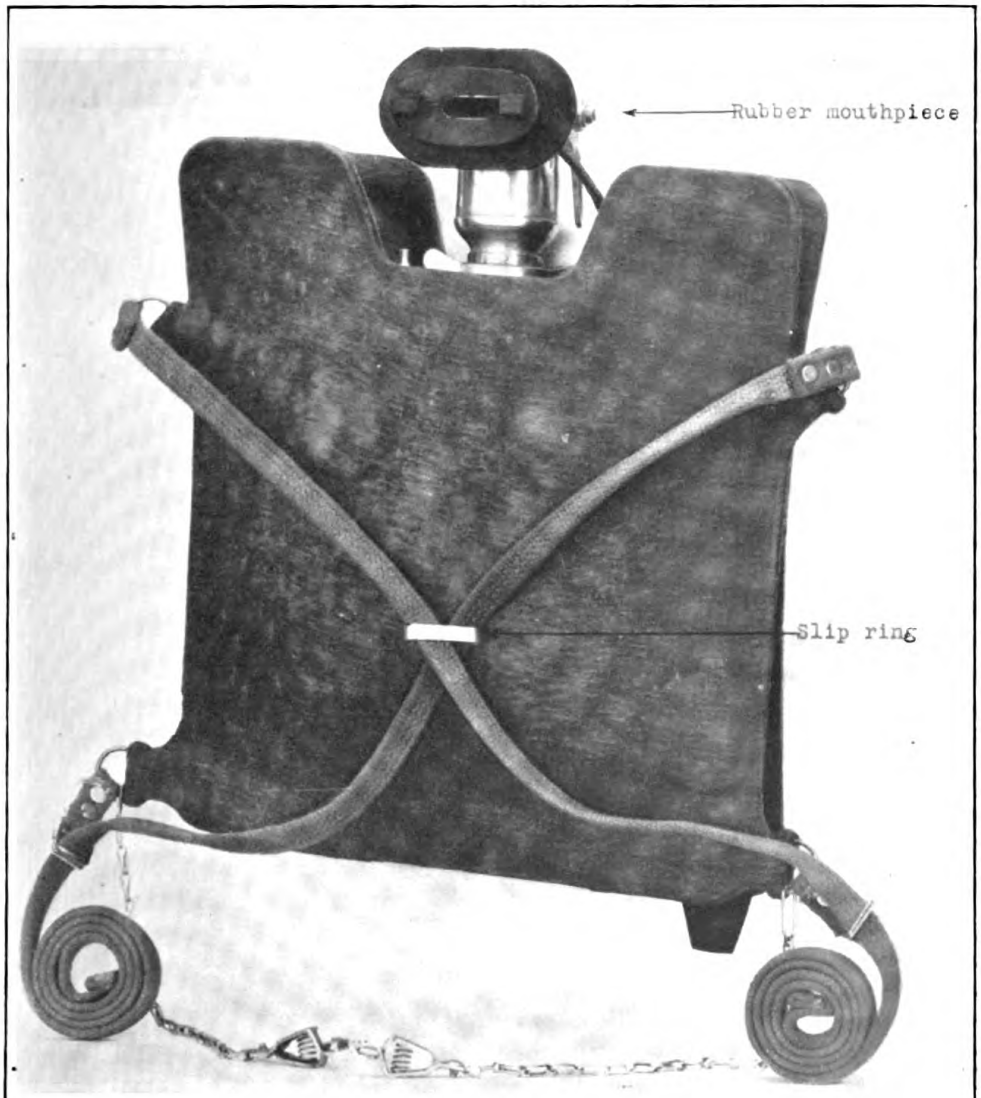
Up to 1900 only those forms of syphilis of the nervous system were known which were manifested by clinical symptoms. At present biological examination of the spinal fluid makes possible the detection of early nervous system involvement before clinical manifestations are developed. Harold C. Torbert (1) has shown the safety of spinal puncture for ambulatory patients, and a comparison between the two groups of those hospitalized for 24 hours, and those leaving the hospital 2 hours after the operation, has been given; there was no reduction in the number of reactions, but an average duration of the symptoms materially shortened in those hospitalized.

All spinal fluids have been examined in the laboratories of one of the following naval hospitals: Bremerton, Wash., San Diego, Calif., Mare Island, Calif., and also aboard the U. S. S. *Relief*.

What is to be considered a positive spinal fluid in neurosyphilis? 1. *Hypertension—No.*—T. A. Hewer (2) reviews 313 cases of syphilis, in which routine examinations of the spinal fluids were made. Hypertension was the least common abnormality and there was no apparent relation between the degrees of pathological change and the pressure of the fluid. These figures contrast strikingly with those of other authors, and are in entire disagreement with the constantly stated textbook opinion that hypertension is one of the earliest signs of syphilitic involvement of the central nervous system. In our group only one patient had a marked increase in pressure, as was shown by the rapidity of flow from an 18-gage spinal puncture needle. An analysis of the fluid was normal (case 18, chart A).

2. *Quantitative determination of the total protein, plus the globulin fraction.—Yes.*—Leo Spiegel (3) in a study of 310 spinal fluids has shown that the protein usually diminishes when treatment and drainage are carried out, and when there is a distinct increase in the globulin, with a total protein approaching 18 milograms per 100 cubic centimeters, it is the first evidence of a pathological spinal fluid in neurosyphilis. He also states that total protein offers a check on the Kahn test during treatment. In our analysis we asked for the globulin fraction and not the total protein, because of the difficulty of the test without the use of the scopometer as recommended by Exton (4).

3. *Cell count.—Yes.*—In the study of a case of general paralysis in which a lumbar puncture was performed every 15 days for a year, E. DeMasary (5) has shown that the number of lymphocytes varied from 0 to 150 per cubic millimeter, and these variations did not correspond with the clinical changes in the disease. In case 5, chart A, the variation in lymphocytes in three spinal fluid analyses varied from 5 to 490. In the last fluid examination 490 lymphocytes per



Back view - Improved Type Submarine Escape Appliance "Lung".

cubic millimeter were found while the colloidal gold and globulin estimation showed marked improvement. We consider that we have in no case attached too much significance to the cell count alone.

4. *Colloidal gold.—Yes.*—Spiegel (3) has shown that there is no definite agreement between the colloidal gold curve and the Kahn reaction, or with the total protein or globulin, although a large increase of the globulin forecasts a precipitation of the gold solution in the first or paretic zone. In case 9, chart A, with the presence of a globulin 2 plus, Kahn 3 plus, the gold curve remained 0000000000. This same case with treatment, developed a normal spinal fluid; which finding is unusual, and there is some possibility of a technical error in the analyses.

5. *Kahn test, spinal fluid—Yes.*

6. *Negative spinal fluid—Yes.*—Solomon (6) has shown that it is not uncommon to find active neurosyphilis in the presence of a negative spinal fluid. This is most apt to occur in vascular types of neurosyphilis, tabes dorsalis, cerebral gumma, and syphilitic psychosis. It is not common, however, to have a change from a normal spinal fluid after the first negative to a later positive fluid. Case 50, chart A, suggests such findings as a negative spinal fluid in the presence of active neurosyphilis.

7. *Blood serum Kahn—No relationship.*—Out of the 116 spinal fluids studied, 40 were positive, and only 33 had a positive blood serum Kahn.

In St. Elizabeths Hospital 25 to 30 percent of the blood tests are negative in general paresis. The spinal fluid is positive in 95 to 100 percent. In early asymptomatic neurosyphilis the blood serum Kahn is perhaps of little significance.

Proper deductions must be drawn as to significance and correlation of positive findings in spinal fluid analyses.

Asymptomatic Neurosyphilis

Recognition of asymptomatic neurosyphilis is made by the finding of a positive spinal fluid in a case in which there are no symptoms or clinical findings of neurosyphilis. A few cases in this series have physical signs, but no subjective symptoms of neurosyphilis. Epstein, Graves, and Sherman (7) have shown that:

1. In 67.6 percent of 34 patients studied with relapsing blood serology the spinal fluid was positive.

2. In 57.4 percent of Kahn fast patients the spinal fluid was positive.

3. That negative blood serology is not uncommon in patients with positive spinal fluids.

4. That active neurosyphilis may exist in a patient with negative spinal fluid.

Since there are so many cases of syphilis in the United States Navy that have serological relapses or recurrence, the findings of these authors are of interest. Serological relapse indicates renewed activity of the disease in the body. The infection lies dormant for a period of time, then there is a recrudescence, and the localized lesions may act as feeders for tissue far removed from the original focus. If, by study of the spinal fluids of members of this group who have had a serological relapse we can determine those who have had an invasion and colonization in the brain tissue, as shown by the spinal fluid tests we can then approximate the total number of cases of asymptomatic neurosyphilis in the United States Navy. This group of patients may be compared with a similar group in civil life. We can also show the necessity of a study of the spinal fluid in men desiring reenlistment. Section 19, page 1507, Manual of the Medical Department, states: "The following conditions are cause for rejection—syphilis in any stage, * * *." What part should blood Kahns and spinal fluid findings play in qualifications for enlistment or reenlistment. On board the U. S. S. *Lexington* there are 11.3 percent of men aboard with a history of syphilis. Out of this 160 cases, 94 spinal fluids have been analyzed, and 30 were positive, or 31.9 percent. We estimate that there are in the United States Navy of 80,000 men, 11.3 percent or 8,800 with a history of syphilis and 31.9 percent or 2,552 with neurosyphilis. What should be the fate of the naval career of this group of men?

Treatment of Neurosyphilis

It certainly is true that nearly all cases of neurosyphilis in the early stages seen in the United States Navy may be treated on board ship with excellent results.

Our general plan of treatment has consisted of the use of large doses of Tryparsamide and Iodobismitol, given in weekly intravenous and intramuscular injections.

Toxicity from Tryparsamide may readily be detected by careful clinical observation and by the use of the perimeter. It causes no immediate reaction following injection as does neosalvarsan. One case aboard this vessel has recently received 30 intravenous injections of Tryparsamide of 3 grams each, and 20 intramuscular injections of Iodobismitol without showing any signs of toxicity and with marked improvement of the spinal fluid findings. Visual fields on the perimeter are required at the beginning of each course of Tryparsamide and following each 5 injections. The fields of vision in most instances have been contracted at the beginning of the treatment and in case 5, chart A, there has been widening of the fields following treatment. There are many causes of contraction

of the field of vision, viz, alcohol, tobacco, etc. It is not uncommon to find contracted fields of vision in men not suffering from a leutic infection. Damage to the optic nerve from arsenic first manifests itself with changes in the visual fields. We have recently used routinely a new form of bismuth called by trade name, "Iodobismitol." This drug was first introduced by P. J. Hanzlik (8) in 1932. It is a soluble bismuth in anion form which possesses certain advantages over bismuth in cation form, in that there is penetration of the brain tissue. It is the only form of bismuth used on the U. S. S. *Lexington* at the present time. Questionnaires answered by the patients indicate a preference for this drug over the Navy supply table "Bismitol." We have used it routinely with the hope of later determining if it will prevent neurosyphilis. Given in 2 cubic centimeter injections, twice a week, during the past 6 months we have given over 2,000 injections. It will require time to prove its efficacy in preventing neurosyphilis. However, it has proven its value in the treatment of early neurosyphilis.

Mercury in any form has not been used during the past 6 months as an adjunct in treatment. It was discontinued in favor of Iodobismitol.

Cases Showing Improvement in the Spinal Fluid Following Treatment

Case 5, chart A, is unusual in that he was transferred to the United States Naval Hospital, Mare Island, Calif., with an undetermined psychosis. The etiological agent was found to be a luetic infection which he acquired 10 years before. Following treatment with Tryparsamide, malaria, and bismuth over a period of 7 months, he was returned to duty. Since return he has carried on his work efficiently and his behavior has been excellent. This case is the only one aboard who has had malaria therapy and is also the only case who developed a psychosis from a luetic infection. Cases 6 and 7, chart A, and case 17, chart C, showed a reduction of a positive spinal fluid to a negative one following one course of Tryparsamide—22 intravenous injections of 3 grams weekly and 30 injections of Iodobismitol, 2 cubic centimeters in each injection, twice weekly. Case 9, chart A, is worthy of discussion. This man came to the sick bay for reenlistment. Four weeks prior to this reporting he had had an analysis of his spinal fluid, the result of which was, cell count 131, globulin 2 plus, Kahn 3 plus, colloidal gold 0000000000, and blood test Kahn 3 plus. While waiting for a waiver because of this finding, he received six injections of Tryparsamide, 3 grams each, and nine injections of Iodobismitol, 2 cubic centimeters each. Subsequently he was transferred to the naval hospital, San Diego, Calif., to await disposition. Here he received one additional injection of Tryparsamide and another spinal fluid analysis. The result of this test was: Cell

count 4; globulin negative, Kahn negative, gold curve 0000000000. Because of this finding the Bureau of Medicine and Surgery approved his reenlistment, having previously denied a waiver. Does this case constitute an error in laboratory procedure, or was the treatment sufficient to constitute a reversal of his spinal fluid?

Case 18, chart B, prior to his spinal analyses had received treatment for a fungus infection of the skin covering the tibia of each leg. These leg ulcerations were tertiary manifestations of his luetic infection and healed rapidly following injections of Tryparsamide. His spinal fluid showed marked pathological changes on analyses.

Treatment of the neurosyphilis aboard the U. S. S. *Lexington* has not been carried on a sufficient period of time to enable further definite conclusions.

SUMMARY

1. In 60 cases of serological relapse the spinal fluid was positive in 25 cases, 41.6 percent. Cases: 1, 5, 6, 7, 9, 10, 11, 17, 21, 24, 26, 27, 28, 30, 31, 32, 33, 35, 41, 51, 52, 53, 54, 55, and 58, chart A.

2. In 18 cases of persistently positive blood serum the spinal fluid was positive in 8 cases, 44.4 percent. Cases: 1, 2, 6, 8, 10, 13, 15, and 18, chart B.

3. In 38 cases in which a spinal fluid analysis was done for check up, the spinal fluid was positive in 9 cases, 23.6 percent. Cases: 2, 13, 15, 17, 18, 19, 23, 25, and 36, chart C.

4. A total of 116 spinal fluids of patients known to have had syphilis were studied; 42 were found positive, 36.2 percent.

5. The percentage of positive spinal fluids in asymptomatic neurosyphilis in the United States Navy is lower than that reported in civilian life by Epstein, Graves, and Sherman.

6. A spinal fluid examination should be required of all men with a history of syphilis prior to discharge from the United States Navy and if positive, a definite procedure should be outlined for treatment and disposition of these men. Reenlistment should be based on the analyses of both blood serum and spinal fluid. The cooperation of the patient should also be considered.

7. Early asymptomatic neurosyphilis responds readily to treatment.

8. Iodobismitol has given high therapeutic results in this series of cases and it is recommended that it be added to the Navy supply table.

REFERENCES

(1) The Safety of Lumbar Puncture for Ambulatory Patients: Harold Torbert. Arch. Dermat. and Syph., Chicago, November 1934, 30:692.

(2) The Spinal Fluid in Syphilis: T. A. Hewer, Lancet 1–1042, 1044, May 14, 1932.

(3) Proteins, Spinal Fluid in Syphilis: Clinical significance of total protein and globuline; preliminary report. Leo Spiegel, Arch. Dermat. Syph. 25–1071–1096, June 1932.

(4) Exton, William G.: Scopometry, a system for optical measurement and study of colloids and other dispersions. Arch. Path. 5–49, January 1928.

(5) E. De Masary: An attempt to interpret the lack of correlation between nerve symptoms and changes in the cerebrospinal fluid. Bull. Acad. de Med., Paris, 1932, cvlll–1174.

(6) Solomon, Harry C.: Neurosyphilis with negative spinal fluid. J. A. M. A. 77; 1701–1706, November 1921.

(7) Spinal Fluid in Syphilis: California and Western Medicine, February 1934. Norman Epstein, John M. Graves, Samuel R. Sherman, Leroy K. Gay, San Francisco.

(8) Iodobismitol: P. J. Hanzlik, J. A. M. A., April 1932.

CHART A.—*Serological relapse*

Case no.	Age of infection in months	Number of injections			Interval between diagnosis and serological relapse	Injections after relapse			Significant physical findings	Spinal fluid				Comment
		As.	Bi.	Hg.						Cell count	Glob.	Kahn	Colloidal gold	
1	78	8	0	8	8	5	0	4	Irritability	36	1 plus	4 plus	5444432 21	Positive.
2	26	16	5	12	6	8	0	0	None	0	Negative	Negative	00000 00000	
3	24	23	14	3	5	11	20	0	do	5	do	do	00011 10000	
4	16	24	22	0	8	9	10	0	do	2	do	do	00111 00000	
5	134	12	0	3	5	14	0	14	Personality change	26 5 490	Positive	4 plus	1123331000 5554432111 0122221000	Do.
6	74	47	23	26	10	28	23	5	None	6 8	2 plus Negative	do Negative	0002221000 00000000	Do.
7	42	16	14	3	10	8	8	0	do	6 3	2 plus Negative	2 plus Negative	1123210000 0001210000	Do.
8	86	36	9	16	8	22	36	0	do	3	Negative	do	00000 00000	
9	46	53	22	16	10	17	28	0	do	131 4	2 plus Negative	3 plus Negative	0000000000 0000000000	Do.
10	38	21	15	9	17	0	0	0	Sluggish knee jerks	22	do	do	00000 00000	Doubtful.
11	87	14	0	0	5	48	9	9	Mild elation	20	4 plus	4 plus	55555 43210	Positive.
12	91	23	0	26	52	9	21	0	None	4	Negative	Negative	00000 00000	
13	42	30	12	18	11	10	8	0	do	4	do	do	00000 00000	
14	44	35	17	4	4	13	8	0	do	8	do	do	00000 00000	
15	40	23	0	6	4	11	11	0	do	2	do	do	00000 00000	
16	40	26	20	3	5	8	20	0	do	1	do	do	00000 00000	
17	156	30	0	25	91	16	14	0	do	12	do	do	00000 00000	Doubtful.
18	37	16	7	0	5	10	3	0	do	3	do	do	00000 00000	

19	39	9	0	0	6	9	18	0	None	2	Negative	Negative	00000 00000	
20	41	8	0	8	16	30	8	15	do.	1	do.	do.	00000 00000	
21	65	12	5	5	34	50	20	0	do.	5	Positive	2 plus	00001 10000	Positive.
22	34	51	49	0	21	32	27	0	Reflexes hyperactive	2	Negative	Negative	00000 00000	
23	90	9	0	19	72	9	0	13	None	4	do.	do.	00000 00000	
24	96	36	19	0	10	32	19	0	Chronic osteomyelitis	96	1 plus	do.	00001 11000	Do.
25	36	18	25	0	18	7	00	0	None	4	Negative	do.	00000 00000	
26	69	34	32	0	13	12	32	0	Surveyed Bantis disease	2	2 plus	4 plus	12232 14000	Do.
27	149	57	0	40	30	31	0	9	Failing vision	0	3 plus	do.	Not made	Do.
28	36	36	28	0	24	0	0	0	Behavior, difficulty	6	do.	do.	24553 20000	Do.
29	33	36	21	15	21	14	0	15	None	5	Negative	Negative	00000 00000	
30	148	31	25	14	91	10	0	14	do.	34	1 plus	4 plus	Positive curve	Do.
31	22	82	26	94	6	7	7	7	do.	133	Positive	do.	do.	Do.
32	7	16	15	8	16	0	0	0	do.	41	1 plus	Negative	22110 00000	Do.
33	60	89	36	16	6	0	0	0	do.	8	do.	4 plus	43321 00000	Do.
34	132	10	16	0	63	6	0	4	do.	7	Negative	Negative	00000 00000	
35	37	22	39	16	22	8	10	15	do.	28	do.	do.	00000 00000	Doubtful.
36	74	43	23	0	48	24	23	0	do.	4	do.	do.	00000 00000	
37	26	16	20	8	8	16	11	0	do.	1	do.	do.	00000 00000	
38	98	44	0	45	52	14	5	0	do.	1	do.	do.	00000 00000	
39	72	48	44	80	21	16	24	0	do.	4	do.	do.	00000 00000	
40	80	40	20	12	65	10	16	0	do.	2	do.	do.	00000 00000	
41	52	50	34	0	12	16	19	0	do.	28	1 plus	(?)	33221 10000	Positive.
42	147	18	8	0	24	9	9	0	do.	4	Negative	Negative	00000 00000	
43	108	45	0	0	7	26	0	0	do.	1	do.	do.	00000 00000	
44	69	58	24	30	24	38	24	10	do.	2	do.	do.	00000 00000	
45	46	80	110	3	12	48	78	0	do.	3	do.	do.	00121 00000	
46	134	78	0	64	72	56	0	40	do.	2	do.	do.	00000 00000	

CHART A.—*Serological relapse*—Continued

Case no.	Age of infection in months	Number of injections			Interval between diagnosis and serological relapse	Injections after relapse			Significant physical findings	Spinal fluid				Comment
		As.	Bi.	Hg.						Cell count	Glob.	Kahn	Colloidal gold	
47	161	20	0	15	9	12	0	5	None	7	Negative	Negative	00000 00000	
48	185	27	23	8	72	23	23	7	do	2	do	do	00000 00000	
49	68	49	3	7	12	29	4	0	do	0	do	do	00000 00000	
50	69	56	19	0	48	24	20	0	Reflexes hyperactive	4	do	do	00000 00000	
51	24	51	4	58	19	50	37	0	None	14	do	do	00000 00000	Doubtful.
52	26	45	7	1	5	6	0	0	do	48	do	do	00000 00000	Do.
53	58	28	0	10	28	70	0	0	do	22	2 plus	4 plus	43211 11000	Positive.
54	81	27	13	0	36	13	0	0	do	24	Negative	Negative	11000 00000	Doubtful.
55	75	27	13	5	10	2	13	0	do	8	3 plus	do	01233 11000	Positive.
56	114	40	15	17	62	34	9	17	do	4	Negative	do	00000 00000	
57	91	26	12	24	15	4	12	0	do	4	do	do	00000 00000	
58	29	23	56	0	20	3	4	0	Late secondaries	35	do	do	01233 21000	Do.
59	132	48	0	26	19	36	20	0	None	2	do	do	00000 00000	
60	85	31	12	12	67	16	12	12	do	7	do	do	00000 00000	

CHART B.—*Persistent Positive blood serum Kahn*

Case no.	Age of infection	Number of injections			Significant physical findings	Spinal fluid				Comment
		As.	Bi.	Hg.		Cell count	Globulin	Kahn	Colloidal gold	
1	53	40	11	0	Reflexes hyperactive.	4 2	1 plus Negative	3 plus Negative	 0000000000	Positive.
2	65	72	62	10	None	4 10	1 plus Negative	4 plus Negative	? 0000000000	Do.
3	24	43	30	0	do	2	do	do	00000 00000	
4	84	55	37	0	Reflexes hyperactive.	4	do	do	00000 00000	
5	42	50	32	13	None	2	do	do	00000 00000	
6	84	47	5	10	Positive Rhomberg.	2	1 plus	do	?	Doubtful.
7	69	22	0	5	None	0	Negative	do	00000 00000	
8	80	43	13	11	do	232	.1 plus	4 plus	12443 24000	Positive.
9	77	49	25	24	do	0	do	Negative	00022 10000	
10	60	50	12	22	do	30	Negative	do	00000 00000	Doubtful.
11	41	19	32	0	do	1	do	do	00000 00000	
12	36	56	24	12	do	4	do	do	00000 00000	
13	52	83	61	3	Syphilitic aortitis	13	3 plus	3 plus	23244 32100	Positive.
14	120	31	0	25	None	8	Negative	Negative	00000 00000	
15	62	67	22	36	do	?	Positive	4 plus	33322 10000	Do.
16	11	30	33	0	Heavy drinker	4	Negative	Negative	00000 00000	
17	14	18	21	0	Uncooperative	6	do	do	00000 00000	
18	78	66	31	8	Tertiary skin lesions	201	4 plus	4 plus	55555 43210	Do.

CHART C.—*Spinal fluid analyses for check-up*

Case no.	Age of infection	Number of injections			Significant physical findings	Spinal fluid				Comment
		As.	Bi.	Hg.		Cell count	Globulin	Kahn	Colloidal gold	
1	36	33	26	4	None	4	Negative	Negative	00000 00000	
2	24	28	34	0	do	19	do	do	00000 00000	Doubtful.
3	25	28	56	0	do	2	do	do	00000 00000	
4	30	16	12	0	do	4	do	do	00000 00000	
5	69	32	25	0	do	1	do	do	00000 00000	
6	72	24	0	14	do	3	do	do	00000 00000	
7	65	27	19	0	do	0	do	do	01111 00000	
8	146	27	0	34	do	4	do	do	00000 00000	
9	124	24	8	34	do	6	do	do	00000 00000	
10	19	21	19	0	do	1	do	do	00000 00000	
11	98	45	0	31	do	4	do	do	00000 00000	
12	65	33	5	40	do	4	do	do	00000 00000	
13	30	32	11	16	Deserted	49	do	do	00000 00000	Do.
14	84	32	0	38	None	8	do	do	00000 00000	
15	41	38	8	10	Developed a psychosis.	22	2 plus	4 plus	55432 11000	Positive.
16	76	41	54	21	None	5	Negative	Negative	00000 00000	
17	50	29	0	19	do	8 4	do	1 plus Negative	4355210000 0000000000	Do.
18	66	40	0	21	do	7	1 plus	1 plus	32243 21100	Do.
19	216	24	8	0	Argylle-Robt. pupils.	24	do	4 plus	54432 10000	Do.
20	49	74	45	11	None	4	Negative	Negative	00000 00000	
21	19	26	17	0	do	3	do	do	00000 00000	
22	14	26	26	0	do	5	do	do	00000 00000	
23	15	23	38	0	do	14	do	do	00000 00000	Doubtful.
24	81	49	22	25	do	5	do	do	00000 00000	
25	38	27	38	0	do	240	do	do	00000 00000	Do.
26	81	33	16	12	do	3	do	do	00000 00000	
27	52	22	31	10	do	1	do	do	00000 00000	
28	26	26	30	0	do	2	do	do	00000 00000	
29	132	24	0	9	Cardiac damage	3	do	do	00000 00000	
30	68	36	20	8	None	4	1 plus	do	01210 00000	
31	88	40	0	52	do	2	Negative	do	00000 00000	
32	14	33	26	0	do	3	do	do	00000 00000	
33	14	32	28	0	do	2	do	do	00000 00000	
34	21	32	46	0	do	4	do	do	00000 00000	
35	56	32	23	7	do	2	do	do	00000 00000	
36	28	24	16	4	do	9	1 plus	do	00000 00000	Do.
37	26	18	25	0	do	2	Negative	do	00000 00000	
38	36	20	28	0	do	7	do	do	00000 00000	

GONORRHEAL INFECTIONS IN SAMOA

By BEN HOLLANDER, Lieutenant Commander, Medical Corps, United States Navy

During 18 months over 800 patients were admitted to the Samoan Hospital for all diseases and conditions; 7,884 patients were seen in the out-patient clinic. No cases of venereal disease were seen in the clinic. The following table shows the admissions due to gonococcus infection.

No.	Sex	Age	Gonococcus infection of—	Complication
6	M	18 years	Urethra	Epididimitis.
	M	19 years	do	Do.
	M	20 years	do	Do.
	M	16 years	do	None.
	M	18 years	do	Epididimitis.
	M	1 year, 8 months	do	None.
4	F	19 years	Uterine cervix	Salpingitis (not operated).
	F	17 years	do	Do.
	F	30 years	do	None.
	F	20 years	do	Do.
6	------	Infants	Conjunctiva	Do.

Due to the few cases of gonorrheal infections noted during this period, one might conclude that gonorrhea is not a prevalent disease in American Samoa. The following table presents the pertinent facts regarding gonorrheal infections among the naval personnel on this station during 2⅓ years.

	Admissions		
	1933	1934	1935 [1]
Naval station personnel	[2] 6	0	1
Fita Fita guard	2	0	0
Average complement	161	171	170
U. S. S. *Ontario*	3	[3] 3	4
Average complement	52	53. 5	54

[1] 4-month period.
[2] 2 cases contracted en route to Samoa.
[3] 2 cases contracted in Hawaii.

During January 1935, three Samoan women asserted they were infected by sailors from the U. S. S. *Ontario*. One of these women had cohabited with at least five sailors at various intervals, one of whom had had an admission for gonococcus infection of the urethra. It is possible—if not probable—that this was the primary focus for the subsequent infection of three women and four sailors. These men and women lived together, occupying a Samoan house in common. Since these patients were placed under supervision, no new cases have developed.

The foregoing would further substantiate a belief that gonorrhea is not a very prevalent disease among Samoans, especially since few places offer more favorable opportunities for sexual freedom as these isles in the midst of the South Seas.

The relative infrequency of gonorrhea among Samoans has been noted by numerous medical officers. They also remarked upon the frequency of "Samoan conjunctivitis" caused by a Gram-negative intracellular diplococci which sometimes attained epidemic form. Capt. E. U. Reed saw only one case of gonorrhea during a 2-year period of duty in Samoa, and that was in a mestizo woman from British Samoa who had contracted the infection prior to coming to American Samoa (1).

Dr. Mitford, medical officer in charge of the hospital in Apia, British Samoa, stated that he rarely saw a case of gonorrhea, and is unable to account for the apparent rarity of the disease (2).

It would be inadvisable to conclude that gonorrhea is a rare disease in Samoa. The native is exceedingly secretive regarding any genital disability or disorder he may have. When he does not improve favorably under native treatment or if his illness becomes worse he will consider reporting to the "papalagi" (foreign) doctor.

Of the six male patients reporting to the hospital, four had epididymitis. The pain and disability incident to this complication frightened them, and the fear that their sexual ability might be destroyed induced them to enter the hospital for treatment. The 16-year-old boy was a student in the Marist Brothers' school at Leone and when a brother discovered that the boy had a urethral discharge he brought him to the hospital for treatment.

None of the female patients reported to the hospital, but were apprehended upon complaints by enlisted men and placed under treatment.

Experience has probably taught the Samoan that the best treatment for gonorrhea is complete rest. Should he have some condition requiring hospitalization and also a urethral discharge, he would probably postpone going to the hospital or clinic until the genital disorder subsided. He would conceal his genital disability as thoroughly as possible lest it be discovered and the exercise of his sexual activity be endangered.

The Samoans are not immoral so much as unmoral. Their civilization does not require sexual contenence. Copulation, birth, and death are no mysteries to the children. They witness these performances from the time they begin to recognize and discern human behavior. Samoan children know that coition results in pregnancy and recognize its outstanding sign. The Samoan boys and girls do not pass through a "trying ordeal" during the period of puberty. Moreover, their adolescence is not fraught with difficulty in gratifying their sexual desires (3).

There are no prostitutes. In the environment of the naval station there are a few women who negotiate their virtue when the opportunity arises during the visit of a ship in port.

In the outlying stations gonococcus infection is rarely seen. Three chief pharmacist mates stated that during their tour of duty on their stations covering a total of 46 months not a single case of gonococcus infection was noted. All of the patients admitted to the Samoan Hospital were from the villages in the vicinity of or within easy access to the naval station.

Pelouze states that about 95 percent of all men in cities contract gonococcus infection sometime during their lives. In another modern textbook 25 to 40 percent of adult males are said to contract the infection.

Urologists agree that gonococcus infection is one of the most prevalent diseases among all peoples and especially common in thickly populated centers. It is also agreed that alcohol renders one more susceptible to infection and that its use must be curtailed to secure favorable results in the treatment of this disease. In a recent paper the following paragraph is pertinent:

> Physiologic factors center largely around the action of ingested alcohol in checking immunity response. (The same action occurs as the result of prolonged inhalation of alcohol or ether fumes.) (6.)

Alcoholism is uncommon among Samoans. They like alcohol and invariably request "elixir terpene hydrate" when troubled with a "cold", and often ask for it when they have no cough. Some frankly state they like it and that it makes them "feel better."

They would purchase alcoholics if they had money. When a Samoan is earning money, every member of his family and relations know how much he will receive. He cannot spend it, since it actually belongs to the family.

The family is the social and economic unit. The welfare of the family is the prime consideration, and no individual in the family group may have more than another without being accused of disloyalty. Among Samoans, such undutiful conduct toward the family group is an "unpardonable sin." The possessions of one family are mostly known to every other family and may be borrowed without notice. The loser may hope to reborrow such possessions or other possessions from the borrower or some other family when the opportunity arises. The accumulation of wealth by an individual Samoan is practically impossible (7).

With the exception of the Fita Fita guard, and those working about the naval station, very few natives have money which they may spend according to their desires. Even among these, only a small portion indulge in alcoholics, since the demand of the family group upon money earned is dominant. Those persons noted to have indulged excessively in alcohol were usually members of the Fita Fita guard and half-castes. The half-caste group of Samoans

are deserting the family group ties and adopting western customs. Very few Samoans away from the naval station imbibe in alcoholics.

The diet of the Samoan is a bland diet. It consists mainly of taro, bananas, breadfruit, papaya, coconuts, fish and shellfish. Peppers and spices are seldom used. Little beef is consumed because the supply is scarce. Pigs and chickens are eaten mostly at feasts and ceremonials, since the supply is insufficient to form a portion of the regular daily ration.

Kava is the native drink, made from the kava root. It is said to be intoxicating, but intoxication such as that caused by alcohol has not been seen. When a small piece of the root is chewed it soon causes numbness of the tongue, and if continued it renders talking difficult, due to paralysis of the organ. Excessive drinking of the substance may paralyze the person sufficiently to render him incapable of speech or locomotion.

Although yaws is exceedingly prevalent, and probably every Samoan has or has had yaws, no cases of locomotor ataxia or neuroyaws were seen. This coincides with the observation of the preceding director of the Samoan Hospital, Lt. J. R. Fulton. Three cases of difficulty in locomotion of the lower extremities were not traceable to cerebrospinal pathology. A period of rest at the hospital resulted in marked improvement. It is believed that these disabilities may have been due to excessive kava drinking. Some Samoans insist on having kava daily, although they may display a complete indifference if food is not supplied regularly.

Father Bellwald, a priest who has been in these islands for over 30 years, stated that "a very strong infusion of kava is considered by the natives to be a very good medicine for genito-urinary diseases." He further stated that he believed "there is very little gonorrhea among the natives."

The climate is tropical. The average daily temperature is 80° to 83°, the humidity ranges from 70 percent to saturation. Two hundred inches of rain is the average annual precipitation. This kind of climate is not conducive to activity but rather to complete relaxation. The splendid physique of these natives is a hereditary and racial characteristic and cannot be attributed to good hygiene, sanitation, or systematic exercise.

The Samoan is not lazy, but prefers to avoid activity except when it is necessary. He is able to perform hard work if the occasion demands it. His life is a satisfactory adaptation to his environment. When he becomes ill, he relaxes completely and weeks and months of inactivity does not appear to have any ill effect upon him.

In the 1933–34 Annual Report of the Department of Public Health of American Samoa (8) there is an interesting statement regarding gonorrhea in these islands. But the estimate that 15 percent males

and 5 percent females of the population are affected with this disease, and a further statement that the estimates are too low are not substantiated by my experience gained while on duty at the Samoan hospital. It may be noted that among over 800 patients seen in the wards at the hospital and 7,884 patients reporting to the out-patient clinic not one case of gonococcus infection was detected.

Upon admission every patient was given a thorough physical examination and caution was taken to avoid overlooking any possible venereal infection. During the past year 690 male patients were examined for possible gonococcus infection. Not one lesion of gonorrhea, chancroid, yaws, or syphilis was seen. Smears were made from the uterine cervix and urethra of 129 female patients. Two cases showed gram-negative diplococci but no macroscopic pus of inflammation was noted. These women had given birth to children but only one gave a history of her offspring having had a purulent conjunctivitis. Not one woman has reported to the hospital for treatment because of leucorrhea. It is doubtful if a native woman would come to the hospital if she had such a disorder unless it disabled her. One may conclude from these findings that women having vaginal discharges do not come to the hospital, and that further investigation is required to determine the prevalence of gonorrheal infection among native Samoan women. It is inadvisable to consider these findings sufficient proof that gonorrhea is rare in Samoa.

Gonococci may lie within the tissues even though not manifested in smears. I have known of cases of acute endocervicitis, clinically indicative of gonorrheal infection wherein the pus was negative for gram-negative diplococci, nevertheless served as the foci of infection for other cases of definite gonorrhea.

The apparent infrequency of gonorrhea among Samoans has evoked considerable speculation and investigation by many medical officers. The primitive person is not a trusting one and is very suspicious. To make a vaginal examination and smears from the uterine cervix is impossible if the patient is free from abdominal complaint. Let her suspect that the examiner is trying to satisfy his curiosity and she will depart and consult her "witch doctor" and also elaborate to her associates, friends, and family about the "papalagi doctor's", (foreign doctor's) curiosity.

An acquired immunity has been given as a possible reason for the scarcity of the infection among Samoans. Some medical officers believe that the bouts of filarial fever result in the destruction of the gonococci. Exceptional results have been reported in the treatment of gonorrhea by fever therapy. Subjecting the patient for 6 to 8 hours to a temperature of 106° to 107° and repeating the session of fever at intervals of 2 days until from two to four or more sessions have been given is believed to effect a cure in many cases.

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Twenty-five out of twenty-nine patients were reported to have been cured by this method. It is further believed that more favorable results would be obtained from daily sessions of fever (9).

The bouts of filarial fever seen during the past 18 months rarely exceeded 104° and the longest duration of such a fever was noted in two cases to be 3 hours. When the fever was prolonged for 2 or 3 days it was never continuous but a remittent type of fever rarely higher than 102°.

Not one case of filarial fever has been seen to equal the malarial type of fever in severity. The filarial fevers that lasted more than a few hours usually proved to be due to one or more large abscesses in muscle tissue. Filarial fever falls far short of the requirement of fever therapy as given in the article by Desjardines et al.

Neither an acquired immunity nor bouts of filarial fever can account sufficiently for the apparent relative rarity of gonorrhea among Samoans. The avoidance of alcoholics, a bland diet, rest, and the absence of drastic treatment contribute largely if not completely to the prompt recovery from acute gonorrhea and the prevention of complications and residual sequelae.

All the patients admitted were young adults. Those with epididimitis and salpingitis had the condition at the time admitted. Arthritis of gonococcic origin has not been seen. Epididimitis and salpingitis are probably very infrequent. All the patients responded favorably to treatment and were cured. Neither fever, intravenous medication or vaccines were used. The cases of opthalmia neonatorum made a very prompt recovery in 2 to 4 days.

Ten percent Argyrol was used for instillations in the urethra. In cases of conjunctivitis, the eyes were irrigated with warm 2-percent boric acid solution followed by the instillation of 10 percent Argyrol.

Gonorrheal endocervicitis was treated by hot vaginal douches of 1 : 6,000 solution of potassium permanganate. In all cases the treatment consisted of rest in bed, the regular Samoan diet, and mild medication.

The youngest patient was a boy 1 year and 8 months of age. With reference to gonococcic infection of children, the following is quoted from modern textbooks:

> Infection in the female may in some case be due to contamination of towels and other inert objects, but in the male, the mode of infection is practically always through coitus. Gonorrhea was seen in boys young as 4 years but never met any case of infection in the male that was not traceable to coitus (5, p. 172).
>
> While the possibility of extra sexual infection of the male urethra must be admitted, the physician is hardly likely to see such a case in a very long lifetime (4, p. 91).
>
> Gonococcus inflammation * * * occurs in infants but most of the cases are in those over 7 years old. * * * Poynter has reported a case in a boy of 3 years, who, when 5 years old, required treatment for a urethral stricture. He was believed to have been infected by a nurse (10, p. 628).

There are cases also in which a gonococcal arthritis occurs in infancy without any apparent primary focus of infection such as opthalmia or a vaginitis; it is seen sometimes in male children.

In this same textbook a reference is given of twenty-six cases of gonorrheal arthritis in children under 3 years of age, reported by Emmett Holt, and Dr. Kimball has reported—

Eight cases in infants not more than 3 months, and several of these cases were males with no trace of gonorrheal infection elsewhere (11, p. 482).

Specific urethritis in male infants and male runabout children is of rare occurrence. Eight patients under 4 years of age have come under my observation. The oldest of the group, aged 4 years, developed a stricture (12, p. 464).

Case Report

A Samoan male child, age 1 year and 8 months, was brought to the hospital by his mother who stated that his penis was swollen for the previous 2 days. The child had always appeared healthy and developed normally. He began walking at 9 months of age. The presence of venereal disease among members of the family or neighbors was denied. The mother had never noticed any precocious genital activity by the child. No information was obtained ascertaining the source of the infection.

Upon examination of the child's genital a marked balanitis and purulent urethral discharge was noted. The testicles were in the scrotum and were not tender. There was a bilateral inguinal lymphadenitis. Smears made from intraurethral pus was positive for the characteristic gram-negative intracellular diplococci of gonorrhea.

Treatment consisted in cleaning the preputial sac by irrigation with warm saline and instilling a medicine dropperful of 10 percent Argyrol into the sac. The end of the medicine dropper was never placed into the urethra. This treatment was administered three times daily and at bedtime (8 p. m.) for 3 days; then three times daily for 5 days, when macroscopic pus had completely disappeared. This was followed by treatments given morning and night for 1 week.

Thereafter the child was seen twice weekly for 1 month.

There has been no recurrence. Upon examination 3 months later no stricture was evident.

CONCLUSIONS

1. Gonorrhea is apparently not a prevalent disease in Samoa, but further investigation to determine the incidence, the possible relative immunity and the reasons therefore is required.

2. Patients affected with gonorrhea report to the Samoan hospital for treatment only as a last resort or when apprehended.

3. The Samoan responds very favorably to treatment.

4. The abstinence from alcoholics, a bland diet, rest and the avoidance of drastic treatment are believed to be the main contributing factors in aiding the body to combat the infection.

5. A case of gonococcus infection of the urethra in a male child age 1 year and 8 months is presented.

REFERENCES

(1) Reed, E. U.; United States Naval Medical Bulletin, October 1934.
(2) Personal communication.
(3) Meade, Margaret; Coming of Age in American Samoa.
(4) Pelouze, P. S.; Gonorrhea in the Male and Female, Second edition.
(5) Eisendrath and Rolnic, Urology.
(6) Pelouze, P. S.; The Immunologic aspects of Gonococcic infection, Journal A. M. A. December 15, 1934.
(7) Keesing, Felix; Modern Samoa, 1933.
(8) Report of the Department of Public Health in American Samoa. 1934.
(9) Desjardins et al: Fever Therapy for Gonococcic Infection, Journal A. M. A. March 16, 1935.
(10) Holt and Howland; Diseases of Infancy and Childhood, 8th ed.
(11) Still, G. F.; Common Disorders and Diseases of Childhood, 3d ed.
(12) Kerley; Practice of pediatrics, 2d ed.

CLINICAL NOTES

CONGENITAL DISLOCATION OF THE HIP—CASE REPORT

By R. D. JOLDERSMA, Lieutenant Commander, Medical Corps, United States Navy

A girl 14 months old was referred to the hospital due to peculiar limping gait. Examination showed 1½-inch shortening of left leg and a dislocated hip. X-Ray in September 1933 showed upward and backward dislocation, no acetabular ridge, and epiphysis of femur not developing. The hip was reduced by the Lorenz bloodless method and cast was applied with the left leg and hip in frog position. The cast included abdomen, hip and leg to the knee, and in 3 months the child was walking in frog position. As this was in the tropics a fresh cast was applied every month. At the end of 8 months the cure was considered complete (picture 2 taken in May 1934). The child walked at once and in 1 week was apparently normal. In March 1935 picture 3 was taken showing an apparently normal condition.

RHINOSPORIDIUM SEEBERI

By O. R. NEES, Lieutenant, Medical Corps, United States Navy

The case herein reported is one of nasal polyps due to an infection with organisms known as Rhinosporidium Seeberi. As far as I have been able to ascertain there have not been more than a half dozen such cases reported in the United States.

Guillermo Seeber (1) first described this parasite in 1900. His patient was an agricultural laborer 19 years of age who lived in Argentina. Seeber also mentioned two other cases making a total of three for Argentina. Thirty or more cases of this infection have been reported from India and Ceylon by O'Kinealy (2), Beattie (3), Ingram (4), R. E. Wright (5), and others. Most of their cases have been infections of the nasal passages. However, Ingram found growths produced by the same organism on the conjunctiva and the glans penis.

The first American case was described by J. Wright (6) in 1907, who had received his material from Dr. E. C. Ellet of Memphis, Tenn. The patient was a young farmer who had lived all his life near Memphis.

Other cases in the United States have been reported by Mary C. Lincoln and Stella M. Gardner (7) in 1929; by Weller and Riker (8) in 1930; by Hanson (9) in 1931; by Graham (10) in 1932.

An excellent article covering the entire field of information concerning Rhinosporidium was written by Ashworth (11) in 1922.

The organism has been called Rhinosporidium kinealyi because of the cases reported by O'Kinealy; but the priority of Seeber was definitely established by the investigations of Ashworth.

Case Report

G. S. M. Chief Yeoman, Fleet Naval Reserve; age 39.

When first seen by me in November 1932 the patient was complaining of a chronic nasal "cold." He stated that his condition had existed since 1928. He was unable to breathe through the left side of his nose. There was no complaint of nose bleed. The family history was of no consequence. The patient had had no serious illnesses. He had been in Mexico from 1915 to 1916; Philippine Islands from 1917 to 1919; southern California from 1920 to 1925; Australia 1 month in 1925 with the United States Fleet; Alabama in 1926 to 1927; Southern California in 1928 to 1930; Hawaiian Islands in 1930 to 1934. He had never had any prolonged contact with farm animals or animals of any kind.

Nasal examination in November 1932 showed a cauliflower-like mass of polyps growing from the left side of nasal septum. They were far back and about 1 centimeter from the floor of the nose. They were dark red in color and had somewhat the appearance of a raspberry. They were removed with a nasal snare in the usual manner. Following their removal the patient was relieved and free from symptoms for about 1 year, when his former complaint returned. He was seen again by me in November 1934 and a nasal examination revealed the same condition as when first seen in 1932. The polyps were again removed and specimens sent to the United States Naval Medical School for pathological examination. The base of the pedicle from which the polyps grew was completely destroyed by repeated cauterizations. At the present time, 8 months after the second removal, there has been no recurrence of the growths.

I am indebted to Lt. Comdr. G. A. Alden, Medical Corps, United States Navy, for the pathological diagnosis and the photomicrographs in this case. His report on these specimens submitted is as follows:

Gross examination.—Specimen consists of six whitish, irregularly shaped pieces of tissue, the largest of which measures 2 by 1.4 by 1 centimeter.

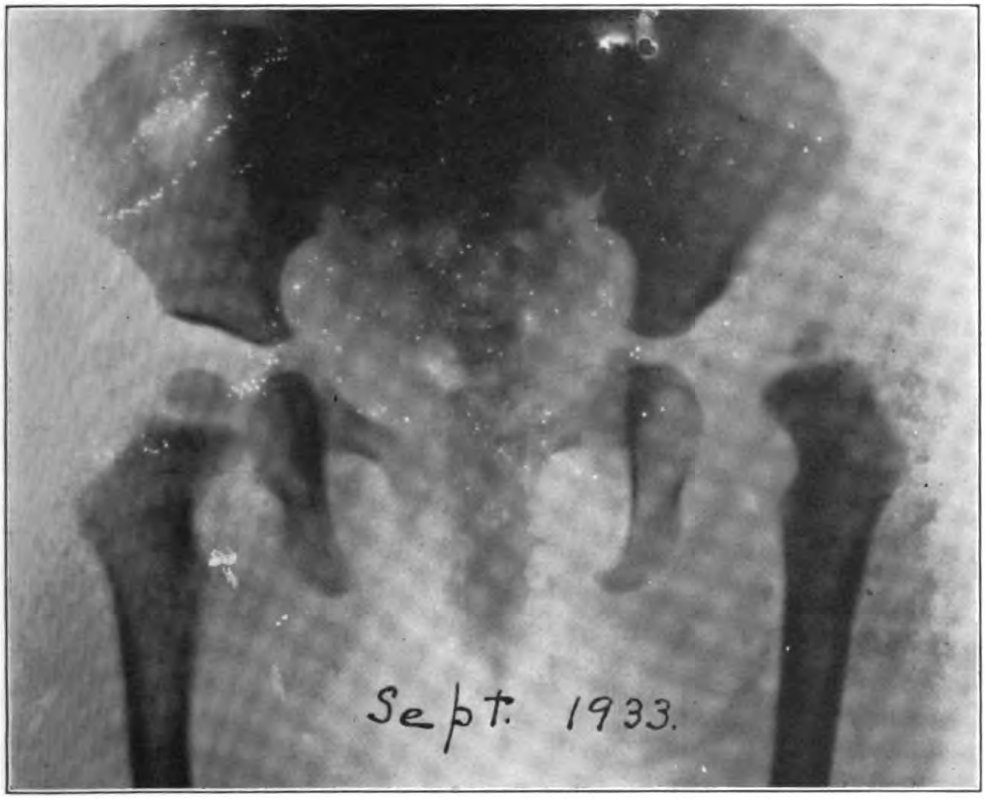
Microscopic examination.—Sections show well formed polypoid structures covered by an edematous, partially degenerated, stratified squamous epithelium.

There is a large amount of cellular stroma containing numerous large round molds of Rhinosporidium seeberi. Some are in the early developmental stage, small in size and without complete development of spores. Others are very large with a distinct, fairly thick membrane containing numerous round, poorly staining spores. Some of these are seen in the early ruptured stage with a scattering of the spores into the tissue. Many of the large fungi have lost their spores by degeneration or otherwise, leaving only the distinct capsule. A few of the very large organisms appear degenerated, the spores taking the hematoxylin stain.

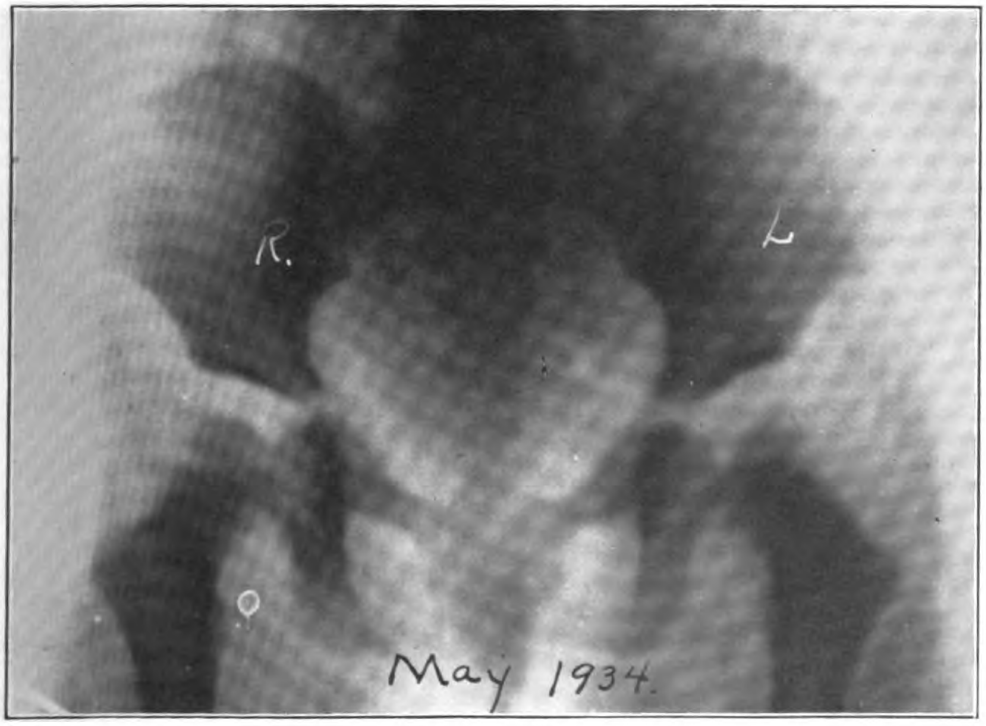
In addition to the fungus growth the stroma is infiltrated with lymphocytes, plasma cells, and a few granular leucocytes.

The polypi are also quite vascular.

The developmental life history of Rhinosporidium seeberi has been worked out by Ashworth (11). He believes that the organism belongs to the lower fungi and not to the sporozoa and tentatively places it in the suborder Chytridineae. There seems to be but one species concerned in the production of the disease in question although the cases reported have come from widely separated geographic



HIP JOINT IN 1933.



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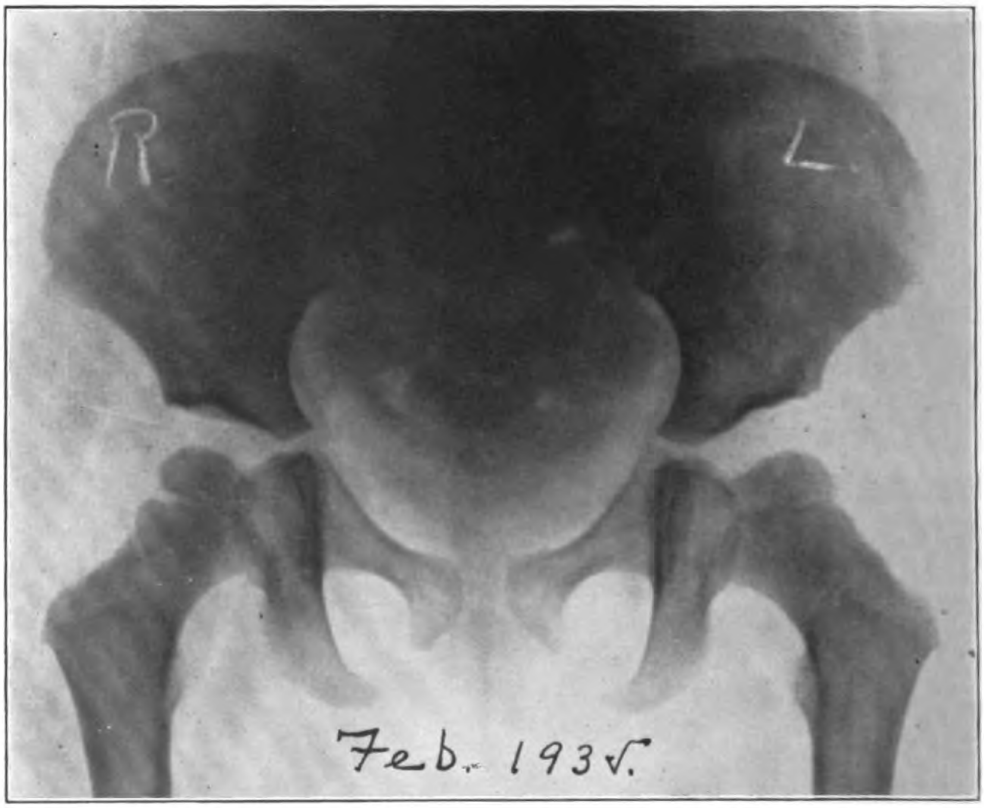


CONDITION OF HIP JOINT IN 1935.

locations. It seems possible that infection with Rhinosporidium seeberi may be more common than the few cases reported indicate. The diagnosis can be made only by a microscopic examination of the tissue removed. All the reported cases have been in the male sex. Most of the cases have been in young men; however, it has been found in boys as young as 10 and in men 60 years of age.

As stated above, all the earlier cases of infection with Rhinosporidium seeberi were of the nasal passages. Later, however, infections have been found of the aural canal, the conjunctiva, and lachrymal sac, the uvula, and the penis. This wider distribution makes the term Rhinosporidium less appropriate.

As to the mode of infection and transmission nothing is known with certainty. Efforts at experimental animal inoculation have failed. It has not been shown that the disease is spread by contact. That there is auto-inoculation is proved by the fact that additional polypoid growths occur in nearby but new sites. There is no evidence that the disease is spread by the blood or lympth stream.

The treatment of choice is complete surgical removal with destruction, by cautery or otherwise, of the base of the pedicle.

Wright (5) reports success in the treatment of his case of Rhinosporidium infection of the conjunctiva with 2 percent tartar emetic dropped into the eye three times a day.

REFERENCES

(1) Seeber, G. R.: Un nuevo Esporozoario Parasito del Hombre dos Casos Encontrados en Polips Nasales. Thesis, Universidad Nacional de Buenos Aires, 1900.

(2) O'Kinealy, F.: Localized Psorospermiosis of the Mucous Membrane of the septum nasi. Proc. Laryng. Soc. London, 1903, 10, 190; J. Laryng. Rhin. & Otol., 1903, 18, 375–378.

(3) Beattie, James M.: Rhinosporidium Kinealyi (Minchin): A Sporozoon of the Nasal Mucous Membrane. Brit. M. J., 1906, 2, 1575–1576. Also J. Path. & Bact., 1906, 11, 270–275.

(4) Ingram, A. C.: Rhinosporidium Kinealyi in Unsual Situations. Lancet, 1910, 2, 726.

(5) Wright, R. E.: Rhinosporidium Kinealyi of the Conjunctiva. India Med. Gaz., 1922, 57, 6–7.

(6) Wright, J.: A Nasal Sporozoon (Rhinosporidium Kinealyi). New York M. J., 1907, 86, 1149–1152.

(7) Lincoln, Mary C., and Gardner, Stella M.: A Case of Rhinosporidium Seeberi in a Resident of the United States. Arch. Path., 1929, 8, 38–45.

(8) Weller and Riker: American Journal of Pathology, vol. VI, no. 6, November 1930.

(9) Hanson: Annals of Otology, Rhinology and Laryngology, 1931.

(10) Graham: American Journal of Clinical Pathology, March 24, 1932.

(11) Ashworth, J. H.: On Rhinosporidium Seeberi (Wernicke, 1903), With Special Reference to its Sporulation and Affinities. Tr. Roy. Soc. Edinburgh, 1922, 53, pt. 2, 301–342. (Issued separately in 1923.)

PULMONARY ASPERGILLOSIS—REPORT OF CASE

By J. G. WRIGHT, Lieutenant, Medical Corps, United States Navy

Pneumomycosis was first described by Hughes Bennett in 1842. In 1853 Rayer and later Gairdner described cases of pulmonary tuberculosis in which the pleurae were invaded by a fungus. Virchow in 1856 described several cases of aspergillary mycoses of the lungs and bronchi. Dieulafoy, Chantmesse, and Widal in 1890 described clinically several cases of pulmonary aspergillosis occurring in persons who were engaged in the work of fattening pigeons for the Paris market and they advanced the idea that the infection was a primary one.

In 1897 Renon brought forth a very complete monograph on the subject, experimental as well as clinical. He considered it a trade disease among those undertaking the artificial feeding of pigeons and those manipulating hair and hair products. He believed the disease rare outside of Paris. The infecting agent he states is Aspergillus fumigatus and rarely Aspergillus niger, spores of which may be mingled with grain, seeds, or flour. Hair sorters make use of rye flour to facilitate separation of hairs and hence their exposure.

F. Galdi in 1921 reported a case in a carpenter's daughter, age 6 years, and which he believed was acquired through the ingestion and inhalation of sawdust. Mary Lapham in 1926 reported 10 cases of primary pulmonary aspergillosis from an obscure village in the Blue Ridge Mountains and divided the cases into two groups namely, the wet or parenchymatous and the dry or interstitial. She also calls attention to a definite relation to asthmatic attacks in certain cases.

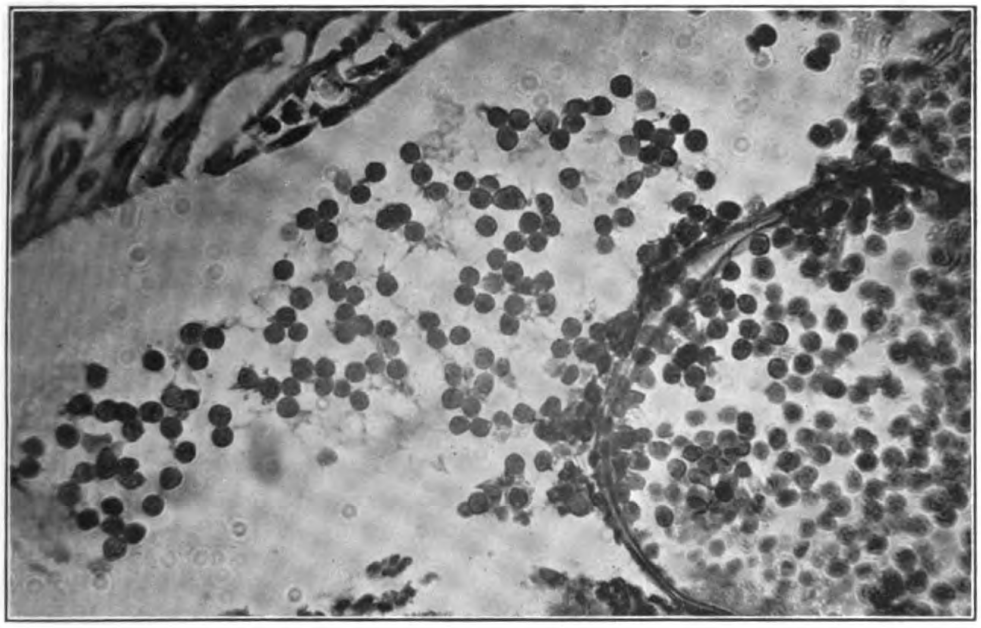
Pinta, in 1935, reports asymptomatic cases picked up in Oklahoma during a routine chest examination of workers in iron mines.

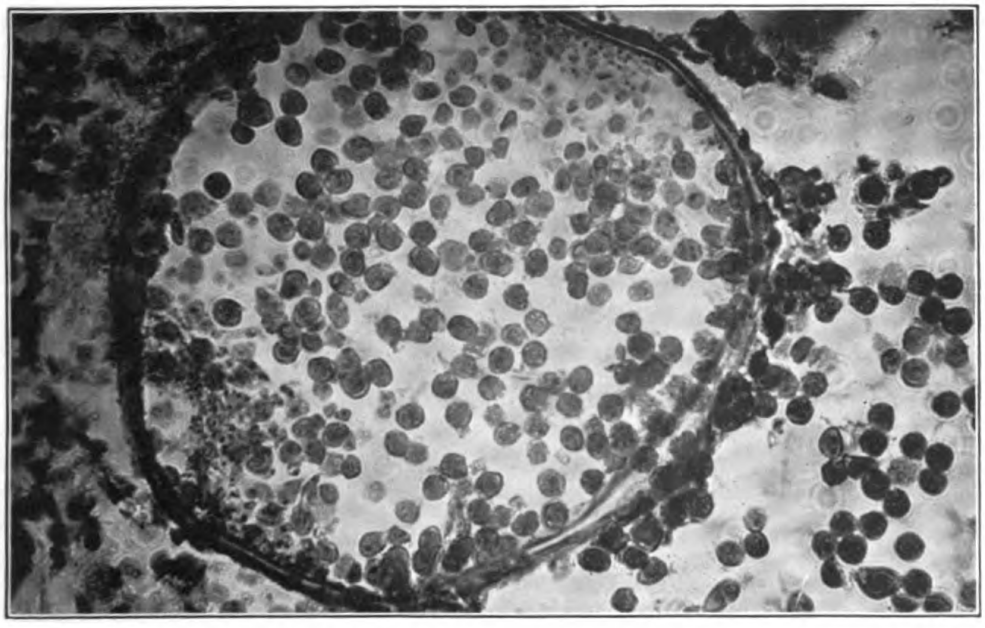
The Genus aspergillus first described in 1725 by Micheli belongs to the family Aspergillaceae, order Aspergillales of class Ascomycetes. This is according to the classification of Castellani. He states that they are usually saprophytes but may become parasites.

The patient O. G. R., watertender second class, United States Navy, was admitted to the sick list on August 13, 1934, complaining of loss of weight, shortness of breath, and an intermittent cough. He first noticed his symptoms about 2 months ago.

Family history.—The patient is the oldest of six children, three of whom died at birth. He has one brother, age 17 years and one sister, 10 years, living and well. His father is age 50, mother age 50, both living and well. His paternal grandparents are living and well, age not known. His maternal grandparents are dead, cause not known. Patient denies knowledge of familial diseases. He comes from a line of Mississippi farmers. He has been married 10 years, has one child, 8 years of age. His wife and daughter in apparent good health.

Past personal history.—Patient was born in Purvis, Miss., on February 16, 1907. He lived on a general farm until entering the Navy on October 21, 1925. He finished seventh grade of school, and denies ever having actively cared for poultry or pigeons. History of mumps at 6 years, typhoid fever at 9 years, malaria at 15 years, measles at 16 years, gonorrhea in 1933, and chicken pox in 1934. He





RHINOSPORIDIUM SEEBERI PHOTOMICROGRAPHS.

drove overland from California to Virginia in April and May of 1934, and stopped off in Arkansas for a few days.

Present illness.—About 2 months ago he began to notice a shortness of breath and a persistent cough, later noticed a loss of weight. The cough was intermittent, and quite troublesome at times but with varying amounts of stringy sputum.

Physical examination.—Temperature 98, pulse 70, respiration 22. Was a fairly well-developed, undernourished, white, male adult. His weight was 130 pounds (weight 153 pounds in January 1934). Presented a fair dental status, superficial glands were not palpable, blood pressure 102 systolic, 60 diastolic. Chest: Respiratory excursions were equal and appeared normal. There was a marked increase in tactile fremitus over the right lung. The breath sounds over the upper portion of the right lung were increased.

W. B. C. 13,100; differential, bands 10, segmented cells 53; lymphocytes 24, eosinophiles 1, monocytes 12. Sputum was thrice negative for acid-fast bacilli. The sputum was tenacious, glary, and of a nonpurulent character. When examined in a preparation unstained it was found abounding in a fungus insoluble in 10-percent potassium hydroxide and resembling Aspergillus fumigatus morphologically. Sputum was not cultured at this time. Kahn test of blood serum was negative. August 16, 1934, had been afebrile, condition was not changed materially. Transferred to U. S. S. *Relief.*

Copy of record from the U. S. S. "Relief".—August 17, 1934: Blood count: R. B. C., 4,900,000; W. B. C., 11,300. Hgb. 80 percent. Band forms 5 percent. Segs. 48 percent. Lymphs. 43 percent. Eosinos. 1 percent. Monos. 3 percent. Urine: Clear amber, acid, sp. gr. 1.032; albumen and sugar negative. Little mucus; occasional epithelium. Blood sedimentation test: Index 6 mm; time 60 min. Sputum: No acid-fast bacilli found. No fungus found. Specimen unsatisfactory for culture.

August 18, 1934: Feces: No ova or parasites found. Sputum: No fungi found (culture made). No acid-fast bacilli found. X-ray of chest: The apices and costophrenic sinuses are clear. There does appear considerable fibrosis in the right lung with thickened interlobar pleura on the right side, and this fibrosis appears in a lesser degree on the left. Impression: Chronic interstitial pneumonia. Kahn test negative. (S.) J. P. Owen, commander (M. C.) U. S. N.

August 22, 1934: Sputum: No acid-fast bacilli found. No fungus found.

August 26, 1934: Sputum: Culture—Aspergollus Niger. Aspergillus fungi found and isolated (Asperilli Niger) in sputum culture. The herpetic lesions on penis cleared up immediately and a Kahn test is negative.

August 28, 1934: Sputum: No acid-fast bacilli found.

August 29, 1934: Urine: Clear straw; acid. Sp. gr. 1.015. Albumen and sugar negative. Much mucus; 1 to 2 leucocytes per field (HD), few epithelium.

August 30, 1934: Dental examination: Prophylactic treatment. (S.) R. S. Davis, lieutenant commander (D. C.) U. S. N.

August 30, 1934: Summary: Admitted August 16, 1934, with diagnosis of pulmonary aspergillosis. The patient presented no symptoms except cough and loss of weight. The physical and X-ray findings were those of chronic interstitial pneumonitis. Smear and culture of sputum were negative for A. F. B. and fungi for the first week, but after repeated cultures on special media the fungus (Aspergillum Niger) was isolated. He has been receiving K. I. since the fungus was isolated, is gaining in weight and feels well, but in view of the chronicity of this infection he will be transferred to United States Naval Hospital for further observation, treatment, and disposition.

August 31, 1934: Transferred to United States Naval Hospital, Portsmouth, Va., for further observation, treatment and disposition. (S.) J. P. Owen, commander (M. C.) U. S. N.

Approved: K. C. Melhorn, captain (M. C.) U. S. N.

Copy of the record from the Norfolk Naval Hospital, Portsmouth, Va.—August 31, 1934: Readmitted with "Aspergillosis (pulmonary)." No. 2204. Not misconduct. C. C. Loss of weight. P. I. About 4 months ago began to lose weight and strength, developed a cough, accompanied by expectoration. Weighed 150 pounds; has lost 18 pounds. No chest pain or hemoptysis. Sputum is dark green in color, not as profuse as previously. No symptoms of toxemia. Appetite good, bowels negative. No symptoms referable to the circulatory, genito-urinary, or nervous systems. P. H. Measles; mumps; chicken-pox; typhoid fever; gonococcus infection. Denies syphilis. Married; one child; no miscarriages. Nine years' enlisted service. F. H. Negative. P. E. Skin shows several excoriations over both thighs and penis. Malnourished. Muscles flabby. Post cervical, axillary, epitrochlear and inguinal glands enlarged. Throat: Tonsils enlarged, pharyngeal irritated. Voice husky. Pupils equal and react to light and accommodation. Heart negative. Lungs expand equally, retraction of supra and infraclavicular fossa. Tactile fremitus is increased over the right infra-clavicular and mammary regions. Resonance is impaired over the left base. There are numerous moist râles and dry râles heard throughout both lungs. Abdomen negative. Extremities: Excoriations over thighs and penis. Nervous system negative. W. B. C. 8,400. Polys 50. Lymphs 41. Eosin 4. Monos 5. Urinalysis negative. Blood: Kahn negative.

September 8, 1934: T. P. R. normal. Complains of slight cough. Scant expectoration. Sputum repeatedly negative for fungi.

September 18, 1934: X-ray examination of chest: There is moderate thickening at the hilum and moderate pribronchial thickening in the central lung fields. In the right upper lobe at the level of the first and second interspace there are areas of productive infiltration, involving the middle zone and extending toward the periphery. There is a small area of productive filtration in the left upper lobe at the level of the first interspace. Findings: Small areas of productive infiltration in right and left upper lobes. Findings are not characteristic of any specific infection, but may be due to early tubercular infiltration or to fungus infection.

September 15, 1934: Sputum negative for A. F. B. W. B. C. 5,800. Polys 42. Lymphs 52. Monos 4. Eosinos 2. R. B. C. 4,190,000. Hgb. 80 percent.

September 24, 1934: Sputum negative for A. F. B.

October 1, 1934: Sputum negative for A. F. B.

October 5, 1934; W. B. C. 10,500. Polys 56. R. B. C. 4,210,000. Hgb. 80 percent.

October 6, 1934: X-ray examination of chest: There is moderate thickening at the hilum with a few small lymphoid and fibrocalcific nodules. There is moderate peribronchial thickening in the central fields of both lungs. In the upper right lobe there are small areas of productive infiltration in the first and second interspaces, involving the outer zone.

There are a few fibrotic strands in the first and second interspaces in the left upper lobe. Findings: Productive infiltration right upper lobe, suggesting incipient tuberculosis.

October 6, 1934: Sputum negative for A. F. B.

October 10, 1934: Sputum negative for A. F. B.

October 24, 1934: Patient complains of urethral discharge. History of G. C. infection urethra April 1933. Denies illicit sexual intercourse since April 1933. Stained smear—numerous pus cells, numerous gram negative intra-and extra-

cellular diplococci. Diagnosis changed to "Gonococcus infection, urethra." No. 1205. Due to own misconduct. Recurrence of old case of April 1933.

October 24, 1934: Received in G. U. service. Purulent urethral discharge present which reveals numerous pus cells and gram negative intra and extracellular diplococci. Rx: Bed, milk diet. Cod-liver oil, T. I. D.

October 25, 1934: Blood: Kahn negative.

October 29, 1934. Regular diet. Furuncle left leg with no suppuration present. Local dressing. Anterior urethral instillation of silvol 10 percent. B. I. D.

October 31, 1934: To surgery, where subcutaneous abscess left leg was incised and drained.

November 5, 1934: Urethral discharge decreasing.

November 12, 1934: Urethral discharge subsided except for morning tear.

November 20, 1934: Only occasional muco-purulent morning tear. Urethral smear showed a few pus cells. No organisms found.

November 27, 1934: Centrifuged urine reveals an occasional pus cell. No organisms. Urethral discharge subsided.

December 5, 1934: X-ray examination of chest: There are no appreciable changes since the last examination of October 3, 1934. The areas of infiltration previously described are still present. Findings: No change since previous examination.

December 7, 1934: Symptom free. Recommend X-ray of chest in about 3 months to compare with present. To duty. (S.) C. L. Burton, lieutenant (junior grade) (M. C.), U. S. N.

Approved: Harry A. Garrison, captain (M. C.), U. S. N.

March 5, 1935: Roentgenologic examination and report. Compared with the examination of December 3, 1934, there has been no change in the lung markings. The accentuation of the markings throughout the central fields of both lungs and the small area of productive infiltration in the second interspace on the right remains unchanged.

Findings: No change in the lung markings since the previous examinations. (S.) R. H. Snowden, Lieutenant-Commander (M. C.), U. S. N.

August 10, 1935: Examination at this time reveals no essential change in the condition. The patient weighs 137 pounds, has a slight cough, and is carrying on his work as a water tender to the complete satisfaction of his division officer.

BIBLIOGRAPHY

Reference Handbook of Medical Sciences, vol. VI.

Fungi and Fungous Diseases: Castellani.

Aspergillus Pneumomycosis: F. Galdi. Riforma Med. 37: 3–8, 1921.

Aspergillosis of the Lungs and Its Association with Tuberculosis. Journal American Assoc. 87: 1031–1033, 1926. Lapham.

Fungous Diseases of the Lungs. A. Q. Penta. Journal-Lancet 55: 131, March 1, 1935.

HEMOTHORAX

By Warren E. Klein, Lieutenant, junior grade, Medical Corps, United States Navy

Hemothorax is comparatively rare in the Navy during peace time. Only four cases were listed under this title in the Surgeon General's Annual Reports for the calendar years 1932 and 1933.[1] Only one case has been admitted to the San Diego Naval Hospital since 1933. The history of that case is reported below, together with comments on the general subject of hemothorax.

A seaman (J. W. W.) was admitted to the hospital on December 25, 1934, for treatment of injuries resulting from a motorcycle accident which occurred about 1 hour prior to admission. He was conscious, rational, and apparently not severely injured.

Physical examination revealed several deep abrasions of the face, swollen lips, ecchymosis about both eyes and over right mastoid. The tympanic membranes were intact, the pupils were equal and reacted normally to light and accommodation. Both fundi were negative.

Examination of the chest showed two points of tenderness over the left sixth rib, one at the vertebral margin, the other at the anterior axillary line. No crepitus could be elicited. Heart and lungs were negative and the blood pressure was 110/66 with a pulse rate of 90.

There were multiple small abrasions of both hands, with swelling and deformity of the left hand, suggesting fracture of the fourth and fifth metacarpal bones. There was no evidence of paralysis of skeletal muscles.

The impressions when first seen were: (1) Multiple abrasions; (2) probable fracture of the left fourth and fifth metacarpals; (3) possible fracture of the skull; (4) possible fracture of the left sixth rib.

About 12 hours after the injury the respirations became embarrassed, the color became pale and sallow and the pulse rapid and thready, accompanied by a fall in blood pressure.

When the pulse rose to 160 and the blood pressure fell to 90/60, 1,000 cubic centimeters of 5 percent glucose were given intravenously. This was followed by a rise of the blood pressure to 108/76 and a fall of the pulse rate to 144.

On examination for visceral injury, the left side of the chest was found to be flat to percussion, with very distant breath sounds and muffled voice sounds. There was no evidence of intra-abdominal injury.

Bedside X-ray of chest taken at this time showed the entire left side of the chest radio-opaque, with the heart and mediastinal shadows displaced to the right.

On the basis of the foregoing findings, it was concluded that a massive hemorrhage into the left pleural cavity had occurred.

The blood pressure continued to fall to 85/54 and the pulse rose to 180, becoming very fine and difficult to palpate. The patient vomited several times, the vomiting being projectile in character.

A transfusion of 500 cubic centimeters of whole blood was given by the Scannel method, followed by an immediate rise in the blood pressure to 100/60 and a drop in the pulse rate to 126. The pulse was much stronger following the transfusion.

The red blood count prior to transfusion was 3,470,000, hemoglobin 80 percent.

W.B.C	23,75	Seg	67
Juv	3	Lymph	12
Bands	15	Monos	3

Two days later the white blood count was 21,300, with little change in the shift, and 3 days after admission the red blood count was 3,610,000, hemoglobin 75 percent, white blood count 17,000 with approximately the same shift.

The day following the transfusion and subsequent days brought marked progressive improvement in the general condition, except for gradually rising temperature which reached 104° F. on January 9, 1935. Since that time the temperature slowly subsided, reaching normal by February 7.

The patient complained of frequent frontal headaches which responded readily to aspirin.

Further X-ray examination showed the left side of the chest remaining radio-opaque from apex to base with the mediastinal shadow displaced to the right. X-ray of the skull revealed a simple linear fracture, right parietal and temporal

bones, and X-ray of the left hand showed impacted fracture of the distal head, fifth metacarpal.

The fracture of the metacarpal was reduced under local anesthesia and the hand placed in a cast with skin traction on the finger.

The patient was kept strictly in bed, being allowed to sit up with a bed rest.

On January 5, 50 cubic centimeters of old blood were aspirated from the left pleural cavity. A culture made from the specimen produced negative results.

On January 7, 75 cubic centimeters of old blood were aspirated, and a like quantity on January 10.

Aspiration was repeated on January 12, with removal of 700 cubic centimeters of old blood, and again on January 14, when 1,900 cubic centimeters were removed. The patient showed no sign of shock or respiratory embarrassment following aspiration. Examination following this aspiration showed resonance as low as the fourth left interspace and breath sounds distinguishable as low as the fifth left interspace.

Fluoroscopy and radiographic examination following the last aspiration demonstrated the heart and mediastinal shadows central, with about 40 percent expansion of the left lung and considerable fluid remaining. On January 27 the patient was allowed up in a wheel chair. During the first week in February he was allowed up and about.

The common causes ascribed to hemothorax are rupture of an aneurysm or of a vessel as an intercurrent incident of disease, and laceration of an intercostal artery, an internal mammary artery, or of the lung.

Preexisting aneurysm or disease may be ruled out at once in our case by the history and subsequent X-ray findings. In hemorrhage from the lung, hemostasis usually occurs spontaneously after partial mechanical compression of the lung. In such instances, hemostasis is favored by the comparatively low blood pressure in the pulmonary artery (25 to 30 millimeters of Hg). As the hemorrhage was very extensive in this case and continued until the left lung field was entirely radio-opaque with the mediastinal contents displaced markedly to the right, it is unlikely that laceration of the lung was responsible, unless a large vessel near the hilus was involved, and laceration of an intercostal or internal mammary vessel appears much more reasonable as the source of the hemorrhage.

The signs of severe hemorrhage, appearing some 12 hours after the injury, suggest that some movement of the patient, probably during physical examination, precipitated the hemorrhage from a previously injured vessel.

Jerome Head (2) reports a case in which the symptoms of intrapleural hemorrhage first appeared 6 weeks after an injury which had seemed negligible at first. Brown and Debenham (3) report a case developing on the second day following injury, on being moved to the X-ray room.

The logical inference is that cases of crushing injuries, or other severe trauma to the chest, may be considered potential hemothorax patients and should not be moved, except for necessary diagnostic procedures.

According to Dolley (4), thoracic shock, which almost invariably follows severe chest injuries, may cause symptoms closely resembling those of extensive intra-pleural hemorrhage and must be differentiated from hemothorax. He administers morphine hypodermically in full doses, causing subsidence of the symptoms of thoracic shock, while those due to hemorrhage are only slightly affected, or not at all.

Most authorities agree that diagnostic aspiration is indicated when there is any doubt as to the presence of blood in the pleural cavity. We did not find this necessary, as physical examination of the chest, checked by X-ray, convinced us of the diagnosis. When a portable X-ray is available, the diagnosis can be confirmed by this means, and we believe aspiration need only be performed when X-ray is not accessible.

Intravenous fluids are indicated, and in the presence of such alarming symptoms as our case presented, transfusion of whole blood is believed to be a life-saving measure. Brown and Debenham report several cases of hemothorax autotransfused using the fluid aspirated from the pleural cavity, followed by uneventful convalescence. One wonders, however, if early removal of a large quantity of fluid in a case where the hemorrhage arises from a lacerated vessel would be followed by further, possibly more extensive hemorrhage and collapse. In our case, the complete filling of the left pleural cavity, with marked displacement of the mediastinal contents to the right suggests that hemorrhage continued until all available space was filled, and ceased only when intrapleural pressure equaled the pressure within the injured vessel. If so, aspiration could only have resulted in further damage. Evarts Graham (5) considers 2 days to be a safe period after which a hemothorax will not increase.

We considered removal of 1,900 cubic centimeters of fluid by thoracentesis on the twentieth day, without producing signs of pulmonary embarrassment, rather heroic treatment, but find that Sandison and Elkin (6) report aspiration of 1,400 cubic centimeters on the first day, 1,400 cubic centimeters on the second day, and 3,400 cubic centimeters on the fourth day, without ill effect.

Blow bottles are useful following aspiration and during absorption, to aid in reexpansion of the lung.

Eugene Grunwald (7) has shown that normal blood is inhibited from coagulating by the addition of relatively small quantities of blood aspirated from the pleural cavity. This bears out the findings of American clinicians, who report that blood in the pleural cavity shows a marked tendency to remain in the liquid state. This is conceded to be due to dilution with an effusion of fluid, which Evarts Graham states almost invariably occurs sooner or later.

Hemothorax patients eventually absorb the greater part of the fluid, sometimes all of it, but this desirable result is not hastened by

repeated aspiration. Sandison and Elkin favor thoracentesis only for pain and dyspnea. However, where absorption is slow, we believe that aspiration should be employed at intervals.

The end result is usually recovery. Empyema has been known to develop following aspiration, but so rarely that it is not considered a valid objection to aspiration.

SUMMARY

A brief outline for the management of hemothorax cases follows:

1. Early recognition of symptoms.
2. Morphine to differentiate from thoracic shock.
3. Physical examination of the chest, confirmed by X-ray (bedside), if available, or by aspiration of a small quantity of fluid.
4. Avoid moving or disturbing patients who have suffered severe trauma to the thorax.
5. Intravenous fluid. Transfusion, if severe symptoms develop.
6. Late aspiration, after the second day, and as indicated for the relief of pain or dyspnea. Repeat if absorption is delayed.
7. Use of blow bottles to favor reexpansion.

REFERENCES

(1) Annual Report of the Surgeon General, U. S. Navy, 1932, and Statistics of Diseases and Injuries in the United States Navy for the Calendar Year 1933.

(2) Head, Jerome: Injuries of the Thorax. Arch. Surg. 25: no. 3, September 1932.

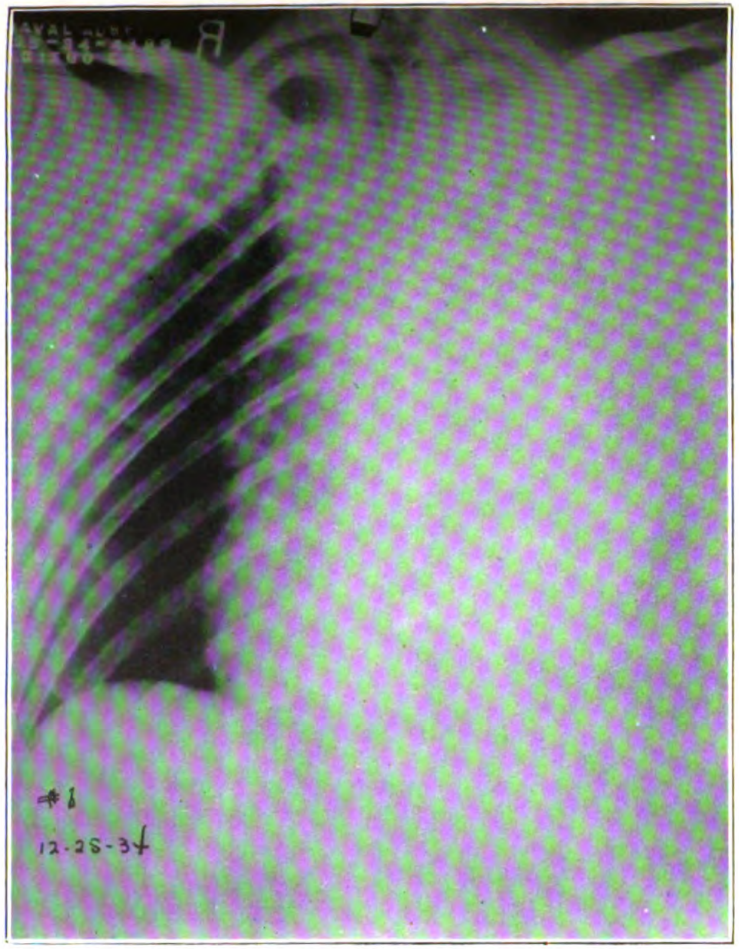
(3) Brown, A. Lincoln, and Debeham, Martin Warren: J. A. M. A. 96: 1233, April 11, 1931.

(4) Dolley, Frank S.: Calif. and West, Med. 27: no. 3, September, 1932.

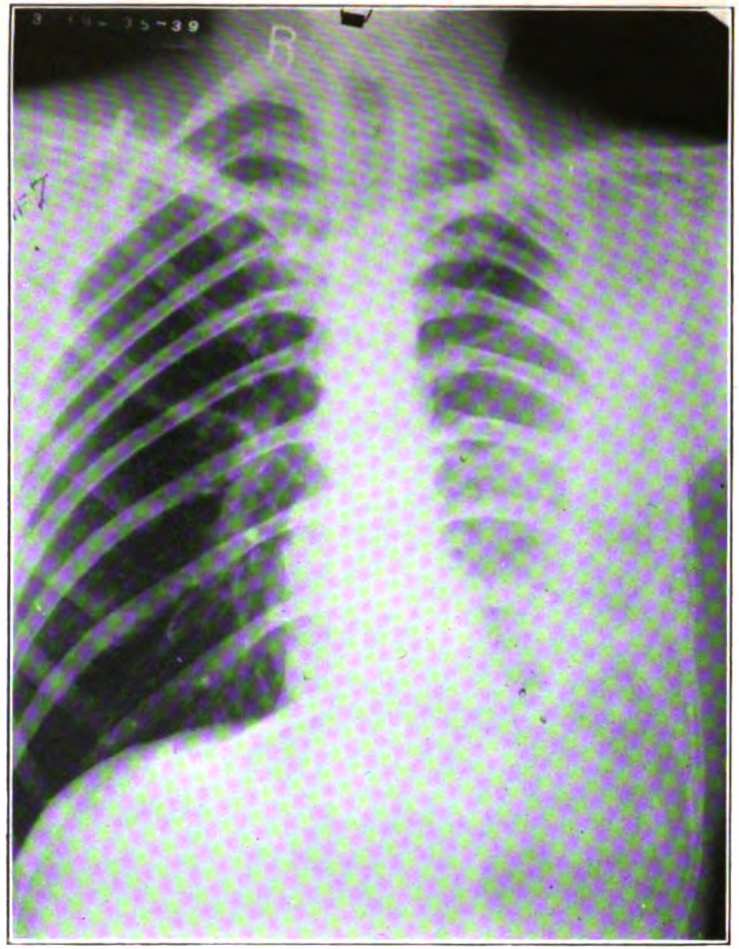
(5) Graham, Evarts: Surgical Diagnosis, 1:16. 3:36–138, 1930.

(6) Sandison, J. Calvin and Elkin, Daniel C.: Penetrating Wounds of the Chest—with Studies on Experimental Hemothorax, The Jour. of Thor. Surg. 11: no. 5, June 1933.

(7) Grunwald, Eugene: Hemothorax Spontane Chez un Malade Porteur de Pneumothorax Artificiel, Revue de la Tuberculose, 4 Serie, Tome 1, no. 6, Juin 1933.



12-25-34.—NOTE DISPLACEMENT OF MEDIASTINAL CONTENTS TO RIGHT AND LEFT SIDE OF CHEST RADIO-OPAQUE.



3-19-35.—EXPANSION RETURNING. MEDIASTINAL SHADOW CENTRAL.

NAVAL RESERVE

MEDICAL CORPS

PROMOTIONS. FOURTH QUARTER, 1935

Lt. Charles Van Epps Waggoner, M. C.-V. (G), U. S. N. R. Promoted from lieutenant (jr. gr.) M. C.-V. (G), U. S. N. R., June 28, 1935.

Lt. Henry Easton McMahon, M. C.-F., U. S. N. R. Promoted from lieutenant (jr. gr.), M. C.-F., U. S. N. R., October 24, 1935.

Lt. Beryl C. Shearer, M. C.-V. (S), U. S. N. R. Promoted from lieutenant (jr. gr.), M. C.-V. (S), U. S. N. R., December 5, 1935.

Lt. James E. Amiss, M. C.-V. (G), U. S. N. R. Promoted from lieutenant (jr. gr.), M. C.-V. (G), U. S. N. R., December 2, 1935.

Capt. John A. McGlinn, M. C.-V. (G), U. S. N. R. Promoted from commander, M. C.-V. (G), U. S. N. R., December 10, 1935.

Lt. John L. Cardwell, M. C.-F., U. S. N. R. Promoted from lieutenant (jr. gr.), M. C.-F., U. S. N. R., December 3, 1935.

Lt. Clyde L. Welsh, M. C.-F, U. S. N. R. Promoted from lieutenant (jr. gr.), M. C.-F., U. S. N. R., December 9, 1935.

RETIREMENT OF CAPT. JOHN A. McGLINN, MEDICAL CORPS, UNITED STATES NAVAL RESERVE FORCE

Captain John A. McGlinn, M. C.-V. (G), of the United States Naval Reserve, will be transferred to the honorary retired list on January 1, 1936, having reached the statutory age for retirement. Captain McGlinn entered the Naval Reserve in 1917, and served during the war at the United States Naval Hospital, League Island, Pa., and on the U. S. hospital ship *Comfort*, throughout the World War. A specialist in surgery and gynecology, he was operating surgeon at that hospital for some time.

RESERVE OFFICERS CLASS IN AVIATION MEDICINE

A class in aviation medicine is being conducted at the third naval district for Reserve medical officers. The class is in charge of Lt. Comdr. J. F. Neuberger (M. C.), U. S. N., and is being held at Cornell University.

The class started on the first of September and will last for a period of 6 months. The lectures are being given by officers of the Medical Reserve Corps who are professors or assistant professors connected with different institutions in New York.

The practical work is being given by Lt. Comdr. Neuberger at headquarters, third naval district, where all candidates for United States Naval Aviation are examined.

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Identification of Reserve Officers

(Photograph, Oct. 10, 1935)

1. Capt. R. H. Gleason
2. First Lt. J. L. Deuterman
3. Capt. F. W. Butler
4. First Lt. R. H. W. Drechsel
5. Maj. G. B. Eusterman
6. First Lt. R. L. Butler
7. Capt. H. O. Whitaker
8. First Lt. W. S. Riley
9. Capt. B. R. Galbraith
10. First Lt. L. K. McMurtry
11. Lt. Col. W. N Lynn
12. Maj. J. J. Catlin
13. First Lt. F. K. Sewell
14. Capt. J. D. Moore
15. Lt. Col. W. B. Cantrell
16. Maj. R. H. Harrison, Jr.
17. Lt. Comdr. L. Humphreys
18. Col. Kent Nelson
19. Brig. Gen. C. H. Mayo
20. Maj. Gen. C. R. Reynolds
21. Col. L. B. Wilson
22. Lt. Col. F. L. Smith
23. Comdr. P. Richmond
24. Comdr. J. J. White
25. Capt. C. G. Sanner
26. Capt. E. A. Swenson
27. Lt. H. A. Erickson
28. Lt. Col. E. G. Condit
29. Lt. Col. W. H. Henry
30. First Lt. J. L. Anderson
31. Lt. (Jr. Gr.) J. H. Costenbader
32. Lt. Col. J. W. Sherwood
33. Capt. S. F. Seeley
34. Second Lt. J. H. Hopkin, Jr.
35. First Lt. B. A. Welch
36. First Lt. M. F. Williams
37. Maj. J. R. Hall
38. Capt. R. C. Hunter
39. First Lt. G. E. Martin
40. First Lt. W. H. Doyle
41. First Lt. L. L. Cooper
42. First Lt. F. E. Butler
43. First Lt. W. H. Newlin
44. First Lt. C. W. Plowman
45. First Lt. P. W. Schriber
46. Maj. F. M. Sanger
47. First Lt. L. C. Jensen
48. Lt. Comdr. A. Wildman
49. First Lt. S. G. Maraventano
50. Lt. Comdr. G. C. DeGrandpre
51. First Lt. I. J. Berkowitz
52. Capt. G. C. Struble
53. First Lt. O. W. E. Nowlin
54. First Lt. D. C. Nelson
55. Capt. G. A. Woodruff
56. First Lt. M. F. Axtholm
57. First Lt. J. Present
58. First Lt. M. E. Barrett
59. Capt. R. W. Henderson
60. Lt. Comdr. N. O. Byland
61. Capt. C. T. Root
62. Maj. J. C. Hubbard
63. Lt. I. L. V. Norman
64. Lt. Comdr. T. B. Magath
65. Capt. W. E. Ryan
66. First Lt. R. N. Washburn
67. First Lt. R. E. Bennett
68. First Lt. E. E. Walcher
69. First Lt. L. E. Kienholz
70. Capt. J. R. Daly
71. Maj. M. W. Lyon, Jr.
72. Capt. W. H. Christian
73. First Lt. D. A. Hoxie
74. First Lt. B. J. Macauley
75. First Lt. M. C. Guernsey
76. First Lt. S. J. Campbell
77. First Lt. M. Griffin
78. Capt. F. E. Hudson
79. First Lt. W. J. Baumgartner
80. First Lt. C. H. Dolph
81. First Lt. A. B. Peterson
82. Maj. J. H. Dameron
83. First Lt. B. M. Knight, Jr.
84. Capt. J. B. Polka
85. Lt. Col. T. J. Ashton
86. First Lt. G. R. Kimbro
87. Capt. A. W. Para
88. First Lt. A. E. Boysen
89. First Lt. O. L. Johns
90. First Lt. L. A. Haen
91. First Lt. J. L. Sanchez
92. First Lt. A. B. Stephens
93. Lt. Col. A. Butler
94. First Lt. F. M. Douglas
95. First Lt. W. M. Blair
96. Capt. E. F. Etter
97. First Lt. J. C. Fitzpatrick
98. First Lt. J. A. Harwig
99. First Lt. J. M. Connor
100. First Lt. P. H. Hemphill
101. First Lt. J. R. Henning
102. First Lt. R. L. Ledbetter
103. First Lt. C. H. Finney
104. First Lt. W. W. Alexander
105. Capt. W. G. Lewis
106. Capt. L. S. Hayes
107. First Lt. H. D. Mosier
108. First Lt. V. T. Amend
109. First Lt. C. A. D. Beer
110. First Lt. J. T. Humphries
111. First Lt. J. E. Beasley
112. First Lt. J. C. Kerr
113. First Lt. R. H. Cantrell
114. Lt. S. Peluse
115. First Lt. H. C. Kraft
116. First Lt. A. J. Kafka
117. First Lt. R. D. Fairchild
118. First Lt. S. B. Lovelady
119. Maj. A. W. Collins
120. First Lt. A. Behrend
121. Capt. A. N. Deaton
122. First Lt. H. K. Speed
123. Lt. G. W. Simpson
124. Lt. B. I. Brindley
125. First Lt. J. W. Reid
126. Maj. S. M. Browne

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NOTES AND COMMENTS

WILLIAM GRIER, SURGEON GENERAL, UNITED STATES NAVY, 1877–78

William Grier, the ninth chief of the Bureau of Medicine and Surgery and the fifth to hold office as Surgeon General of the Navy, was born in Ireland on October 5, 1818, and came to this country in childhood with his parents. He was appointed from Maryland on March 7, 1838, as an assistant surgeon, his first service being on the sloop *Cyane* in the Mediterranean Squadron. After service at the naval hospital, New York, and the North Pacific Squadron he was promoted to surgeon on April 14, 1852, and appointed fleet surgeon of the North Pacific Surveying Expedition from 1853 to 1856. This expedition was designed to supplement the exploration of the famous Wilkes Expedition in the southern Pacific. During the Civil War, Dr. Grier was attached to the sloop *Macedonian*, and from 1863 to 1865 to the hospital *Pinckney* at Memphis, Tenn., the temporary naval hospital of the Western Flotilla on the Mississippi. After the Civil War, he served at the naval hospital, Annapolis, and as a member and the president of the board of medical examiners at Washington. He was appointed chief of bureau by President Grant on February 3, 1877, and Surgeon General with the relative rank of commodore. Surgeon J. B. Parker was assistant chief of bureau during his term of office. Dr. Grier retired on October 5, 1878. His death occurred at Washington, D. C., on January 11, 1911, at the advanced age of 93 years.

The career of this Surgeon General is a rather remarkable one. Coming as an immigrant from Ireland and rising to a high office in the Navy, he must have possessed marked abilities. In the span of 93 years he saw the changes in the nineteenth century that completely altered conditions of life—the coming of railroads, telegraphs, and steamships. He entered the Navy in the administration of President Van Buren and died in that of President Taft.

The advent of the anaesthesia, asepsis, the germ theory of disease, diphtheria antitoxin, the X-ray, and all the other revolutionary changes in his own profession, were witnessed by him. In the Navy, he saw the change from sail to steam, from wood to steel, from broadside to turret, from the old wooden line of battleship to the dreadnaught.

ARTICLES OF SPECIAL MERIT PUBLISHED IN THE NAVAL MEDICAL BULLETIN IN 1935

It has been customary for a number of years for a board to select from the articles published in the NAVAL MEDICAL BULLETIN during each calendar year, those having special merit. The writers of articles thus singled out then receive from the Surgeon General a letter of appreciation which, of course, is highly valued by the recipient and may be made a part of his official record. In recent years the Postgraduate Board has excercised the function of selecting these articles. In 1935 the following authors were selected to receive special letters from the Surgeon General:

Paul F. Dickens, lieutenant commander, Medical Corps, United States Navy, and Charles Armstrong, surgeon, United States Public Health Service, for the article entitled "Benign Lymphocytic Choriomeningitis (Acute Aseptic Meningitis). A new Disease Entity", published in the October number.

Carl J. Robertson, lieutenant commander, Medical Corps, United States Navy, for the article entitled "A Comparative Study of the Measurement of the Speed of Adjustment of the Eye to Near and Far Vision", published in the April number.

James A. Hawkins, D. Sc., Charles W. Shilling, lieutenant, Medical Corps, United States Navy, and Raymond A. Hansen, lieutenant, United States Navy, for the article entitled "A Suggested Change in Calculating Decompression Tables for Diving", published in the July number.

THE PREVENTION OF DENTAL CARIES

There have been three important studies of the Navy ration in the present century. The first of these, made more than 30 years ago by Gatewood, was concerned chiefly with the total caloric requirements of the ration and the ratio of the various components of the ration. The second, carried out by Phelps more than a decade ago, was largely concerned with mineral deficiencies. In this number of the Naval Medical Bulletin is a study by Jones and Crosland of the acid base balance and vitamin content of the Navy ration. Dr. Martha Jones, the well-known American student of nutrition, has been a pioneer in this field, and has particularly directed attention to the importance of the acid base balance and vitamin content in the diet in relation to dental caries, pyorrhea, and to the general health. In collaboration with Lieutenant, junior grade, George N. Crosland, Dental Corps, United States Navy, she has made an extensive study of the naval dietary with particular reference to these factors. While evidence is insufficient to accept all the conclusions and inferences of

WILLIAM GRIER.
Surgeon general of the Navy 1877–78.

her work, if they are, in the main, correct, this study should yield important results. Miss Jones and her co-worker believe that dental caries at least, and possibly pyorrhea, can be largely prevented by the proper maintenance of the acid base balance and the vitamin content of the diet. If this belief is correct and this study proves to be the dietetic key to the cause of dental caries and pyorrhea, it would be a great triumph in preventive dentistry.

THE ELEVENTH REVISION OF THE UNITED STATES PHARMACOPOEIA

The eleventh United States Pharmacopoeia does not contain a number of articles which were official in the tenth revision. One hundred and thirty titles were dropped, including such drugs and preparations as buchu, cinchophenum, cubeba, elaterinum, and such well-known fluid extracts as belladonna foliorum, rhei, hydrastis, hyoscyami, rosae, scillae, and uva ursi. Liquor aresni et hydrargyri iodidi, Donovan's solution, is gone. Mel rosae, the old honey of rose, was deleted, as well as pepo, rosa, senega, syrupus zingiberis, tinctura gambir composita, ulmus, and unguentum iodoformi.

There are 58 additions. Some of the more important of these are scarlet fever antitoxin, scarlet fever streptococcus toxin, diptheria toxoid, diptheria toxin for the Schick test, rabies vaccine, typhoid vaccine, typhoid and paratyphoid vaccine, antimeningococcic and antipneumonic serum type I, ephredrine, and ephedrine hydrochloride, carbon dioxide, extract of liver, corn oil, and irradiated ergosterol solution. Soluble fluorescin is an addition of particular interest to ophthalmologists. An addition that recalls William Withering, who usually prescribed the powdered leaves of digitalis, "dried and rubbed up into a beautiful green powder", is digitalis pulverata, or the powdered leaf of the foxglove.

The oil of roses is an addition in the interests of elegant pharmacy. Extensively used, as are all the additions, it now becomes possible to establish pharmacopoeial standards for them.

There are a number of changes in both official Latin and English titles. Mistura Glycyrrhizae Composita now becomes Mistura Opii et Glycyrrhiza Comp. The old Oleum Cadinum becomes Pix Juniperi. Pituitarium becomes pituitarium posterius, and the old compound licorice powder of the U. S. P. X. and many previous revisions, is now the compound senna powder, Pulvis sennae compositus.

As directed by the U. S. P. convention, the board of trustees has also fixed the date when the standards of the new Pharmacopoeia shall become official, superseding the tenth revision. This date is June 1, 1936.

THE WELCOME PRIZE ESSAY FOR 1936

The subject for the Wellcome prize for 1936 is "The Importance of Coordinating the Military and Naval Medical Services with the Civilian Medical Profession."

The prize consists of $500 in cash and a gold medal suitably inscribed. The essay may contain from 3,000 to 10,000 words, and must be in the hands of the committee charged with the award of the prize by August 15, 1936. Each person submitting an essay in the competition places on it a nom de plume and the same on a sealed envelope in which the name and address is given. The essays are read and the winners selected before the envelope containing the name of the winning competitor is opened. Five copies of the essay must be furnished to facilitate the reading by the various judges of the contest. All members of medical services of the Army, Navy, United States Public Health Service, National Guard, Organized Reserves, and Veterans' Administration are eligible to participate in the contest. While not essential it is desirable that contestants be members of the Association of Military Surgeons. The essays should be mailed to the secretary of the Association of Military Surgeons, Army Medical Museum, Washington, D. C., in time to reach him by August 15, 1936.

THE SPINAL FLUID IN SYPHILIS

The spinal fluids obtained from 47 cases of syphilis occurring among naval personnel were examined by Lt. Comdr. J. G. Dickson, Medical Corps, United States Navy, for the object of determining the presence of pathology in the central nervous system in the ordinary case of syphilis treated in the Navy. The duration of the disease ranged from 1 to 17 years with an average of approximately 6 years. All cases had received the treatment usually given in the Navy. During the 12 to 18 months prior to the tests, observation as to the Kahn blood reaction had been very close and treatment, where indicated, very intensive. Nearly all the patients were blood Kahn negative with the spinal punctures done. Examination of the spinal fluids were made by several laboratories. The reports were practically 100 percent negative. Kahn reaction and colloidal gold curve were negative in each instance; globulin was negative except there was an occasional plus-minus. Cell counts were either reported as negative or where given there were only 2 instances of a count over 10, one 14, and one 17.

These facts are at variance with the reports of some other observers and, of course, the series was too small to draw any important conclusions, and the real incidence of spinal fluid abnormalities among syphilitics in the Navy must await the results of more studies. The results, however, of Dickson's work would indicate the extension of the disease to the nervous system was relatively limited.

IMPORTANT ANNUAL MEETINGS IN 1936

The following are some important meetings of medical and scientific societies during 1936:

The American Medical Association, Kansas City, Mo., May 11–15, 1936.

The American College of Physicians, Detroit, Mich., March 2–6, 1936.

The American College of Surgeons, Philadelphia, October 19–23, 1936.

The Association of Military Surgeons, Detroit, Mich., October 29–31.

The American Public Health Association, New Orleans, La., in October 1936.

The American Pharmaceutical Association, Dallas, Tex., the third week of August 1936.

The American Dental Association, San Francisco, Calif., July 13–17, 1936.

The American Association for the Advancement of Science, Rochester, N. Y., in June 1936; Washington, D. C., December 28, 1936 to January 3, 1937.

The Society of Tropical Medicine, Baltimore, Md., November 17–20, 1936.

The Academy of Ophthalmology and Otolaryngology, New York, N. Y., in October 1936.

The American Association of Pathologists and Bacteriologists, Boston, Mass., April 9–10, 1936.

The American Nurses Association, Los Angeles, Calif., June 22–28, 1936.[1]

THE AMERICAN BOARD OF OPHTHALMOLOGY—NOTICE OF EXAMINATIONS IN 1936

The American Board of Ophthalmology has requested that a notice of their examinations for 1936 be printed in the United States Naval Medical Bulletin. The examinations will be held at Kansas City, Mo., May 11, 1936, at the time of the meeting of the American Medical Association in New York City in October, and at the time of the meeting of the American Academy. All applications and case reports must be filed at least 60 days before the date of examination. For further information, syllabuses and application forms, please write to Dr. Thomas D. Allen, assistant secretary, 122 South Michigan Avenue, Chicago, Ill.

ADVANCES IN MEDICINE AND THE MEDICAL SCIENCES DURING THE YEAR 1935 [1]

The following is a brief résumé or calendar of the more recent advances in the clinical branches of medicine, as well as the medical sciences. An attempt has been made to confine it to discoveries or important advances that were made during the past year, though this has not always been possible as some of the work extends several years back and has only become recognized during 1935. Furthermore, it is naturally possible in so brief a compass as a few pages to mention but the most outstanding achievements. With work so recent, too, it is difficult to appraise with absolute accuracy that which will stand the test of future experience.

The calendar of recent advances in the medical sciences published in the April 1933 and April 1934 numbers of the BULLETIN has proved so popular that it has been decided to repeat it each year. As in the résumé of last year, only the most outstanding events are mentioned and as far as possible limited to advances made in 1935, though this latter purpose cannot always be realized as the original work may have been done on a subject in previous years and brought to a final and successful conclusion in 1935. Of course, it is also obvious that when discoveries so recent have to be appraised without the trial of use which time alone can give, errors of commission or omission may naturally result.

MEDICINE

The value of choline as a new vitamin essential for liver function is pointed out by the work of Best, Hershey, and Huntsman at the University of Toronto. Choline is produced by the pancreas and is believed to be a factor in the control of diabetes. Dr. C. H. Best, the senior in this investigation, was the codiscoverer of insulin.

Benzedrine has been reported as of value in the treatment of narcolepsy. Work was done by Prinzmetal and Bloomberg at the Boston City Hospital and Harvard Medical School.

Electrical impulses generated by the brain, similar in character to those detected by the electrocardiograph in the heart, have been detected in studies made by several investigators. These impulses vary in sleep and during the convulsions of epilepsy.

Two of our own Government physicians, Armstrong of the United States Public Health Service and Dickens of the United States Navy,

[1] This material was prepared with the assistance of the staff of the Naval Medical School, Washington, D. C.

have definitely discovered that benign lymphocytic meningitis was caused by the virus of Armstrong.

Armstrong discovered the virus, and Dickens at the United States Naval Medical School worked on the clinical aspects of the disease. This is apparently a new disease entity which Armstrong and Dickens have designated as benign lymphocytic choriomeningitis.

Enterogastrone, a new hormone produced in the mucosa of the upper intestinal tract, causes inhibition of stomach activity, and it is believed that it may be of value in the treatment of gastric ulcer. This work was largely done by Ivy of Northwestern University Medical School.

Evidence of a toxic origin for pernicious anemia was produced by the work of Wakerlin and Bruner at the University of Louisville School of Medicine. They showed that kidney excretions from untreated pernicious anemia patients produced a decrease in the reticulocytes in experimental animals.

The cultivation outside the body of what appears to be the virus of influenza is reported by Francis and Magill of the Rockefeller Institute.

SURGERY

Successful treatment of undescended testicle by the use of the gonadotropic hormones from the urine of pregnant women, is reported by Spence and Scowen of London. This would appear to be a great advance over the usual operative treatment.

Continued advance in surgery of the sympathetic system in the treatment, not only of heart disease and hypertension, but of gastrointestinal, kidney, and vesical conditions, has been made during the past year. The Rockefeller Institute, the Mayo Clinic, and the University of Michigan have been centers for much of this work.

The successful treatment of a number of cases of Pick's disease, by resection of a part of the pericardium, has been reported. This work was reported from the Massachusetts General Hospital at Boston, by White and Churchill.

OBSTETRICS AND PEDIATRICS

Many years of research by numerous investigators have at last resulted in the isolation of the active principle in ergot which acts to produce contraction of the uterus.

Prolactin, a hormone from the pituitary gland, has been demonstrated to be the agent causing the co-called maternal instinct in animals. This study was made by Riddle, Lahr, and Bates of the Carnegie Institute at Washington.

The smallest viable baby reported in medical literature was born this year at El Paso, Tex.; weight at birth was 1 pound.

The treatment of stuttering by exercises which consist essentially in walking on all fours for a certain period, has been suggested as a result of the work of Geniesse of the University of Michigan.

It is a widespread belief that children learn faster than adults. This theory has been disproved by the case work of Thorndyke of Columbia, who has demonstrated that adults learn faster than children and a man 65 can learn more per hour than he could at the age of 10.

EMBRYOLOGY

The Nobel prize in medicine was given this year to Dr. Hans Spemann, of Freiburg, Germany, for work in experimental embryology. Studies particularly concerned the so-called "leader" cells and their work in the development of embryonic organs. Their function seems to be exercised through a chemical activator. It is of interest to remember that the Nobel prize in medicine in 1933 was given to an American embryologist, Dr. Thomas Hunt Morgan.

HISTOLOGY

At least one source of the blood platelets was discovered by Howell of Johns Hopkins University. They are apparently formed by certain giant cells in the lungs.

PHYSIOLOGY

Important functions of the pineal gland have been described by Rowntree and associates. It appears that the gland is definitely concerned with control of body growth and sexual development.

Control of the reticulo-endothelial system appears to be a function of the pituitary gland according to the work of Dodds and Noble of London.

DENTISTRY

A new anesthetic, which can be applied to the dentine in order to prevent pain during drilling and some other dental procedures, has been discovered by Hartman of Columbia University.

Further studies in the relation of nutrition to dental caries has also been a feature of dental research in 1935.

RADIOLOGY[1]

Further study of short-wave therapy was a feature of radiology during the past year. Improvement and increase in size of X-ray tubes has continued. A tube developed in the Massachusetts Institute of Technology by Van der Kraaff is intended for use with a 7-million-volt electro-static generator.

The Nobel prize in chemistry for 1935 was received by Prof. J. Joliot and his wife Mme. Irne Curie-Joliot, the daughter of Mme.

[1] Prepared with the assistance of Lieut. Comdr. Otis B. Spalding, Medical Corps, U. S. Navy.

Curie, for work on the artificial production of radioactive elements. As Mme. Curie shared the Nobel prize with M. Becquerel in 1911, and again with her husband in 1902, this makes the third prize received by this family for research on radium.

Roentgen therapy is being used more extensively today than ever before. Improvements in apparatus and increased knowledge as to application has established it as a therapeutic agent of the greatest value.

PROGRESS IN THE STUDY OF CANCER

Relation of heredity to cancer is indicated by a study of simultaneous occurrence of cancer of the stomach in identical twins. This study was reported by Militzer of the Massachusetts Department of Public Health.

Immunity of mice to mice cancer was reported as having been developed by the inoculation of cancer cells into the skin. Reported by Besredka and Gross of the Pasteur Institute, Paris.

Several new hydrocarbons capable of producing cancer were described by various observers during the past year.

Connective tissue tumors were found to contain sex hormones which affected the growth of the tumors. Reported by Geschickter and Lewis of Johns Hopkins University.

Leukemia, considered by some a malignant disease, is apparently transmitted through inheritance on the maternal side, indicated by breeding experimental mice. Reported by MacDowell of Carnegie Institute.

BOOK NOTICES

Publishers submitting books for review are requested to address them as follows:

The Editor,
United States Naval Medical Bulletin,
Bureau of Medicine and Surgery, Navy Department,
Washington, D. C.
(For review.)

PRINCIPLES AND PRACTICE OF UROLOGY, by *Frank Hinman, A. B., Leland Stanford University; M. D., Johns Hopkins Medical School; Clinical Professor of Urology, University of California Medical School.* 1,078 pages with 513 illustrations and 48 tables. W. B. Saunders Co., Philadelphia, 1935. Price, $10.

This book has many features that commend it so highly that it can be ranked without qualification as standing among the most valuable of texts in urology.

It is unique in its departure from the time-worn and conventional arrangement of subject matter and literary style. It goes more profoundly into the fundamentals of the science of urology than any one-volume text yet produced. In fact, the first 250 pages are occupied by a most masterly treatise on the comparative anatomy, the embryology, and the structure and function of the urogenital organs.

The remaining 800 pages make up a very concise and extremely lucid exposition of exactly the kind of information that is sought by the general practitioner and the student. Between all the lines one can see the marks of genius of a profound scholar and scientist, a great teacher, a master urologist, and a human who knows humans.

One of the fine features in this book—often lacking in other urological texts—is the section on venereal infections, gonorrhea, syphilis, lymphogranuloma, and granuloma inquinale. This section constitutes a splendid résumé of the most modern concepts of these diseases and could serve as a reliable guide in their diagnoses and treatment.

THE STOMACH AND DUODENUM by *George B. Eusterman, M. D., Head of Section in Division of Medicine, the Mayo Clinic,* and *Donald C. Balfour, M. B., M. D., Head of Section in Division of Surgery, the Mayo Clinic, and Members of the Staff, the Mayo Clinic and the Mayo Foundation for Medical Education and Research.* Cloth. 958 pages, with 436 illustrations. W. B. Saunders Co., Philadelphia and London, 1935. Price, $10.

An excellent book covering all phases of diseases of the stomach and duodenum, and their relationship to other diseases. Eusterman and Balfour, with their wealth of experience, are particularly well qualified to write on both the medical and surgical aspects of the subject. Their colleagues, at the Mayo Clinic and the Mayo Foundation for Medical Education and Research, have undoubtedly contributed a mass of medical knowledge and clinical experience which has helped greatly in giving this work its broad and balanced viewpoint. The various branches of the subject have been smoothly developed and well coordinated. The most recent diagnostic methods are fully described and the chapter on roentgenologic diagnosis is excellent. The illustrations by Russell Drake and Eleanora Fry merit special praise.

The student and general practitioner will find this book a clear and concise textbook, while the gastroenterologist and surgeon will also find it an authoritative reference work.

HOSPITAL ORGANIZATION AND MANAGEMENT, by *Malcolm T. Mac Eachern, M. D., C. M., D. S. C. Associate Director of the American College of Surgeons, and Director of Hospitals.* Physicians Record Co., Chicago, 1935. Price, $7.50.

Twenty-two years of experience in administering hospitals, in making surveys of hospitals, and in directing the hospital standardization program of the American College of Surgeons has given the author a wealth of experience.

In this volume, he has in a clear, readable style summarized the lessons of these many years. What the minimum standard for hospitals, enunciated only a few years ago, has done to improve the care of the hospital patient, is well known. For hospital administrators who are striving to maintain that standard, this book comes as an able assistant and an accurate yardstick. For those who strive to carry their hospitals to an even higher standard of efficiency this book is an inspiration.

An enthusiast for standardization, Dr. Mac Eachern nevertheless believes that every hospital has an individuality that can and should be maintained and developed whenever such individuality increases efficiency. One cannot read this book, however, without learning that certain standards of administration and organization are necessary and that a degree of uniformity among hospitals can be helpful without loss of efficient individuality. The chapter on medical staff organization and the methods given for audits of work done by the various clinical departments is particularly valuable.

If the chapter devoted to medical records could be taken as a guide by the Medical Departments of the Army and Navy, for the purpose of making their hospital clinical record forms similar to the forms used in civilian hospitals, much difficulty, confusion, and lost motion

on the part of Reserve medical officers and other civilian physicians recruited by these services for wartime duty could be avoided.

MODERN OFFICE AND GENERAL PRACTICE, by *Deane R. Brengle, M. D.* 320 pages. Southern Publishers, Inc., Kingsport, Tenn., 1935. $3.25 postpaid.

This is a handbook for the general practitioner, full of practical points on internal medicine, obstetrics, and general surgery. An interesting feature is the little autobiographical sketch of the author, which shows him to have been a true son of the Middle Border. Incidently, he served in the Naval Reserve during the World War. The therapeutic measures given in this book are particularly good. Simple and practical measures that have stood the test in the experience of the author.

THE PATHOLOGY OF INTERNAL DISEASES, by *William Boyd, M. D., Professor of Pathology in the University of Manitoba, Pathologist to the Winnipeg General Hospital.* Second edition. 904 pages, 335 engravings. Lea and Febiger, Philadelphia, Pa., 1935. $10.

On the flyleaf is this striking sentence from Morgagni, the founder of gross pathologic anatomy: "Those who have dissected or inspected many bodies have at least learned to doubt; when others, who are ignorant of anatomy and do not take the trouble to attend to it, are in no doubt at all." An excellent thought for any medical man to keep in mind. Many new features have been added to this edition and important alterations made in others. The historical aspects, because of the interest shown in those of the first edition, have been much added to in this edition.

A TEXTBOOK OF BACTERIOLOGY, by *Thurman B. Rice, A. M., M. D., Professor of Bacteriology and Public Health at the Indiana University School of Medicine.* 551 pages. 121 illustrations. W. B. Saunders Co., Philadelphia and London, 1935. $5.

A new textbook of bacteriology excellent in subject matter and illustration. Devoted almost entirely to pathogenic bacteria, particularly those of man.

A GEOGRAPHY OF DISEASE. A PRELIMINARY SURVEY OF THE INCIDENCE AND DISTRIBUTION OF TROPICAL AND CERTAIN OTHER DISEASES, by *Earl Baldwin McKinley, M. D., Dean and Professor of Bacteriology, School of Medicine, George Washington University.* The George Washington University Press, Washington, D. C.

This extensive study was made possible by a grant from the American Leprosy Foundation, formerly the Leonard Wood Memorial, to the Division of Medical Sciences of the National Research Council. It is published as a supplement to the American Journal of Tropical Medicine. The volume contains 495 pages and is packed with valuable statistical information. Furthermore, the descriptive notes regarding the various countries are entertainingly written and inform-

ative and prevent the account from being too dull. It is a little remarkable that this geography of disease should be so relatively neglected a subject, the number of books devoted entirely to it being few indeed. The first of course is Hippocrates' "On Airs, Waters, and Places", one of the medical classics.

METHODS OF TREATMENT, by *Logan Clendening, M. D., Clinical Professor of Medicine, Medical Department of the University of Kansas.* Fifth edition. 879 pages, and 102 illustrations. C. V. Mosby Co., St. Louis. Price $10.

The fifth edition of this well-known work has received its most thorough revision since its original publication in 1924. The general plan has not been altered, but each chapter shows evidence of careful scrutiny, and the entire work reveals additions and revisions which have brought it up to date in conservative and well-founded methods of therapeutics.

The general plan of this volume is unique and practical. It is divided into two parts: part one includes the actions, preparations, and dosages of drugs, including special chapters on bacterial and glandular therapy, dietetics, electro and radiotherapy, psychotherapy, etc.; part two treats with the applications of therapeutic measures to specific diseases.

PRACTICAL DIETETICS, by *Alida Frances Pattee, former Instructor in Dietetics, Bellevue Training School for Nurses.* 847 pages, illustrated. 20th edition. A. F. Pattee, Publisher, Mount Vernon, N. Y., 1935.

When a textbook has been through 20 editions it may be said to have arrived at a secure place in the esteem of those who use it. This is the case with Miss Pattee's excellent handbook of dietetics. The external features of the work are equal to its contents, the binding, paper, and printing being remarkably handsome.

DISEASES OF THE SKIN, by *Frank Crozier Knowles, M. D., Professor of Dermatology, Jefferson Medical College, Colonel Medical Reserve Corps, United States Army.* Third edition. 640 pages, 240 illustrations, and 11 plates. Lea and Febiger, Philadelphia. 1935. $6.50.

This is an excellent moderately-priced handbook of dermatology.

WHAT YOU SHOULD KNOW ABOUT HEART DISEASE, by *Harold E. B. Pardee, M. D., Assistant Professor of Clinical Medicine, Cornell University.* 127 pages. Second edition. Lea and Febiger, Philadelphia. 1935. $1.50.

Brief handbook for the information of the patient.

TEXTBOOK OF PHYSIOLOGY, by *William D. Zoethout, Ph. D., Professor of Physiology in the Chicago College of Dental Surgery (Loyola University).* Fifth edition. 694 pages. Illustrated. C. V. Mosby Co., St. Louis, Mo. 1935. $4.

Excellent textbook in moderate compress, particularly well adapted for the use of dental or pharmaceutical students or for courses in human physiology in the zoology departments of academic colleges.

PERSONAL AND COMMUNITY HEALTH, by *Clair Elsmere Turner, M. A., Dr. P. H., Professor of Biology and Public Health in the Massachusetts Institute of Technology.* Fourth edition. 680 pages, illustrated. C. V. Mosby Co., St. Louis. 1935. $3.

An excellent textbook, moderate in size and cost, on personal and public hygiene. A practical sensible book containing the essentials.

INTRODUCTION TO HUMAN ANATOMY, by *Clyde Marshall, M. D., Department of Anatomy, School of Medicine, Yale University.* 385 pages, 252 illustrations, 14 in color. W. B. Saunders Co., Philadelphia and London. 1935. $2.50.

PRINCIPLES OF BACTERIOLOGY, by *Arthur A. Eisenberg, M. D., Director of Laboratories, Sydenham Hospital, New York;* and *Mabel F. Huntly, R. N., Director of Nursing, Wesson Memorial Hospital, Springfield, Mass.* Sixth Edition. 378 pages, illustrated. C. V. Mosby Co., St. Louis. 1935. $2.75.

DERMATOLOGY AND SYPHILOLOGY FOR NURSES, by *John H. Stokes, M. D., Duhring Professor of Dermatology and Syphilology, School of Medicine, University of Pennsylvania.* Second edition. 368 pages, illustrated. W. B. Saunders Co., Philadelphia and London. 1935. $2.75.

These three books are all new editions of excellent texts for use of nurses or, in the case of Dr. Marshall's book, of secondary schools.

THE BACTERIOLOGY OF TYPHOID, SALMONELLA, AND DYSENTERY INFECTIONS AND CARRIER STATES, by *Leon C. Havens, M. D., Director of Laboratories, Alabama Department of Public Health.* 158 pages, illustrated. The Commonwealth Fund, New York. Published by Humphrey Milford, London, Oxford University Press. 1935. $1.75.

Excellent, well-documented monograph.

THE PATIENT AND THE WEATHER, by *William F. Petersen, M. D.* Vol. 1, pt. 1. Edwards Brothers, Inc., Ann Arbor, Mich. 1935. $3.75.

This is one of a series of monographs on the relation of climate to disease in man. While one cannot but feel that some of the conclusions drawn by Dr. Petersen are not fully justified by the data presented, he is to be highly praised for approaching in a scientific manner this problem which is usually dismissed as fanciful. As a matter of fact, the importance of climate and weather to disease, and the well-being of man, is obvious, yet few subjects have been so neglected. It is a subject to which the naval surgeon might well devote attention possessing as he does almost unparalleled opportunities to observe the effects of rapid change of climate on the human organism. Although neglected by our profession for so long, it is yet another one of those subjects that wise old Hippocrates did not neglect. Dr. Petersen's quotation from Hippocrates is not the least interesting portion of his interesting book.

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THE DIVISION OF PREVENTIVE MEDICINE

S. S. Cook, Commander, Medical Corps, United States Navy, in Charge

MEASLES, MUMPS, SCARLET FEVER, DIPHTHERIA, AND CEREBROSPINAL FEVER ON BOARD SHIPS OF THE UNITED STATES NAVY, 1927–34

By S. S. Cook, Commander, Medical Corps, United States Navy

The appearance of mumps, measles, scarlet fever, cerebrospinal fever, or diphtheria aboard ship is a matter of concern to medical officers and commanding officers. Epidemics of these diseases interfere with ship's routine and when extensive may virtually cause a suspension of all activities. The opinion was formerly held that isolation of cases, drastic quarantine, and vigorous disinfection of noses and throats of contacts would prevent the spread of these diseases. After years of experience and study of epidemics modern public health authorities advise reasonable precautions and are convinced that the extreme measures were not effective and are not warranted. A question which confronts a medical officer when one of these diseases appears aboard his ship is whether an outbreak is imminent and if so how many men will be involved.

In this article is presented a compilation of Navy experience with the five diseases over a period of 8 years in the hope it may serve as a guide in deciding the proper course of action.

The data which are available for the 8-year period 1927–34 give the place and date of admission for each of the five diseases under consideration. In order to determine which of the admissions were primary and which were secondary it is necessary to define these terms. By primary admission is meant a case which occurs outside the usual incubation period for the disease in question and a secondary admission is one which occurs within the incubation limits. In other words, a primary case is one which is not regarded as secondary to another case on the same ship, and by secondary case is meant one which is possibly due to another case on the same ship. It is realized that it is frequently impossible to state whether the infection was acquired on board ship or from outside sources. However, if every case which follows another case within the incubation period for that disease is regarded as secondary the maximum spread will be recorded.

It is further necessary to adopt fixed incubation periods for each disease. They are:

	Days
Mumps	12–26
Measles	7–14
Scarlet fever	2– 7
Cerebrospinal fever	2–10
Diphtheria	2– 5

A case which does not follow another within these limits is regarded as primary, and one which does, as secondary.

MEASLES

In the 8-year period 1927–34 there were 514 primary cases of measles aboard ship. In 103 instances there was one or more secondary cases. Thus in one time out of five a secondary case appeared. The ratio by single years is:

Year	Number of primary cases	Number of times secondary cases appeared	Ratio	Year	Number of primary cases	Number of times secondary cases appeared	Ratio
1927	106	25	1–4	1932	42	9	1–5
1928	37	3	1–12	1933	78	16	1–5
1929	46	10	1–5	1934	71	15	1–5
1930	58	12	1–5				
1931	76	13	1–6	Total	514	103	1–5

The term "outbreak" is used in the exaggerated sense that a single secondary case is considered as an outbreak or as evidence of spread.

In the 8-year period, 1927–34, secondary cases appeared in 103 instances for a total of 348 cases, or an average of 3 cases per outbreak.

Year	Number of outbreaks	Number of cases	Number per outbreak	Year	Number of outbreaks	Number of cases	Number per outbreak
1927	25	77	3	1932	9	31	3
1928	3	10	3	1933	16	35	2
1929	10	46	4	1934	15	33	2
1930	12	20	2				
1931	13	96	7	Total	103	348	3

A tabulation of the 103 outbreaks according to the number of secondary cases in each reveals the following:

Number of cases	Number of outbreaks	Number of cases	Number of outbreaks
1	40	7	4
2	25	8	2
3	9	10	1
4	7	13	1
5	5	19	1
6	7	50	1

From these figures it may be noted that in more than half the instances the outbreak consisted of only one or two cases.

MUMPS

In the 8-year period 1927–34 there were 759 primary cases of mumps on board ship and in 176 instances there was 1 or more secondary cases. Thus in one time out of four a secondary case appeared. The ratio by single years is:

Year	Number of primary cases	Number of times secondary cases appeared	Ratio	Year	Number of primary cases	Number of times secondary cases appeared	Ratio
1927	224	61	1–4	1932	28	6	1–5
1928	76	23	1–3	1933	89	24	1–4
1929	129	21	1–6	1934	81	16	1–5
1930	94	17	1–5				
1931	38	8	1–5	Total	759	176	1–4

In the 8-year period 1927–34 secondary cases appeared in 176 instances and there were 1,133 secondary cases or an average of 6 per outbreak.

Year	Number of outbreaks	Number of cases	Number per outbreak	Year	Number of outbreaks	Number of cases	Number per outbreak
1927	61	354	6	1932	6	56	9
1928	23	248	10	1933	24	167	7
1929	21	92	4	1934	16	99	6
1930	17	73	4				
1931	8	44	5	Total	176	1,133	6

The outbreaks of 1927 and 1929–34 are tabulated according to the number of cases in each outbreak (data for 1928 are not available):

Number of cases	Number of outbreaks	Number of cases	Number of outbreaks
1	53	15	1
2	23	16	2
3	15	17	1
4	10	21	1
5	9	23	1
6	6	26	1
7	9	28	2
8	3	29	1
9	2	30	2
10	3	38	1
11	1	42	1
12	2	65	1
13	2		

From these figures it may be seen that about half of the outbreaks consisted of one or two cases and in 110 instances there were not more than five cases. The numbers exceeded 20 in 11 instances.

CEREBROSPINAL FEVER

In the 8-year period 1927–34 there were 91 primary cases of cerebrospinal fever aboard ship and in 6 instances there was one or more secondary cases. Thus in one instance out of 15 there was a secondary case. The ratio by single years is:

Year	Number of primary cases	Number of times secondary cases appeared	Ratio	Year	Number of primary cases	Number of times secondary cases appeared	Ratio
1927	14	0	.....	1931	1	0	.....
1928	20	2	1–10	1932	5	0	.....
1929	28	2	1–14	1933	2	0	.....
1930	15	1	1–15	1934	6	1	1–6

In the six instances when secondary cases occurred there was one secondary case on four occasions, five on another, and eight on another.

DIPHTHERIA

In the 8-year period 1927–34 there were 52 primary cases of diphtheria aboard ship and no secondary cases.

SCARLET FEVER

In the 8-year period 1927–34 there were 273 primary cases of scarlet fever aboard ship and in 15 instances there was one or more secondary cases. Thus in 1 time out of 18 a secondary case appeared. The ratio by single years is:

Year	Number of primary cases	Number of times secondary cases appeared	Ratio	Year	Number of primary cases	Number of times secondary cases appeared	Ratio
1927	44	2	1–22	1932	21	3	1– 7
1928	28	1	1–28	1933	36	2	1–18
1929	85	2	1–28	1934	22	0	.....
1930	21	3	1– 7				
1931	16	2	1– 8	Total	273	15	1–18

In the 15 outbreaks there were 57 cases or an average of 4 cases per outbreak.

Year	Number of outbreaks	Number of cases	Number per outbreak	Year	Number of outbreaks	Number of cases	Number per outbreak
1927	2	2	1	1932	3	3	1
1928	1	1	1	1933	2	2	1
1929	2	29	10	1934	0	0	0
1930	3	14	5				
1931	2	6	3	Total	15	57	4

A tabulation of the 15 outbreaks according to number of secondary cases in each reveals the following:

Number of cases:	*Number of outbreaks*
1	10
3	1
5	2
6	1
28	1

SUMMARY

The data which have been presented in this article show for the 8-year period 1927–34 and for each of the five diseases under consideration, (1) the number of primary cases, (2) occurrence of secondary cases, (3) average number of secondary cases per outbreak, and (4) the number of outbreaks arranged according to the number of cases in each.

There were 514 primary cases of measles and in 103 instances there was one or more secondary cases giving a ratio of 1 to 5. There were 348 secondary cases or an average of 3 cases per outbreak and in more than half the instances there was only one or two cases. Using these figures as a basis for estimating the expectancy of measles epidemics on board ship one might expect that only one primary case in five would give rise to a secondary case; that the average outbreak would not exceed three cases; and in at least half the instances there would not be more than one or two cases.

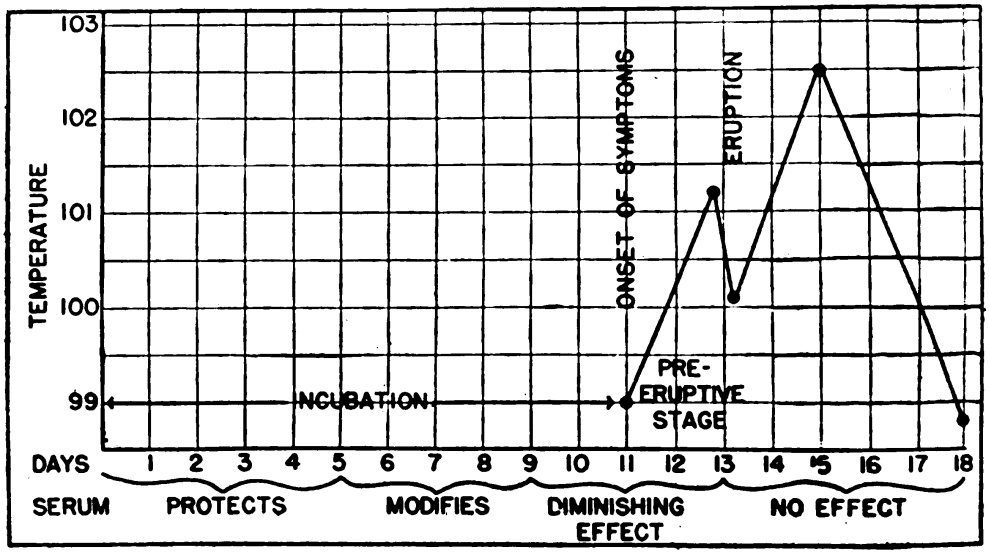
There were 759 primary cases of mumps and in 176 instances there was one or more secondary cases, a ratio of 1 to 4. There were 1,133 secondary cases or an average of 6 cases per outbreak. Data for the 23 outbreaks of 1928 with respect to the number of cases in each are not available but for the 153 outbreaks of the other years there were 53 outbreaks of 1 case, 23 of 2 cases, and 15 of 3 cases. Thus, in over half the outbreaks the number of cases did not exceed three. Basing an estimate on these figures one would expect one primary case in four to give rise to a secondary case. The average epidemic would not exceed six cases and in fully half the instances there would be one, two, or three secondary cases.

There were 91 cases of cerebrospinal fever and in only 6 instances was there a secondary case. On the six occasions there was one secondary case in four instances, five in another, and eight in another. Based on these figures one would seldom expect a secondary case.

There were 52 primary cases of diphtheria and no secondary cases.

There were 273 primary cases of scarlet fever and in 15 instances there was 1 or more secondary cases, a ratio of 1 to 18. There were 57 secondary cases or an average of 4 cases per outbreak. In 10 of the 15 instances there was only 1 secondary case. These figures

show plainly that a case of scarlet fever aboard ship rarely gave rise to another case and when this did occur there was usually only one secondary case.



Measles prophylaxis

The use of convalescent serum or whole blood from an individual who has had measles is a recognized procedure in the prevention or modification of measles. The result depends to a large extent on the interval between exposure and administration of the serum or whole blood. The chart above illustrates the result to be expected.

VENEREAL DISEASES FROM A LINE OFFICER'S POINT OF VIEW

Lt. (j. g.) J. R. Haile, United States Navy, in a study of the venereal disease situation aboard a destroyer presents this trying problem in its relations to military efficiency. His findings are summarized in a table which follows:

1. Number of men attached to the ship, 115.
2. Number of men with entries of syphilis, 31 or 27 percent of the crew.
3. Number of men with entries of gonorrhea, 46 or 40 percent of the crew.
4. Number of men with entries of chancroids, 20 or 17.4 percent of the crew.
5. Total number of men with one or more entries for one or more venereal diseases, 59 or 51.3 percent of the crew.
6. Total number without venereal history, 56 or 48.7 percent of the crew.
7. Total number of sick days for venereal diseases alone, 2,379.
8. Total number of sick days for all other diseases, 5,585.
9. Total sick days, all diseases, venereal group, 5,909.
10. Total sick days, all diseases, nonvenereal group, 2,055.
11. Total years of service, venereal group, 698.6 years.
12. Total years of service, nonvenereal group, 346 years.
13. Average length of service, venereal group, 11.8 years.
14. Average length of service, nonvenereal group, 6.2 years.
15. Sick days per year, venereal group (59 men), 501.5 days.
16. Sick days per year, nonvenereal group (56 men), 331.4 days.
17. Sick days per man per year, venereal group, 8.5 days.

18. Sick days per man per year, nonvenereal group, 5.9 days.
19. Average age of venereal group, 29.7 years.
20. Average age of nonvenereal group, 26 years.
21. Number of advancements in rating made by 59 infected men in 698.6 years of service, 136.
22. Number of advancements made by 56 noninfected men in 346 years of service, 117.
23. Years of service per advancement, venereal group, 5.13 years.
24. Years of service per advancement, nonvenereal group, 2.95 years.
25. Total mast reports entered in current records, venereal group, 58.
26. Total mast reports entered in current records, nonvenereal group 12.
27. Debt letters on file (6-month period), venereal group, 62.
28. Debt letters on file (6-month period), nonvenereal group, 12.
29. Number of bad conduct discharges (6 months), venereals, 2.
30. Number of bad conduct discharges (6 months), nonvenereals, 0.
31. Number of undesirable discharges (6 months), venereals, 1.
32. Number of undesirable discharges (6 months), nonvenereals, 0.
33. Number of desertions (6 months), venereal group, 3.
34. Number of desertions (6 months), nonvenereal group, 0.
35. Number of seamen, second-class, and firemen, third-class, in venereal group, 5.
36. Number of seamen, second-class, and firemen, third-class, in nonvenereal group, 17.
37. Average length of service of seamen, second-class, and firemen, third-class, venereal group, 30 months.
38. Average length of service of seamen, second-class, and firemen, third-class, nonvenereal group, 11 months.

PREVENTION OF TRICHOPHYTOSIS

The medical officer of the U. S. S. *Augusta* in his annual sanitary report describes a method for prevention of trichophytosis which has proved highly satisfactory.

On the 24th of September 1934, 17 men reported to the sick bay for treatment for trichophytosis. The enlisted men were inspected for this condition and 301 men were found to be infected with this fungus. The following method of treatment was carried out: The method consists of a foot bath of 15 percent sodium thiosulphate solution before and after the shower bath. Each man steps into the foot bath as he enters the shower, bathes with mild soap, then dries all but his feet after the bath, then steps into the solution again in the second bucket and lets the solution dry on his feet. The solution must reach well above the ankles assuring complete wetting of the feet. After the solution has dried on the feet, carefully dry between the toes before donning socks and shoes. Foot tubs must be scrupulously scrubbed daily. Daily bathing is necessary in order to be of value in this treatment. Frequent changes and airing of footwear. Scrupulous scrubbing of shower floors. Frequent changes to clean athletic supporters. Treatment of athletic shoes, leather balls (baseballs, footballs, and basketballs) as well as gloves, mitts, and shoes by dusting weekly with a powder of 20 percent sodium thiosulphate in boric acid powder. Two months later another careful inspection was made. Only eight cases showed signs of infection. It was through the help, keen interest, and cooperation of the commanding officer and the executive officer that these satisfactory results were obtained.

YELLOW FEVER [1]

Staff members of the international health division of the Rockefeller Foundation have demonstrated the existence in South America of two types of yellow fever. It appears from their studies that in addition to the urban yellow fever of history there exists widely disseminated throughout South America a type of rural or jungle yellow fever which occurs remote from urban centers and in the complete absence of the notorious yellow fever mosquito, *Aedes aegypti*. The rural type of the disease is being studied and efforts made to find the vector. There may be animal reservoirs of the disease.

HEALTH OF THE NAVY

The following tables are summaries of morbidity rates per thousand for the third quarter of 1935 in comparison with rates for the corresponding quarter of the preceding 5 years:

ENTIRE NAVY

	All diseases	Injuries	Poisonings	All causes	Communicable diseases		Venereal diseases
					A	B	
1930	397	52	2.74	452	(1)	(1)	127
1931	470	59	.47	530	(1)	(1)	142
1932	549	54	.62	604	(1)	(1)	141
1933	404	65	7.10	476	9	86	115
1934	510	65	4.28	580	21	120	105
1935	373	53	.25	426	12	92	75

FORCES ASHORE

1930	450	60	0.58	511	(1)	(1)	85
1931	542	40	.30	583	(1)	(1)	93
1932	544	85	.93	629	(1)	(1)	107
1933	382	74	10.81	468	7	66	72
1934	637	91	1.35	730	31	181	64
1935	426	57	.20	484	14	127	43

FORCES AFLOAT

1930	367	48	3.92	419	(1)	(1)	150
1931	432	70	.56	502	(1)	(1)	169
1932	552	37	.45	590	(1)	(1)	159
1933	414	61	5.32	481	10	95	136
1934	449	53	5.69	507	16	91	125
1935	343	51	.27	395	11	72	92

[1] Not available.

There were 980 admissions for acute infections of the respiratory type reported by all shore stations in the United States during the quarter, of which 331 were notified by the United States naval training station, Norfolk, Va., and 142 by the United States naval training station, San Diego, Calif. Acute catarrhal fever constituted 72 percent of these admissions. The United States naval training station,

[1] Annual Report, 1934, The Rockefeller Foundation, International Health Division.

Norfolk, Va., reported that the cases of catarrhal fever, acute, admitted on that station were of a mild type and mostly restricted to raw recruits. Only six cases of influenza were reported from all shore stations in the United States.

Two cases of cerebrospinal fever were admitted at shore stations in August. A private, United States Marine Corps, with 1 9/12 years' service was admitted to the sick list at the Norfolk Navy Yard, Portsmouth, Va., on August 6 and died on September 15. The other case was reported by the United States naval training station, Norfolk, Va. The patient, a recruit with 3 months' service, was admitted on August 26 and returned to duty after 92 days on the sick list.

There were 170 admissions for respiratory diseases reported by shore stations outside of the continental limits of the United States, 138 of which occurred in the Fourth Marines, Shanghai, China. The annual rate for the third quarter of 1935 was 497.76 per 1,000 as compared with 96.85 per 1,000, the rate for the corresponding quarter of 1934.

A total of 1,186 cases of acute respiratory diseases was reported by all ships during the third quarter, the greatest numbers being reported from the U. S. S. *Trenton*, U. S. S. *Saratoga*, and the U. S. S. *Hannibal*.

Four cases of cerebrospinal fever were reported from forces afloat as follows:

Rate	Age	Place of original admission	Date of admission	Length of service (years)	Disposition
Seaman second class	20	U. S. S. Kane	July 3, 1935	1	Duty Oct. 11, 1935.
Do	19	U. S. S. Canopus	Aug. 12, 1935	1 9/12	Duty Sept. 30, 1935.
Seaman first class	24	U. S. S. Nevada	Aug. 21, 1935	6 5/12	Duty Nov. 22, 1935.
Fireman third class	20	U. S. S. Milwaukee	Aug. 24, 1935	1 5/12	Duty Nov. 19, 1935.

Two moderately severe cases of paratyphoid fever B, without complications, were admitted at the Regimental Hospital, Fourth Marines, Shanghai, China, one in July and one in August. The health records of the patients indicated that one completed a course of typhoid vaccine 5 months prior to infection and the other completed two courses, one 6 years and one 1½ years prior to infection. No record made as to type of vaccine used. One moderately severe case of paratyphoid fever A, without complication, was transferred from the U. S. S. *Dallas* to the United States Naval Hospital, Puget Sound, Wash., in July. This patient had been given two complete courses of typhoid prophylaxis, one course in 1924 ("apparently triple") and the other course 4 years later ("triple").

Seven cases of chickenpox were reported for the quarter, as follows: In July, 2 from the U. S. S. *Pennsylvania;* in August, 1 from the U. S. S. *Nevada* and 2 from the U. S. S. *Saratoga* (fleet air detachment, San Diego, Calif.); and in September, 1 each from the U. S. S. *Nevada* and the U. S. S. *New Mexico*.

TABLE 1.—*Summary of morbidity in the U. S. Navy for the quarter ended Sept. 30, 1935*

Average strength	Forces afloat 73,134		Forces ashore 40,096		Entire Navy 113,230	
	Admissions	Rate per 1,000	Admissions	Rate per 1,000	Admissions	Rate per 1,000
All causes	7,219	394.84	4,847	483.54	12,066	426.25
Disease only	6,279	343.42	4,275	426.48	10,554	372.83
Injuries	935	51.14	570	56.86	1,505	53.17
Poisonings	5	.27	2	.20	7	.25
Communicable diseases transmissible by oral and nasal discharges (class VIII):						
(A)	201	10.99	143	14.27	344	12.15
(B)	1,325	72.47	1,277	127.39	2,602	91.92
Venereal diseases	1,699	92.93	429	42.80	2,128	75.17

TABLE 2.—*Deaths reported, entire Navy, during the quarter ended Sept. 30, 1935*

Cause		Navy			Marine Corps		Nurse Corps	Total
Primary	Secondary or contributory	Officers	Midshipmen	Men	Officers	Men		
Average strength		9,683	1,934	83,951	1,208	16,110	344	113,230
DISEASE								
Alcoholism, acute	Dilatation, stomach, acute.	1						1
Appendicitis, acute	Peritonitis, general, acute.			2		1		3
Cellulitis, face and lip	Septicemia			1				1
Cerebrospinal fever	None					1		1
Leukemia	do			1				1
Myelitis, transverse	Pneumonia, lobar					1		2
Myocarditis, chronic	Thrombosis, coronary	1		1				1
Do	Arterial hypertension			1				1
Do	Pneumonia, lobar					1		1
Pneumonia, broncho	None			1				1
Pneumonia, lobar	do			1				1
Do	Abscess, lung			1				1
Do	Abscess, retropharyngeal					1		1
Do	Meningitis, cerebrospinal, acute.			1				1
Do	Pleurisy, suppurative			1				1
Psychosis, intoxication, alcoholic.	Pneumonia, broncho			1				1
Sinusitis, ethmoidal	Septicemia			1				1
Syphilis	Malaria, quartan, therapeutic, and Myocarditis, acute.			1				1
Thrombosis, coronary	None	1		2				3
Tonsillitis, acute	Abscess, lung			1				1
Tuberculosis, pulmonary, chronic.	None			1				1
Tuberculosis, meninges	do			3				3
Tumor, malignant, mixed	Dilatation, cardiac, acute	1						1
Ulcer, stomach	Abscess, liver	1						1
Valvular heart disease	None			1	1			2
Total for disease		5		22	1	5		33
INJURIES AND POISONINGS								
Asphyxiation, illuminating gas.	None			1				1
Burn, multiple	do			1				1
Caisson disease—(bends and diver's paralysis.)	do			1				1
Dislocation, vertebra, cervical	do			1				1
Drowning	do			5		2		7
Injuries, multiple, extreme	do	3		4		4		11
Intracranial injury	do					2		2
Strangulation, neck	Dementia, praecox			1				1
Wound, gunshot, head	None			6		1		7
Wound, punctured, heart	do			1				1
Poisoning, acute, cyanide	do			1				1
Total for injuries and poisonings.		3		22		9		34
Grand total		8		44	1	14		67

TABLE 2.—*Deaths reported, entire Navy, during the quarter ended Sept. 30, 1935*—Continued

Primary cause	Navy			Marine Corps		Nurse Corps	Total
	Officers	Midshipmen	Men	Officers	Men		
Annual death rate per 1,000:							
All causes	3.30	------	2.10	3.31	3.48	-------	2.37
Disease only	2.07	------	1.05	3.31	1.24	-------	1.17
Drowning	------	------	.24	------	.50	-------	.25
Poisonings	------	------	.05	------	------	-------	.04
Other injuries	1.23	------	.76	------	1.74	-------	.91

ADMISSIONS FOR INJURIES AND POISONINGS, THIRD QUARTER, 1935

The following table, indicating the frequency of occurrence of accidental injuries and poisonings in the Navy during the third quarter, 1935, is based upon all form F cards covering admission in those months which have reached the Bureau:

	Admissions, July, August, and September, 1935	Admission rate per 100,000 per annum	Admission rate per 100,000, year 1934
INJURIES			
Connected with work or drill	643	2,271	2,397
Occurring within command but not associated with work	412	1,455	2,064
Incurred on leave or liberty or while absent without leave	450	1,590	1,699
All injuries	1,505	5,316	6,160
POISONINGS			
Industrial poisoning	0	0	15
Occurring within command but not connected with work	3	11	244
Associated with leave, liberty, or absence without leave	4	14	15
Poisonings, all forms	7	25	273
Total injuries and poisonings	1,512	5,341	6,433

Percentage relationships

	Occurring within command				Occurring outside command—leave, liberty, or A. W. O. L.	
	Connected with the performance of work, drill, etc.		Not connected with work or prescribed duty			
	July, August, and September, 1935	Year 1934	July, August, and September, 1935	Year 1934	July, August, and September, 1935	Year 1934
Percent of all injuries	42.7	38.9	27.4	33.5	29.9	27.6
Percent of all poisonings	0	5.3	42.9	89.3	57.1	5.3
Percent of total admissions, injury and poisoning titles	42.5	37.5	27.4	35.9	30.0	26.6

NOTE.—Poisoning by a narcotic drug or by ethyl alcohol is recorded under the title "Drug addiction" or "Alcoholism", as the case may be. Such cases are not included in the above figures. There were no cases during the third quarter of 1935 worthy of notice from the standpoint of accident prevention.

STATISTICS RELATIVE TO MENTAL AND PHYSICAL QUALIFICATIONS OF RECRUITS

The following statistics were taken from monthly sanitary reports submitted by naval training stations:

July, August, and September 1935	United States Naval Training Station			
	Norfolk, Va.	Newport, R. I.[1]	Great Lakes, Ill.	San Diego, Calif.
Recruits received during the period	1,760	----------	351	1,612
Recruits appearing before Board of Medical Survey	11	----------	3	0
Recruits recommended for discharge from the service	11	----------	3	0
Recruits discharged by reason of medical survey	12	----------	3	0
Recruits held over pending further observation	0	----------	0	0
Recruits transferred to the hospital for treatment, operation, or further observation for conditions existing prior to enlistment	36	----------	5	20

[1] No data received.

The following table was prepared from reports of medical surveys in which disabilities or disease causing the surveys were noted as existing prior to enlistment. With certain diseases, survey followed enlistment so rapidly that it would seem that many might have been eliminated in the recruiting office.

Cause of survey	Number of surveys	Cause of survey	Number of surveys
Abscess, periapical (multiple)	1	Hallux valgus	1
Acne, vulgaris	1	Hernia, inguinal	1
Adhesions, abdominal	1	Hydrocele	1
Arterial hypertension	6	Malformation, congenital	2
Arthritis, chronic, right knee	1	Malocclusion, teeth	1
Asthma	1	Myopia	1
Caries, teeth	2	Nephritis, chronic	2
Chancroid, penis	1	Otitis, media, chronic	2
Cholecystitis, chronic	1	Perforated nasal septum	3
Color-blindness	1	Pes cavus	1
Constitutional psychopathic inferiority, without psychosis	1	Psychoneurosis, hysteria	2
		Psychoneurosis, neurasthenia	1
Constitutional psychopathic state, inadequate personality	1	Pyorrhea alveolaris	2
		Sprain, lumbo-sacral	1
Deafness, unilateral	1	Stammering	1
Deformity, acquired, feet	2	Synovitis, chronic, left knee	1
Deformity acquired, right leg	1	Tuberculosis, subcutaneous fascia	1
Deviation, nasal septum	1	Union of fracture, faulty	1
Effort syndrome	2	Valvular heart disease, combined lesions, aortic and mitral	1
Enuresis	4		
Epilepsy	2	Valvular heart disease, mitral stenosis	2
Flat foot	12	Valvular heart disease, pulmonic lesions	1
Foreign body, left foot	1	Varicose veins, right leg	1
Gonococcus infection, urethra	4		

VOLUME XXXIV JULY 1936 NUMBER 3

United States Naval Medical Bulletin

PUBLISHED *for the* INFORMATION OF
MEDICAL DEPARTMENT *of the* NAVY

THE MISSION OF THE MEDICAL CORPS OF THE NAVY

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TO KEEP AS MANY MEN AT AS MANY GUNS AS MANY DAYS AS POSSIBLE

Issued Quarterly by the Bureau of Medicine and Surgery
Washington, D. C.

VOL. XXXIV JULY 1936 No. 3

UNITED STATES NAVAL MEDICAL BULLETIN

PUBLISHED QUARTERLY FOR THE INFORMATION OF THE MEDICAL DEPARTMENT OF THE NAVY

Issued by

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NAVY DEPARTMENT

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COMMANDER LOUIS H. RODDIS
MEDICAL CORPS, U. S. NAVY, IN CHARGE

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NAVY DEPARTMENT,
Washington, March 20, 1907.

This UNITED STATES NAVAL MEDICAL BULLETIN is published by direction of the Department for the timely information of the Medical and Hospital Corps of the Navy.

TRUMAN H. NEWBERRY,
Acting Secretary.

Owing to the exhaustion of certain numbers of the BULLETIN and the frequent demands from libraries, etc., for copies to complete their files, the return of any of the following issues will be greatly appreciated:

Volume IX, no. 1, January 1915.
Volume X, no. 2, April 1916.
Volume XI, no. 3, July 1917.
Volume XII, no. 1, January 1918.
Volume XII, no. 3, July 1918.
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PREFACE

THE UNITED STATES NAVAL MEDICAL BULLETIN was first issued in April 1907 as a means for supplying medical officers of the United States Navy with information regarding the advances which are continually being made in the medical sciences, and as a medium for the publication of accounts of special researches, observations, or experiences of individual medical officers.

It is the aim of the Bureau of Medicine and Surgery to furnish in each issue special articles relating to naval medicine, descriptions of suggested devices, clinical notes on interesting cases, editorial comment on current medical literature of special professional interest to the naval medical officer, and reports from various sources, notes, and comments on topics of medical interest.

The bureau extends an invitation to all medical and dental officers to prepare and forward, with a view to publication, contributions on subjects of interest to naval medical officers.

In order that each service contributor may receive due credit for his efforts in preparing matter for the BULLETIN of distinct originality and special merit, the Surgeon General of the Navy will send a letter of appreciation to authors of papers of outstanding merit.

The bureau does not necessarily undertake to endorse all views or opinions which may be expressed in the pages of this publication.

P. S. ROSSITER,
Surgeon General, United States Navy.

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Contributions to the BULLETIN should be typewritten, *double spaced*, on plain paper, and should have wide margins. Fasteners which will not tear the paper when removed should be used. Nothing should be written in the manuscript which is not intended for publication. For example, addresses, dates, etc., not a part of the article, require deletion by the editor. The BULLETIN endeavors to follow a uniform style in heading and captions, and the editor can be spared much time and trouble, and unnecessary changes in manuscript can be obviated if authors will follow in these particulars the practice of recent issues.

The greatest accuracy and fullness should be employed in all citations, as it has sometimes been necessary to decline articles otherwise desirable because it was impossible for the editor to understand or verify references, quotations, etc. The frequency of gross errors in orthography in many contributions is conclusive evidence that authors often fail to read over their manuscripts after they have been typewritten.

Contributions must be received at least 3 months prior to the date of the issue for which they are intended.

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U. S. NAVAL MEDICAL BULLETIN

VOL. XXXIV JULY 1936 No. 3

SPECIAL ARTICLES

REVIEW OF THE PATHOLOGY OBSERVED IN 1,018 POST-MORTEM EXAMINATIONS IN HAITI[1]

By J. H. CHAMBERS, Commander, Medical Corps, United States Navy

In an attempt to establish some further data on the relationship between yaws and syphilis, an investigation was undertaken in the Republic of Haiti, where these spirochetal diseases are prevalent. A 3-year investigation was originally planned, aided financially by the committee on research in syphilis, to consider the etiologic, clinical, anatomical, immunological, and epidemiological relationship of the two diseases. In this report these conditions are referred to as separate clinical entities, not with an assumption of proven duality but to avoid possible confusion on the part of the reader. It has been well established that yaws predominates in the rural districts and syphilis is confined largely to the seacoast towns of Haiti. Very rarely is chancre seen in the country. Recognizing the similarity of causative organisms, serum reaction, and occasionally the type of external lesion, it became customary in the Public Health Service of Haiti to list both conditions under the one title of treponematoses in statistical returns when their differentiation was not evident. Thus, often dependent upon the personal leaning of medical officers in different districts, the ratio of yaws, syphilis, and treponematosis varied from time to time, but the annual reports show a high incidence of all of them.

The report for 11 months of 1930–31 shows 7,924 Kahn reactions in Port au Prince, with 3,309, or 67 percent, positive. During this same period 4,357 tests were recorded in district laboratories. The report does not show the percent of positives among them, but personal examination of the laboratory records previously showed a rate never below 70 percent.

During the first year of this investigation, Dr. T. B. Turner brought rabbits and monkeys to Haiti, and yaws lesions were established in rabbits and a preliminary report made (reference 1). These strains

[1] This investigation was aided by funds from the committee on research in syphilis.

have been continued in animals at Johns Hopkins Hospital to observe the persistance of type lesions and cross-immunity reactions with syphilis strains.

Arrangements were made for the examination of selected anatomical material by the late Dr. A. S. Warthin at the University of Michigan, and pieces of all organs from selected cases were forwarded to him. Dr. Warthin hoped to study this tissue particularly for the distribution of spirochetes and for any evidence of consistent variation in cell structure or arrangement, or of characteristics of the organisms in lesions of known syphilitic or yaws origin. The following basis was adopted for the selection of the material. At autopsy any change in the aorta, other than frank arteriosclerosis in elderly subjects, was accepted as of possible spirochetal origin, and part of that aorta and pieces of all organs were fixed in formalin. Microscopic sections of all organs from all subjects were studied later, and if any evidence of spirochetal infection was noted, pieces of all organs of that case were also forwarded in formalin to Dr. Warthin. On this basis of selection, material from over 200 subjects was forwarded. Following the untimely death of Dr. Warthin, his associate, Prof. Carl Weller, undertook to complete the study, and he has reported his findings elsewhere.

At the Haitian General Hospital, in Port au Prince, the patients came from the city and also the surrounding country, giving somewhat of a cross section of the populace. However, the Port au Prince Hospital rather rarely had any patients from the more remote country districts where yaws is supposed to exist more or less uncontaminated by syphilis. All patients dying at the Haitian General Hospital were subjected to post-mortem study in the laboratory of the National School of Medicine of Haiti. All patients dying in the other charitable institutions in the city and the bodies of all indigent natives were also brought to this laboratory for study. From these sources over 1,000 post-mortem examinations were made, and microscopic section of all organs were routinely studied.

Clinical abstracts were recorded from the hospital records, but all too frequently these records were very scant. The Haitian peasant has had for generations fear and distrust of hospitals that has just begun to break down, and as a result he usually arrived at the hospital in a moribund state. Coupled to this there was a fatalistic attitude that "what is to be is to be", making history taking extremely difficult. The peasant is naturally of a kindly disposition and in giving his history tries very hard to be helpful and to agree with whatever the white doctor desires, so that extreme care must be exercised or the patient will give the history that he feels the doctor wants. Careful questioning will develop histories entirely contradictory to those carelessly or briefly taken.

It was originally hoped that an authentic clinical history would be obtained on each of the subjects selected for study by Dr. Warthin, particularly as to whether there was a history of yaws or syphilis. After a time it was felt that absolutely no dependence could be placed on the recorded history, except in the comparatively few instances when a history was carefully taken by an interested naval medical officer expressly for the purpose of this record. Hence it is believed that no weight should be given the ordinary history of previous infection—except that we know that a majority of Haitians, especially in the rural districts, have had yaws and that about 70 percent of the population show a positive Kahn reaction. In order to have some helpful evidence available, at autopsy there was a note made of the presence or absence of a scar on the glans penis. While yaws is not a venereal disease, lesions occasionally appear on the genitalia, but they do not as consistently affect the glans as does the chancre. It was felt that if a scar of the glans penis existed, syphilis could not be ruled out, regardless of the recorded history.

At the end of the first year of investigation the committee on research in syphilis found it necessary to markedly curtail their grants, and only sufficient money was available to pay the salary of a technician in histopathology, thus greatly handicapping the clinical investigation. A second year of study of anatomical material and a third year devoted largely to clinical work had been planned originally, but the abrupt withdrawal of the naval medical officers in September 1931 prevented the start of any comprehensive clinical study. An attempt was made to abstract clinical records where available on all subjects that had been autopsied, but they were found to offer little worth-while data, more than 50 percent showing no record of Kahn reaction, and the history was considered of doubtful authenticity in about 85 percent of the cases.

As a result of this series of unexpected developments, this report can properly comprise only a review of the anatomic findings in 1,018 consecutive autopsies, with some brief comment on the few cases where dependable history is available.

The cause of death as determined by post-mortem examination has been classified in accordance with the International List of Causes of Death, and arranged for sex and age groups in tables nos. 1 and 2. A plotted comparison of the deaths by ages in Haiti and in the colored population of the United States for 1931–32 shows a rate among Haitians considerably higher during the period between 25 and 40 years. This difference is also shown in a similar comparison for 1928 and for 1934.

The apparently low rate in those under 1 year of age in Haiti does not indicate the true infant mortality rate. The Haitian table is from autopsies only, and while the child and adult figures are representa-

tive, very rarely were infants brought to the hospital. Accurate vital statistics for Haiti are not available, as reports of births and deaths from most rural districts are never made.

Considering the deaths from general diseases, including malignant tumors, the Haitian group shows 40 percent of males and 42 percent of females classed in this group as contrasted with 22 percent and 28 percent for colored people in United States of America. This striking difference is due to the prevalence of tuberculosis in Haiti. In the United States group 49 percent of male and 52 percent of females die of tuberculosis, as contrasted with 78 percent for both sexes in the Haitian group. Considered in relation to deaths from all causes, the United States group shows 12 percent and 13 percent for males and females, contrasted with 31.6 percent and 32.7 percent for Haitians. Choisser (reference 2) reported 31.6 percent of deaths due to tuberculosis from 300 autopsies in 1928 and later 28 percent of 400 additional necropsies. It is of interest to note the lack of any betterment in this condition. Many factors may be to blame. The Haitian has not been educated up to preventive medicine and makes no attempt at sputum control or contact prevention. He is not interested in hospital care until it becomes urgently necessary, and, of course, this is too late in tuberculosis.

Efforts by the Service d'Hygiene to establish a hospital of the sanitarium type for treatment of early cases were never successful, because of lack of funds. All available money had to be used for more urgent needs. The author did succeed in persuading two patients with minimal lesions to submit to hospitalization. Both had definite physical and X-ray findings of single lobe involvement, fever, and positive sputum. One patient was comparatively fever free in 3 weeks and could see no need of staying in the hospital. The other patient remained for 3 months, and on the ordinary hospital ration and rest had become free of fever and cough, the moderately coarse rales had largely disappeared, and the X-ray showed definite fibrosis instead of the original soft shadow. The parents of this patient (a girl of about 12 years) insisted on taking her home, and within a year she was back with a widespread lesion. In spite of the poor racial resistance to tuberculosis, these cases with other scattered observations, convince one that education of the native to measures of prevention, prolonged treatment of early cases, and, most important, financial provision for the latter would tremendously reduce the mortality from this disease even in a Negro population.

Anatomically, all types of tuberculosis occurred, but the one striking feature was the enormous extent of the lesions. Frequently the massive lung destruction, coupled with abdominal and gland involvement, made one wonder how the patient had enough strength to make his way to the hospital to die.

Malaria was accredited as the cause of death in eight cases, seven of these occurring during the latter months of the period. Unusually heavy and persistent rains inundated large areas and lowered the general living conditions of all the population in varying degree. This was accompanied by a marked rise in the positive-smear rate from the level of 6 percent, where it had remained for almost 2 years, up to over 40-percent positives in the routine daily smears from out patients and those in the hospital in Port au Prince. While malaria is not such a frequent immediate cause of death, it unquestionably helps pave the way for other fatal conditions.

Deaths from circulatory diseases again offer an interesting contrast with statistics for the colored race in the United States, where 14.4 percent males and 14.6 percent females appear in this group, while only 9.8 percent males and 8.1 percent of female Haitians. This definite difference is no doubt largely due to the almost complete absence of acute rheumatic heart disease syndrome. Acute articular rheumatism is rarely seen and the resultant cardiac damage is lacking. Four hearts showed mitral valve damage, and only one of these, an active endocarditis.

Aneurysm of the ventricle appeared twice, one being of such size as to be worthy of comment. The subject was an elderly male of about 55 years, dying outside the hospital, so no history was obtainable. The body was described as well nourished, fair musculature, and scant subcutaneous fat. There was no record as to presence or absence of either yaws scar or of scar on the glans penis. The lower extremities, penis, and scrotum were markedly edematous. On removing the breast plate, the entire lower-left chest appeared to be occupied by a large sac that had displaced the left lung upward. The pericardium over the heart appeared normal, fitted closely about the junction between sac and heart and completely enveloped the latter. Except for a few fine adhesions, the heart and aneurysmal sac were free in the pericardium and its extension. The communication between ventricle and aneurysm measured about 10 cm in diameter. On section, this junction showed a firm muscular wall, with a heavy fibrous ring and lined by apparently good endocardium. Muscle fibers extended outward and with fibrous tissue made up a wall, which thinned somewhat on the part most distant from the heart, but showed no leakage of blood and no signs of beginning rupture. In the sac, there were some layers of partially organized blood clot. The ventricular wall showed extensive scarring and some perivascular round cell infiltration. The coronaries were patent and all valves were essentially normal. The autopsy showed the usual signs of congestive heart failure with advanced atheromatous change in the aorta. There was a beginning bronchopneumonic process in each lung and a large submacillary abscess. Tissue from this patient was forwarded to Dr. Warthin for examination for spirochetes.

The three illustrations show (1) the heart with aneurysm enclosed in pericardial sac, (2) after turning back the pericardium, and (3) the heart opened with an incision through the aneurysmal sac and orifice. The latter shows fairly well the fibrous ring about the communication and the well-muscled wall of the sac.

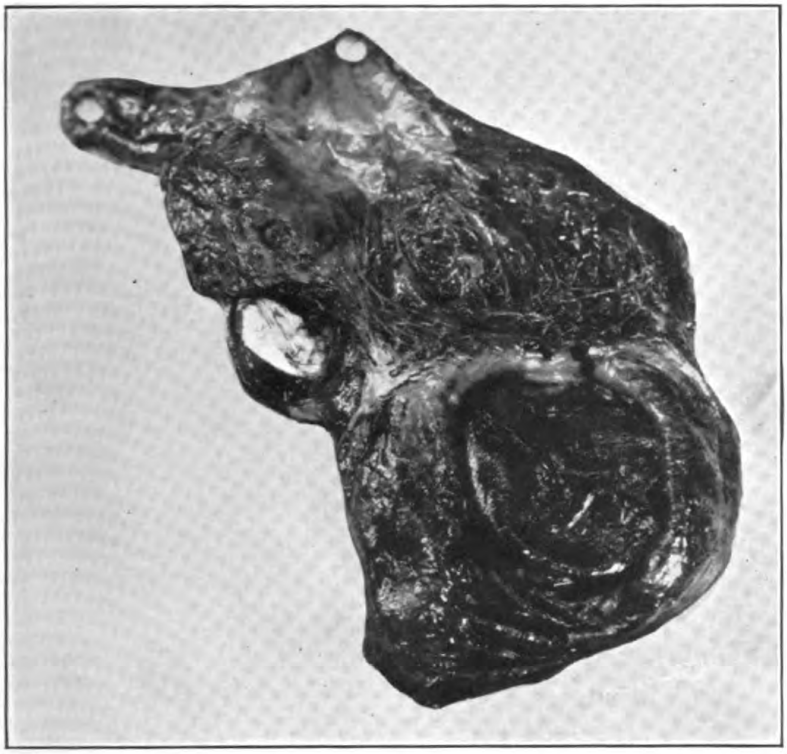
It was felt that a careful tabulation of the relative frequency of involvement of various organs and tissues by any pathological process might be of interest or value so such a study was made. It was most interesting to note how greatly personal impressions of relative frequency of involvement was at fault when the actual tabulation was completed. Considerable labor was involved, as each record comprised six pages and the tabulation of gross and microscopic changes necessitated over 50 inspections of each of more than 3,000 sheets. The organs or tissues involved are listed in age groups for each sex in the relative frequency of their involvement in tables 3 and 4. Generally the frequency is the same in males and females, with occasional minor variations, and shows approximately the age distribution that one would expect. This tabulation includes all departures from normal; congestion of the lung in heart disease, congestion of kidney in fever, and mild chronic passive congestion of the liver are all included.

A few points are of special interest. The relatively low coronary damage, I believe, is quite striking. The frequent kidney damage is to be expected. The liver shows 438 males and 294 females with gross changes in the liver. In many instances this represented merely abnormal fat and varying degrees of chronic passive congestion. Choisser reported only 10 cases of cirrhosis and commented on the low incidence. In this group almost 75 percent show some gross changes and there were 46 cases showing well-marked frank cirrhosis, a much higher incidence than that referred to above.

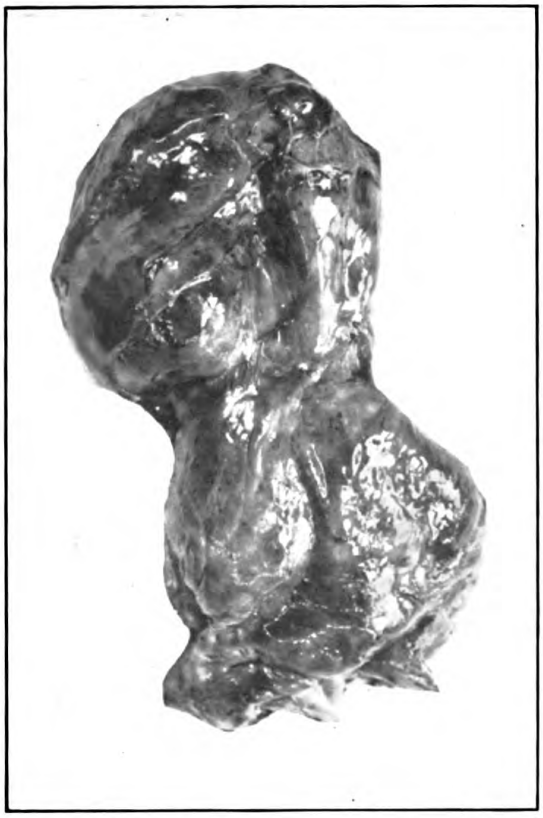
The results of the microscopic study of many of the tissues and organs are similarly arranged in tables 5 and 6. Once more the liver shows a very high rate, with the kidneys showing the greatest damage. In these tables the percentage is omitted for certain tissues, as they were not routinely examined microscopically in all cases.

Keeping in mind the original object of the investigation, evidences of spirochetal infection were of primary interest. In this connection, changes indicative of syphilis were noted in the adrenals of several subjects, and Dr. Weller (reference 3) reported the demonstration of spirochetes in the adrenals in two of the cases forwarded to him. Recognizing the relative frequency of circulatory system damage from syphilis in members of the colored race in temperate zones, the changes in the aorta will be considered in more detail.

Reference to tables 3 and 4 shows that the aorta in 152 females and 242 male cases was recorded as presenting gross abnormalities. Of

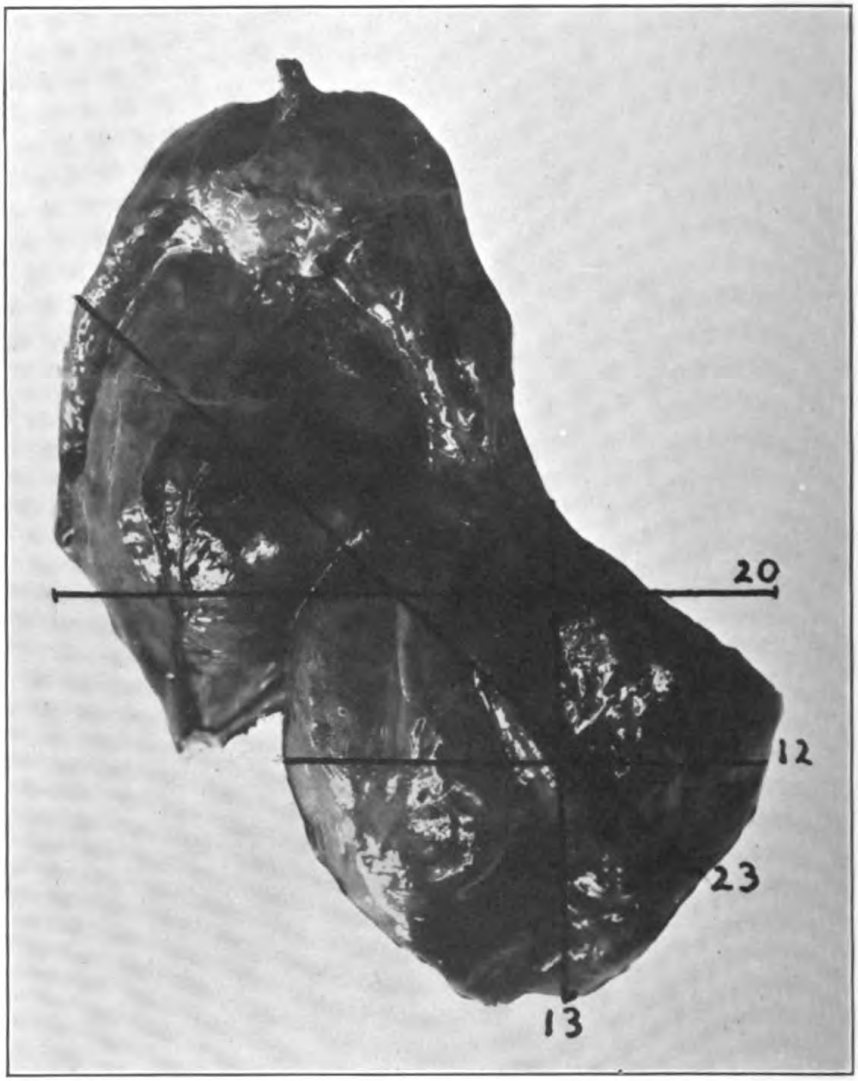


HEART AND ANEURYSM INCISED AND REFLECTED.



HEART AND ANEURYSM SHOWING COMPLETE PERICARDIAL INVESTMENT.

290—1



HEART AND ANEURYSM WITH PERICARDIUM REMOVED.

Measurements in centimeters.

this total of 394 only 50 presented gross changes justifying the diagnosis of syphilis, the remaining 344 showed atheromatous changes only. The latter were classified, according to severity, as 176 slight, 95 moderate, and 73 severe. An additional 165 cases showed slight yellowish deposits of fat beneath the intima, but were not recorded in the table of gross changes.

The microscopic examination, made without reference to the gross findings, shows a higher incidence of abnormalities with changes in 294 males and 164 females, a total of 458. The degree of damage was recorded as 225 slight, 104 moderate, and 74 severe cases of atheroma, and 55 of syphilis. Included in the latter group were 20 aortic aneurysms, 15 of them in males and 5 in females.

A basis for comparison is furnished by Turner's (reference 4) review of 10,000 cases of syphilis at Johns Hopkins Hospital. He considered a total of 4,322 colored males and females showing cardio-vascular lesions of late syphilis and found 331, or 7.65 percent of these showed either aortitis, or aneurysm. This figure does not include angina and other cardio-vascular lesions. Accepting the 70 percent positive Kahn rate in Haiti as evidence of yaws, or syphilis, there is a normal expectancy of these diseases in 70 percent of the 1,018 autopsies, or 713 cases. Microscopic evidence of syphilis was found in 55 instances, giving a rate of 7.71 percent of aortic involvement, a figure approximately the same as in United States Negroes reported by Turner.

There was nothing striking in the gross or microscopic picture in these cases, the lesions showing the expected variation in extent and stage of involvement. Nor could the author observe any variations of tissue arrangement that suggested any difference in origin of the changes present.

Considering the group of 55 showing evidence of syphilis in the aorta, 47 were males and 8 females, the youngest a female of 19 with aneurysm and the oldest a female of 95 also with aneurysm. The average age was 49.8 years. In the case of the girl of 19 with ruptured aneurysm, there is no clinical data whatsoever, except that she came from the country near Port au Prince. As previously mentioned the history is usually not dependable, but in a certain number of the cases the history was taken by a naval medical officer especially for the purpose of this record, paying particular attention to the positive and negative record of yaws or syphilis. It is felt that these histories may be accepted as definite evidence of syphilis especially since in every positive history for a male subject the latter demonstrated the scar of his chancre on the glans penis. Patients admitted to the Haitian General Hospital and there having the diagnosis established and treatment given, are considered as syphilitic. In those bodies arriving from outside sources with no clinical data, it was felt that syphilis could not be ruled out if there was a scar on the glans penis, and therefore these scarred bodies are listed among the syphilitics.

In those cases where a naval medical officer recorded a history negative for lues, it was considered proper evidence and to be classed under nonsyphilitic and if the yaws history was positive, classed as yaws. In the absence of positive history and of penile scar, and presence of scars probably due to yaws, to be classed as yaws.

With this basis of selection applied it is found that 37 of the 55 cases showed evidence placing them in the potential luetic group. One case, a man of 40, gave an acceptable history negative for chancre and chancre scar and positive for yaws and demonstrated the mark of the mother yaw on his left heel. One case showed yaws scars and no scar on the penis, but, coming from the outside, there was no history of clinical data. Sixteen cases, including five females, were without any clinical data and unfortunately there was no record made at autopsy or presence or absence of any scars. The Kahn reaction was positive in 17, negative in 5, and not recorded in 33 subjects. Some cases (3 in number) showed involvement by a gummatous lesion in the region of the aortic valves and in one case a valve leaflet had ruptured. In a fair number of cases the luetic process was associated with an atheromatous one rendering the diagnosis more uncertain until after microscopic examination.

In addition to the 20 saccular aortic aneurysms there was one considered a true dissecting aneurysm, involving the lower thoracic aorta and causing death by rupture.

The youngest female with aneurysm was 19, the oldest 95, with an average age of 42.8 years.

The youngest male was 39, the oldest 80, and the average age 58.8.

Applying the same criteria as for aortitis, there were 9 cases of syphilis, 1 of yaws (the same individual included among the aortitis), and 10 cases from outside on which there was no history or record of presence or absence of scars. Four had a positive Kahn, 2 negative and 14 not recorded. Seven aneurysms were from the ascending portion, four from the transverse, and four from the descending thoracic aorta, with three from the abdominal. One case showed a saccular aneurysm at the beginning of the transverse portion and another in the lower thoracic aorta, the latter having ruptured. Another case showed a saccular aneurysm of the ascending portion, of the descending portion (ruptured), and two aneurysms of the abdominal aorta. In 7 of the 20 cases there had been spontaneous rupture.

SUMMARY AND CONCLUSION

1. This report falls woefully short of the objective originally planned as outlined in the opening paragraphs.

2. This partial failure is largely due to the unexpected and uncontrollable factors which curtailed the work.

3. The bodies examined were from Port au Prince and the surrounding country, but represented very few of the remote country people.

4. Over 1,000 autopsies were performed from August 1929 to August 1931, the gross findings recorded on standardized forms, and the microscopic findings likewise recorded.

5. The microscopic examination comprised a minimum of 12 tissues routinely examined and such additional tissues as showed gross changes.

6. Material from over 200 cases showing some aortic change or evidence of syphilis in other organs, was forwarded to the Department of Pathology of the University of Michigan.

7. Tuberculosis with a rate of 32 percent is still the outstanding cause of death in Haiti.

8. There was no special gross or microscopic pathology of the aorta or any other organ that would distinguish yaws from syphilis.

9. In 55 cases showing syphilis of the aorta, there was no recorded means of determining whether the patients ever had yaws or syphilis in 16 cases. Thirty-seven presented what was considered satisfactory evidence of syphilitic infection. One gave a definite history of yaws without syphilis and one other was presumed to be yaws in origin.

10. In 20 aneurysms of the aorta, 10 were without data. Nine were considered due to syphilis and one (the same individual listed under aortitis) due to yaws in the absence of syphilis.

11. In conclusion it is felt that aortic changes due to syphilis or yaws are not unduly common in Haiti, appearing in only 5.4 percent of these autopsies. The percentage of cases of luetic aortitis is approximately the same as that found in Negro syphilitics in United States. Since all but 2 of the 39 syphilitic aortitis cases upon which there was evidence for diagnosis were found due to syphilis, it is felt that yaws very rarely causes aortitis.

TABLE No. 1.—*Post-mortem determination of the cause of death in males*

	1	1–4	5–9	10–14	15–19	20–29	30–39	40–49	50–59	60–69	70–79	80–89	90	Total
I. General diseases	0	3	4	4	11	73	56	36	18	20	16	1	1	243
II. Nervous diseases	2	0	1	0	2	2	5	4	1	3	1	0	0	21
III. Circulatory	0	0	0	1	0	6	14	13	9	9	5	2	0	59
IV. Respiratory	1	0	0	0	1	11	13	16	9	9	7	0	1	68
V. Digestive	2	3	1	3	3	8	8	19	12	14	1	0	0	74
VI. Nonvenereal G. U.	2	1	1	0	0	10	12	12	12	21	9	2	2	84
VIII. Skin and cellular tissue	0	0	0	0	0	0	0	1	0	0	0	0	0	1
IX. Bone and locomotion	0	0	0	0	0	1	1	0	0	3	0	0	0	5
X. Malformation	1	0	0	0	0	0	0	0	0	0	0	0	0	1
XI. Early infancy	4	0	0	0	0	0	0	0	0	0	0	0	0	4
XII. Old age	0	0	0	0	0	0	0	0	0	0	0	0	0	0
XIII. External	0	2	0	0	4	7	12	8	4	2	2	0	0	41

TABLE NO. 2.—*Post-mortem determination of the cause of death in females*

	1	1–4	5–9	10–14	15–19	20–29	30–39	40–49	50–59	60–69	70–79	80–89	90	Total
I. General diseases	0	3	4	5	15	49	41	23	13	14	5	2	0	174
II. Nervous diseases	0	0	0	1	0	0	2	1	2	0	1	0	1	8
III. Circulatory	0	0	0	0	1	3	6	4	2	6	8	3	1	34
IV. Respiratory	4	3	2	0	1	2	9	8	5	4	2	0	1	41
V. Digestive	0	0	0	0	1	8	9	12	3	2	2	2	0	39
VI. Nonvenereal G. U.	0	0	0	0	5	9	11	7	11	9	21	3	0	76
VII. Puerpural state	0	0	0	0	1	9	5	0	0	0	0	0	0	15
VIII. Skin and cellular tissue	0	0	0	0	0	0	0	0	1	0	0	0	0	1
IX. Bones and locomotion	0	0	0	0	0	0	0	0	0	1	0	0	0	1
X. Malformation	0	1	0	0	0	0	0	0	0	0	0	0	0	1
XI. Early infancy	0	0	0	0	0	0	0	0	0	0	0	0	0	0
XII. Old age	0	0	0	0	0	0	0	0	0	0	0	0	0	0
XIII. External	0	0	0	1	0	3	16	2	2	1	1	0	0	26

TABLE NO. 3.—*Gross abnormalities—male*

1	1–4	5–9	10–14	15–19	20–29	30–39	40–49	50–59	60–69	70–79	80–89	90	Total		Percent
0	0	0	0	0	0	0	3	1	2	1	0	1	8	Prostate	1.3
0	0	0	1	0	0	3	4	6	1	3	0	1	19	Coronaries	3.1
0	0	0	0	2	2	3	7	0	5	1	0	0	20	Thymus	3.3
2	0	1	0	1	2	6	5	2	1	2	0	0	22	Skull	3.6
0	0	0	0	0	5	2	5	4	5	4	0	1	26	Ureter and bladder	4.3
1	1	0	0	3	4	12	6	4	2	2	0	0	35	Meninges	5.8
0	0	1	1	0	7	9	3	8	11	4	1	1	46	Adrenals	7.6
0	1	0	2	0	9	9	12	4	3	2	0	1	43	Stomach	7.1
1	1	2	0	3	5	16	9	3	5	5	0	0	48	Brain	7.9
0	0	0	1	1	7	14	12	3	8	2	0	0	48	Pancreas	7.9
1	1	0	0	4	12	11	17	6	3	10	0	0	65	Appendix	10.7
0	0	0	0	1	3	11	14	11	14	11	0	2	67	Testes and vesicle	11.5
1	2	0	1	4	13	31	14	10	4	5	0	0	85	Head and face	14.2
2	0	0	2	5	22	17	14	14	8	9	1	0	94	Pericardium	15.5
1	1	2	0	4	20	26	16	17	8	8	0	0	103	Trunk	17.0
0	0	0	0	2	18	25	21	14	16	11	0	2	109	Genital scar	18.0
2	3	4	1	10	29	32	16	9	19	6	1	0	132	Intestine and colon	21.7
1	1	3	2	7	26	28	21	16	15	11	1	1	133	Peritoneum	21.9
0	3	0	2	8	49	44	28	7	13	9	0	0	163	Mediastinal node	26.9
2	5	5	3	16	49	47	30	12	20	12	1	0	202	Retroperioneal node	33.3
2	6	5	7	13	53	50	33	10	21	12	1	0	213	Mesenteric node	35.1
0	1	0	1	0	23	47	58	38	40	27	3	4	242	Aorta	39.2
0	1	2	1	7	46	61	44	23	41	22	2	3	253	Extremities	42.5
1	1	1	2	6	48	55	51	33	46	22	3	1	270	Heart	44.5
2	3	1	1	9	68	62	56	34	34	21	5	2	298	Pleura, left	49.2
2	5	1	2	9	58	60	60	35	42	21	3	2	300	Spleen	49.5
2	1	1	3	11	66	71	55	36	35	18	4	2	305	Pleura, right	50.3
2	7	1	4	14	91	85	69	43	39	22	5	4	386	Lung, left	63.7
3	7	1	4	16	89	103	73	45	45	25	5	3	419	Lung, right	69.1
6	6	6	4	15	83	94	81	51	53	30	4	5	438	Liver	72.7
3	5	7	3	13	77	92	90	54	63	30	7	5	449	Kidney, left	74.1
3	6	7	4	12	87	98	92	56	60	31	7	5	468	Kidney, right	77.2

TABLE NO. 4.—*Gross abnormalities—female*

1	1–4	5–9	10–14	15–19	20–29	30–39	40–49	50–59	60–69	70–79	80–89	90	Total		Percent
0	0	0	0	0	0	1	0	2	1	2	0	0	6	Coronaries	1.5
0	0	0	1	0	2	1	2	1	0	0	0	0	7	Genitalia	1.7
0	1	0	0	0	1	2	1	1	0	1	0	1	8	Skull	2.0
0	1	0	0	2	2	1	0	0	2	2	0	0	10	Thymus	2.5
0	0	0	2	1	3	2	3	3	1	1	0	1	17	Meninges	4.2
0	0	0	0	1	2	0	5	4	3	4	0	0	19	Ureter and bladder	4.7
0	0	1	0	0	4	5	2	3	2	5	0	0	22	Adrenals	5.5
1	0	0	0	3	3	3	3	4	1	5	1	0	24	Stomach	6.0
0	0	0	2	2	2	5	4	4	1	2	1	1	24	Brain	6.0
0	0	0	0	4	10	5	4	1	2	3	0	0	29	Pancreas	7.2
1	2	1	1	0	8	11	8	4	4	4	2	0	46	Pericardium	11.4
0	1	2	1	3	17	10	6	6	0	3	1	1	51	Appendix	12.7
1	2	0	2	1	9	10	7	8	5	7	1	1	54	Head and face	13.4
0	0	1	2	0	19	20	9	7	1	8	0	0	67	Ovary and tubes	16.7
1	0	2	1	2	18	15	12	6	5	9	0	1	72	Trunk	18.0
0	0	0	1	1	15	22	12	8	8	9	1	0	77	Uterus	19.0
0	1	2	3	1	27	27	16	10	8	8	1	1	105	Peritoneum	26.1
0	1	1	6	9	29	24	16	6	9	4	2	1	108	Intestine and colon	26.9
0	4	2	1	6	32	36	15	9	4	5	0	2	116	Mediastinal node	28.9
0	4	3	4	11	39	29	17	13	7	4	1	1	133	Retroperitoneal node	33.8
0	4	4	4	12	39	35	18	11	9	4	1	1	142	Mesenteric node	35.3
0	1	2	2	6	36	32	22	14	17	13	4	1	150	Extremities	37.3
0	0	2	0	5	19	24	23	27	21	28	2	1	152	Aorta	37.8
0	1	2	2	11	40	41	20	13	10	13	3	1	157	Pleura, left	39.0
1	1	3	2	10	38	43	23	12	15	11	4	1	164	Pleura, right	40.8
1	1	2	2	8	24	38	29	23	14	25	4	3	169	Heart	42.0
2	1	3	3	11	39	47	28	18	12	18	2	1	185	Spleen	46.0
3	3	3	3	13	59	64	40	19	19	16	5	3	250	Lung, left	62.2
4	3	5	4	16	58	69	39	22	24	24	5	3	276	Lung, right	68.6
1	5	5	3	18	59	67	45	35	28	22	5	1	294	Liver	73.1
2	1	4	3	15	59	69	48	34	31	29	4	2	301	Kidney, right	74.8
2	1	6	3	16	53	70	49	33	33	30	4	2	302	Kidney, left	74.9

TABLE NO. 5.—*Microscopic abnormalities—male*

1	1–4	5–9	10–14	15–19	20–29	30–39	40–49	50–59	60–69	70–79	80–89	90	Total		Percent
1	0	1	2	3	7	4	6	5	3	0	0	0	31	Intestine	-----
2	1	2	0	5	4	18	11	5	2	2	0	0	52	Brain and meninges	-----
0	0	0	0	1	13	14	17	8	15	3	0	0	71	Pancreas	11.4
0	0	0	0	3	11	22	18	11	21	10	2	1	99	Testes	16.0
0	1	0	1	2	22	25	16	9	20	9	0	0	105	Adrenals	17.0
1	0	0	0	2	29	44	74	42	60	36	3	3	294	Aorta	47.7
4	3	5	5	14	63	74	55	35	49	19	1	1	328	Spleen	53.2
4	1	1	4	10	61	73	64	47	52	29	2	4	352	Heart	57.1
4	6	6	6	17	100	106	99	60	72	42	3	2	523	Liver	84.9
5	6	5	7	21	104	114	95	59	74	40	4	4	538	Lung and pleura	87.3
6	5	6	6	19	102	118	99	61	76	40	6	3	547	Kidney	88.8

TABLE NO. 6.—*Microscopic abnormalities—female*

1	1–4	5–9	10–14	15–19	20–29	30–39	40–49	50–59	60–69	70–79	80–89	90	Total		Percent
0	0	0	1	0	3	1	0	0	0	0	0	0	5	Tube and ovary	-----
0	0	0	0	0	6	3	3	1	0	1	0	0	14	Uterus	-----
1	0	0	0	3	2	1	4	5	0	4	1	0	21	Brain and meninges	-----
0	0	0	0	1	3	3	8	3	2	2	1	1	24	Pancreas	5.97
0	2	1	0	4	5	7	5	4	0	2	0	0	30	Intestine	-----
1	2	2	1	2	10	14	6	8	1	6	3	0	56	Adrenals	13.9
0	1	2	0	3	17	31	25	28	25	26	5	1	164	Aorta	40.8
2	5	5	2	6	49	49	31	18	18	19	8	0	212	Spleen	52.7
1	5	4	4	12	44	44	37	25	24	26	3	2	231	Heart	57.5
3	7	4	4	21	82	72	52	34	31	35	7	2	357	Liver	88.8
4	6	5	6	21	78	78	55	35	31	36	5	2	362	Lung and pleura	90.0
4	6	6	6	20	80	78	56	36	34	38	8	2	374	Kidney	93.0

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REFERENCES

(1) Turner, T. B., and Chambers, J. H.: Experimental Yaws, Bulletin Johns Hopkins Hospital, vol. 50, April 1932.

(2) Choisser, R. M.: Pathology in the Tropics, United States Naval Medical Bulletin, vol. 27, July–October 1929.

(3) Prof. Carl V. Weller: Personal communication.

(4) Turner, T. B.: Race and Sex Distribution of Lesions of Syphilis in 10,000 Cases, Bulletin Johns Hopkins Hospital, vol. 46, February 1930.

THE ETIOLOGY AND MANAGEMENT OF NEPHROLITHIASIS

By A. J. Desautels, Lieutenant Commander, Medical Corps, United States Navy

I feel that calculous disease of the urinary tract is, at the moment, the most timely subject which the urologist has to offer his colleagues, who are struggling with the problems and difficulties of their own various specialties. I have, therefore, chosen this opportunity to bring to your attention the hopeful progress being made in uncovering the basic etiology of urinary lithiasis, which has remained so obscure throughout the many centuries this condition has been known and described, and also to offer you an individual viewpoint of the current trend in the management of nephrolithiasis.

Surprisingly enough, the advent of the cystoscope, X-ray, and aseptic surgery, though affecting very radically other aspects of calculous disease, such as diagnosis, prognosis, and management, has brought about little or no change in the all-important matter of cause and prevention. Theories abounding in the ancient days, all unproven, of course, have, it is true, been discarded by the new knowledge brought to us by these modern agents of diagnosis and therapy, and other hypotheses have sprouted to replace them, most of these in turn to be thrown aside as they too were shown by facts to be untenable.

An entirely new era in lithiasis research was opened up in 1917 when Osborne and Mendel (*1*) made the important discovery that certain of their experimental animals, lacking an adequate supply of vitamin A, developed urinary stones. This monumental observation was surprisingly slow in making its impression, and it was not until 1926 that Fujimaki (*2*), at the Imperial State Institution for Nutrition in Japan, published his results, that a diet deficient in vitamin A regularly produced calculi in the bladder and kidney of experimental rats. Then followed several others, chiefly McCarrison (*3*) in India, and more recently Higgins (*4*), of the Crile Clinic in Cleveland, to confirm definitely this laboratory production of lithiasis in animals by vitamin starvation.

Joly (*5*), of England, addressing the American Urological Association at their Atlantic City convention last May, after a brilliant treatise on the etiology of stone, formulated the latest theory, based

on the above vitamin research: "The hypothesis," he states, "that stone is a deficiency disease is the most plausible and probable that has as yet been advanced. It explains not only all the principal features of the condition known today, but it also gives a reason for the changes in its incidence during the past years." By this he means the marked decline in the incidence of bladder stones in infants and children since the modern era has brought about its revolutionary changes in their feeding. "I believe," he continues, "that the vitamin starvation acts primarily on the renal epithelum and through it on the colloid mechanism of the urine. I also consider that once this mechanism is deranged, stone formation must follow as a direct result of the laws of physical chemistry."

Let us not forget that this pronouncement, momentous as it undoubtedly is, must nevertheless be considered purely theoretical, as its author admits earlier in the paper, with these words, "Its conclusions may be proved wrong by the next generation, but such proof will require additional knowledge."

Randall (*6*), of Philadelphia, discussing this paper, cautioned that the theories presented were all secondary factors in the formation of a stone, influencing that stone's chemical character and its growth, but that there still remained hidden a basic cause as yet unrecognized.

Barney (*7*), of Boston, continuing the discussion emphasized what is wholly unrelated to the vitamin theory, and barely mentioned by the author in his paper, and that is the thorough and practical clinical research going on at the Massachusetts General Hospital, striving to demonstrate the association of urinary lithiasis with parathyroid disease, in which efforts a remarkably close cooperation exists between surgeon, internist, pathologist, urologist, and biochemist. I have fortunately been privileged to observe some of the work this brilliant clinical research array has accomplished in demonstrating apparently that at least 10 percent of urinary lithiasis has parathyroid disease as the etiological factor.

I have only touched the surface of part of the work, that which seems the most important, which is being done to clear up the befogged etiology of urinary lithiasis, primarily to remind you that there remains much to be accomplished before we can hope to apply the wonderful preventive measures which will come only from the complete knowledge of an exact etiology. Happily, the day of this relatively important millenium does not seem so far distant, and with it will come a greatly needed change in the handicapped management of our stone cases.

I have made a rough classification of renal calculus patients, based on my own experience with a goodly number of them, the majority of whom came via the Veterans' Administration. This classification does not have the sound solid foundation of known etiology, but it

does have to recommend it and to justify its practical use the grief and regret of bitter lessons, perhaps more properly labeled errors in judgment.

I have in mind, and I propose them to you, three groups of kidney-stone patients. I feel reasonably certain that most of our cases will fit a niche in one of these groups.

In the first I place those patients, usually otherwise healthy young or middle-aged adults who come to us in acute renal colic, or with constant dull pain over the kidney, and in whom we find a single calculus lodged in the pelvis of the kidney or engaged in the ureter. It is largely a matter of chance whether this particular patient passes the stone spontaneously or whether surgical intervention will be necessary. He does not worry us greatly and offers a simple problem. If the calculus is loose in the pelvis and too large to pass into the ureter, we recommend a definitely indicated pyelolithotomy to relieve him of his distressing symptoms and to save the kidney from probable progressive hydronephrotic change. The operation is not difficult, the risk is small, convalescence is usually uneventful, and cure is effected. We are permitted considerable leeway in this group of cases, and are justified in delaying surgical intervention if our studies reveal the likelihood that the obstructing calculus will move along toward spontaneous passage, meanwhile carrying out the various measures afforded by the cystoscope, ureteral catheters and stone dislodgers to assist the descent. There is no definite time limit when these ureteral maneuvers must give way to surgical interference. In this group we find no anatomical defect, such as ureteral stricture or aberrant vessel, to account for the formation of calculus because of stasis, there is usually no associated urinary infection, and the etiology is extremely obscure. The well-developed and well-nourished appearance of these patients discounts in them the vitamin starvation theory. We do know that stone rarely recurs in this group, either after its passage or surgical removal, and we can fairly safely assume that cure has been achieved with its disappearance.

In the second group I include those cases with a small silent non-obstructing calculus lodged in a calyx of the kidney, the finding of which is often accidental, or incidental to other examinations. These stones have all the salient features of the first group, except location. They too usually occur in well-nourished adults, and their etiology is also absolutely obscure. Their formation, as a rule, takes place in a lower calyx where the force of gravity works against their expulsion into the renal pelvis. They rarely cause symptoms; any subjective distress is probably due to the breaking off of a small piece, which finds its way into the pelvis and thence into the ureter, with resultant colic. The problem then becomes the same as in the first group. They are, therefore, only potentially menacing. They should, I

believe, be left alone, just as are other inaccessible harmless foreign bodies. There are naturally exceptional cases in which surgical removal is indicated by accompanying persistent infection, with the clinical picture of pyelonephritis.

We now come to the very important third group of my broad classification of kidney stones. These cases offer the urologist his most disconcerting problems, and present the most controversial aspect of calculous urinary disease. They include practically all multiple stones, cases with massive calculus formation, bilateral nephrolithiasis, patients with demonstrable stasis and constant infection, rapidly forming stones occurring in undernourished and obese patients, poor surgical risks, anomalous individuals, people whose glands of internal secretion seem out of control. These are the veritable calcium malignancies of the upper urinary tract, and it is this group that an unfolded etiology would benefit most materially.

Statistics reveal that following operative removal of kidney stones reformation occurs in approximately one-third of the patients. The recurrences are those reported by the most accredited of urological surgeons. It is not an exaggerated estimate, and there have been higher proportions bravely submitted. My observations lead me to suspect that this amazingly high percentage of recurrence is almost exclusively made up of this large difficult third group, and that if the figures for this group alone were known they would be unbelievably discouraging.

The problems offered for our solution by this large complex group of kidney-stone patients are indeed difficult. Our responsibility for them is inescapable, for they cannot be managed by the dictum of others. Let us always grant them the full exercise of our most thoughtful judgment, withheld until all the facts are known and carefully weighed, rather than ever plunge headlong into a decision precipitated by an illusory finding. May I cite, as an example, that most astounding paradox of a kidney filled with calculi, forming the familiar staghorn cast of the renal pelvis and calyces, and yet maintaining normal functioning capacity, structural integrity, and freedom from infection? Nature seems to bestow upon many of these stone-bearing kidneys her most bountiful favor, providing an unobstructed zone around the calcareous mass which permits freedom of the urinary flow from its source in the renal parenchyma to its outlet in the ureter. The lure of surgery in these cases is great, in spite of the subjective and physical silence of the massive lithiasis and the unimpaired renal structure and function. I have become firmly convinced that surgical interference under these circumstances is both meddlesome and foolhardy. I am not intimating that all staghorn calculus kidneys are so blessed, nor do I contend that they should never be operated. Their problems, as are those of this entire malignant group of nephro-

lithiasis, are extremely variable and require the most diligent individual study before we can hope to pass the mature sound judgment which their gravity merits. When, in managing patients of this group, we deem that surgery is indicated, either in emergency or for the best ultimate outlook, we should proceed cautiously with our operative procedure. The choice between nephrectomy and nephrolithotomy is often a delicate one to make in these cases, where renal structure and function are so precious. The immediate risk of operation is much greater in these patients than in our other two groups, and there is no place in this realm for the careless or inexperienced surgeon.

REFERENCES

(1) Osborne and Mendel: Jour. Amer. Med. Assoc., 1917, lxix, 32.
(2) Quoted in the Lancet, July 30, 1927, p. 239.
(3) McCarrison: Causation of Stone in India, Brit. Med. Jour. 1931, i, 1009.
(4) Higgins: The Experimental Production of Urinary Calculi in Rats. Urol. and Cutan. Review, January 1934, 33.
(5) Joly: The Etiology of Stone. Jour. of Urol., December 1934, 541.
(6) Quoted in the Jour. of Urol., December 1934, p. 571.
(7) Quoted in the Jour. of Urol., December 1934, p. 573.

PAROXYSMAL HEMOGLOBINURIA

With Report of a Case Followed for 13 Years

By J. G. Dickson, Lieutenant Commander, Medical Corps, United States Navy

Paroxysmal hemoglobinuria is a rare condition generally accepted to be a late manifestation of syphilis, either congenital or acquired, is characterized by transient attacks of hemoglobinuria almost always following exposure to cold and is due to the existence in the patient's blood of a hemolysin which hemolyzes his own red blood cells.

In 30 years among some 156,000 ward patients at the Massachusetts General Hospital there were only nine cases (*1*); among 74,186 consecutive admissions to the Peter Bent Brigham Hospital only three cases were recorded; while at the Boston City Hospital there had never been a case up to 1931 (*2*). McCarthy and Wilson (*3*) say that less than 300 cases have been reported.

The presence of the characteristic hemolysin on which the symptoms of this very interesting condition are based was first demonstrated in vivo by Ehrlich by the immersion of the ligated finger of a patient in ice water producing hemoglobinemia locally. The commonly used in vivo test is that of Rosenbach in which hemoglobinuria is induced by an ice foot bath. In vitro the hemolysin can be demonstrated by the classical test of Donath and Landsteiner in which with chilling and then warming appropriate mixtures of cells and serum, the serum of the patient hemolyzes not only his own but the red blood cells of

a normal individual. Thus there are both an auto- and an iso-hemolysin. They are believed to be identical.

Apparently the hemolysin exists ordinarily in an inactive form and and in some obscure manner is activated by cold upon which it unites with the red cells, hemolysis occurring upon subsequent warming and through the mediation of complement. This is the generally accepted hypothesis. However, the conception of any biological process being initiated by cold runs counter to the practically universal experience of physiology, cold being an inactivating or inhibiting agent. Two other possibilities may be suggested: That the hemolysin is prevented from acting at body temperature by an antihemolysin which is, itself, inactivated by cold; or that the red-cell membrane is permeable to the hemolysin only in the cold.

Because of the association of the attacks with chilling, the symptoms are nearly always confined to winter. Mackenzie (*4*) however, cites several cases from the literature in which exertion without exposure to cold initiated attacks. One of his patients had numerous spontaneous attacks while in bed in a well-heated ward and was seen in an attack in mid-July in New York City. These cases are paradoxical and far from explained.

The appearance of hemoglobinuria is dependent upon hemolysis of sufficient red cells to produce hemoglobinemia above the renal threshold. Destruction of about one-sixtieth of the red cells of the body is believed to be necessary. During an attack the red cell count may drop as much as 1,000,000. Thus anemia may be produced by frequent or severe attacks. There is often a fall in the white cells during the early stages of an attack quickly followed by a leukocytosis with increase in percentage of neutrophils.

For years it has been recognized that paroxysmal hemoglobinuria is closely related to syphilis. Cooke (*5*) in 1912, only 6 years after the discovery of the complement fixation test for syphilis, was able to collect 37 cases from the literature in which the Wassermann was recorded. It was positive in 33 or 89 percent. This is very significant considering the refinement of the serum test for syphilis in the past quarter century. In recent years reports without a positive Wassermann are almost unknown. Today it is almost unanimously agreed that syphilis is the sole cause of the condition. Such was the opinion of Dr. Kolmer in 1922 (*6*). Burmeister (*7*) believed that he had demonstrated the hemolysin to be identical with the Wassermann producing substance and he thought that it might be produced by some other agency than syphilis in paroxysmal hemoglobinuria. Smith (*8*), Puris (*9*), and Mackenzie (*4*) were all unable to confirm Burmeister's results and found the hemolysin and the substance responsible for the Wassermann reaction to be separate and distinct. This was confirmed by Thurmon and Blain (*2*) who also showed that the hemo-

lysin was separate from the substances producing the Hinton and modified Kahn reactions. Mackenzie (*4*), who probably has done more research on paroxysmal hemoglobinuria than any other modern authority, states that the condition is usually and perhaps always a manifestation of syphilis. Purvis (*9*) says that the condition is probably due to syphilis but there is a reasonable doubt whether the positive Wassermann of these cases is really due to syphilis.

Joltrain (*10*) has recently reported two cases of hemoglobinuria following blood transfusion. Wassermann was negative in both cases by several methods. In each case the Donath and Landsteiner test was positive not only with the red cells of the patient but also with the red cells of a normal control. The first patient showed no apparent clinical evidence of syphilis. The second patient, a woman of 36, had bilateral recurring hemorrhages into vitreous and retina. There was history of a miscarriage. Syphilis was suspected. An error in blood grouping was discovered following the transfusion (by direct method). During the attack the patient had urticaria and local syncope and asphyxia of the hands, similar to Raynaud's disease. Such symptoms have frequently been reported in paroxysmal hemoglobinuria. This case was probably syphilitic. Such reports have not been encountered elsewhere. It is still safe to say that hemoglobinuria following exposure to cold and with a positive Donath and Landsteiner reaction, in the absence of yaws, is pathognomonic of syphilis.

It is interesting to note that numerous instances have been recorded where the hemolysin of paroxysmal hemoglobinuria has been demonstrated by the Donath and Landsteiner reaction in certain syphilitics who had never had an attack. Thus Donath and Landsteiner (*11*) performed their test on the blood of 93 paretics, only 1 of whom had paroxysmal hemoglobinuria, and found it positive in 7. Two of these showed a positive Ehrlich test. Kumagai and Namba (*12*) studied 35 late luetics and demonstrated the presence of the hemolysin in 7, all of whom also showed a positive Ehrlich test. In one of these cases a cold foot bath produced hemoglobinuria. Thurmon and Blain (*2*) found in the half brother of a congenital luetic with paroxysmal hemoglobinuria, a positive Donath and Landsteiner reaction though the patient had never shown hemoglobinuria, there were no other stigmata of syphilis and Wassermann, Kahn, and Hinton tests were all negative. McCarthy and Wilson (*3*) reported in two younger sisters of a congenital luetic with paroxysmal hemoglobinuria, a positive Donath and Landsteiner test though both showed a negative Wassermann.

It would be of great interest if paroxysmal hemoglobinuria should be found to occur in yaws in view of the close biological kinship between that condition and syphilis. However, reports of paroxysmal hemoglobinuria occurring in the tropics have not been encountered.

This is not surprising considering the rarity of the condition and its association with cold weather. But attacks may occur at times from slight chilling and the condition might be encountered at high altitudes in the tropics. The possibility of the presence of the hemolysin in yaws should be investigated.

The treatment of paroxysmal hemoglobinuria is, naturally, the treatment of syphilis. Kumagai and Namba (*12*) gave 14 patients antiluetic treatment. Two were lost to observation, 1 died, 1 was still under treatment at time of writing, and 10 were clinically cured. Thurmon and Blain (*2*) reported a case that had been followed 14 years and had been clinically cured for 12 years following antisyphilitic treatment and another that had been improved for 2 years. Two of Mackenzie's patients after thorough antiluetic treatment passed through two consecutive winters in New York City without an attack (*13*). Although the available reports of cases followed for prolonged periods are extremely rare, it appears that the condition per se causes little damage to the patient and there is no evidence that he is any poorer life insurance risk than any other syphilitic. Mackenzie (*4*) observed a patient who had attacks for 23 years with no impairment in health.

Treatment of the condition by inoculation with rat-bite fever was tried by Inamori (*14*). The fever was arrested by a course of neoarsphenamine. This patient remained clinically cured when last seen 2 years later but an ice foot bath induced hemoglobinuria (*15*). The same treatment was tried by Ito (*16*), the fever being cured by a course of sulpharsphenamine. The attacks, in winter, were arrested for 2 months when the patient was last seen. It is difficult to see any value in this treatment other than from the arsphenamines used to stop the rat-bite fever.

It is very probable that there are less than a dozen cases of paroxysmal hemoglobinuria on record that have been followed for 10 years or longer. Therefore it is believed that the following case should be recorded:

Clinical history.—The patient, a white male, aged 40, a member of United States Navy for the past 24 years, first came under observation January 11, 1922, complaining of chills and bloody urination following exposure to cold. He dated the present illness to the summer of 1918 when, on duty of mine planting in the North Sea, he became abnormally susceptible to cold. Regardless of how heavily he clothed himself, after the watches on the bridge he usually had a severe chill from which it took from 10 to 15 minutes to get warm. In November 1919, after his return to the United States (in Portsmouth, N. H.), the chills following exposure to cold first became associated with bloody urination and this condition has continued until the present. During the winter of a year ago (1920–21) he began having moderate fever with the chills. Throughout the first two winters the attacks were quite frequent, averaging at least one each week during cold weather. In the present winter (the patient being stationed in Virginia) the attacks have been much less severe and decidedly less frequent.

He has had only about six so far this winter. He has never voided bloody urine but once during any one attack and he was quite positive that he had never voided bloody urine except after exposure to cold and never in the summertime. The attacks have never caused him to lose an hour from work. He has never had any pain whatever during the attacks. He never had any pain on urination, no frequency, no nocturia. There were no sysmptoms referable to any other body system. His familial and past history were entirely irrevelant to his present illness. He thought that he had never had malaria but stated that he had been given huge doses of quinine because of his bloody urination. The patient repeatedly and emphatically denied ever having had any venereal disease whatsoever.[1]

Physical examination.—Patient was well developed and well nourished and did not appear to be ill. Face very florid. Pupils were somewhat sluggish in their reaction to light. The left epitrochlear lymph node was palpable. Elbow, knee, and ankle jerks were all present, normal and equal. Head, neck, mouth, teeth, and throat were all grossly normal. Heart and lungs were normal to routine examination. Neither the liver nor the spleen could be palpated and the abdomen was negative throughout. No tenderness over the kidneys. External genitalia normal; no scars. No palpable sclerosis of rhe arteries. Blood pressure 124/80; pulse rate 82.

Laboratory examinations.—[2] The blood showed: red cells 4,340,000 per cubic millimeter, hemoglobin 90 percent (Tallqvist); white cells 16,500 with neutrophils 53.5 percent, lymphocytes 41 percent, and mononuclears 5.5 percent. On another occasion the white blood count was 18,000. The stained blood smear showed no abnormalities and no malaria parasites could be found. Urinalysis at this time was entirely negative chemically and microscopically.

One week prior to the examination as above recorded the patient brought in a specimen of urine voided the night before during a light chill following exposure to cold. This urine was of a port-wine color, acid, with a specific gravity of 1.025, and showed a heavy cloud of albumin (by heat and acetic acid), no sugar. There were many finely and coarsely granular casts but no red or white cells were seen.

On January 24, 1922, it had been planned to induce an attack by giving the patient an ice foot bath (Rosenbach test) but the patient reported to the dispensary after a walk of about a mile in freezing weather and immediately voided port-wine-colored urine. This had the same properties and reactions as the hemoglobinuric specimen previously examined. In addition, the benzidine test for blood was strongly positive and no red blood cells could be found microscopically. At this time the patient felt perfectly well, had no chill or chilly sensations, and the temperature was normal. White blood cells numbered 14,600 per cubic millimeter. The coagulation time was 8 minutes. Bloody urine was voided only once during this attack.

Blood Wassermann tests were done by three different laboratories on three separate occasions: (1) doubtful, (2) strongly positive (ice box incubation), and (3) strongly positive (Kolmer Wassermann performed by Dr. Kolmer).

Red cell fragility test showed a normal reaction: hemolysis began at 0.43 percent and was complete at 0.27 percent of sodium chloride in distilled water.

[1] Transcript of patient's medical record for his entire service was obtained from the Bureau of Medicine and Surgery on Dec. 9, 1932, and showed the following pertinent entries: (*a*) patient was on the sick list from Mar. 27 until May 4, 1901 (38 days), with diagnosis of "syphilis consecutiva", with a large nonsuppurating bubo in the left groin, specific eruption and mucous patches. Primary lesion not described. Patient given "specific treatment" (no details) and discharged to duty as well; (*b*) on sick list from Nov. 14 to 21, 1920 (7 days), with diagnosis of malaria-chills, temperature of 102, and history of repeated chills during previous summer. Intensive quinine treatment for a 3 months period started.

[2] All tests were done between attacks except where noted to the contrary.

The hemolysin in the patient's blood was demonstrated by the reaction of Donath and Landsteiner. There was marked hemolysis of the red cells of the patient as well as of those of a normal control.

On March 20, 1922, the patient was transferred to the United States Naval Hospital, Washington, D. C. The Donath and Landsteiner test was repeated and found to be positive. In addition the patient was placed in an ice box at 5° C. (41° F.) for three-fourths hour. He had a slight chill soon after coming out and a half hour later passed port-wine-colored urine. The urine was clear before and after this single passage. The test was repeated the following day with identical results. The Wassermann was positive on three separate occasions. The white blood count was 13,950. Spinal fluid examination showed a cell count of three, globulin negative, Wassermann negative, collidal gold curve normal. Complete blood chemistry determination including plasma CO_2 and diastatic activity showed normal results except that blood uric acid was 4.0 mg per 100 cc of whole blood.

The patient was given injections of mercury succinimide and neoarsphenamine and discharged to duty after three of each had been given with recommendation that five more injections of neoarsphenamine be given before allowing a rest period.

The patient has not been seen since 1922 but on April 15, 1935, he wrote that he is now (at age of 54) in excellent health. He says that he has never had any "bloody discharges" and that he has received no medication internally or by injection since leaving the hospital in 1922. This is confirmed by the abstract of his medical record up to the date of his retirement in 1930. His record remained entirely clear except for a laceration of the face in 1927. He was again on duty in Virginia during the greater part of three winters from 1925 to 1928.

COMMENT

Here was a typical case of paroxysmal hemoglobinuria of rather mild type as the patient was never incapacitated, only a single voiding of urine was ever discolored and the vasomotor phenomena, such as Raynaud's syndrome and urticaria, were not observed. The presence of syphilis was proved by history and a repeatedly positive Wassermann. There were several interesting features. The patient concealed or forgot having had syphilis 21 years before. There was slight confusion at first by a "doubtful" Wassermann. The constant leukocytosis remains unexplained. Apparently this finding has not been described before. It is not unlikely that the chills suffered by the patient in the summer of 1918 (more than a year before the first appearance of frank hemoglobinuria) were caused by larval attacks in which the hemoglobinemia did not exceed the renal threshold. A clinical cure lasting, so far, for 13 years was attained with little treatment. This is not unreasonable considering the mildness of the attacks.

REFERENCES

(1) Jones, B. B., and Jones, C. M.: Paroxysmal Hemoglobinuria, in Nelson's Loose-Leaf Living Medicine, New York, Thomas Nelson and Sons, 1920–29. vol. 4. p. 173A.

(2) Thurmon, F., and Blain, D.: Paroxysmal Hemoglobinuria, Observations Based upon a Study of Three Cases, Am. J. Syph. 15: 350 (July) 1931.

(3) McCarthy, F. P., and Wilson, R. Jr.: Paroxysmal Hemoglobinuria. Report of Cases with Familial Findings, New England J. Med. 207: 1019 (Dec. 8) 1932.

(4) Mackenzie, G. M.: Paroxysmal Hemoglobinuria. A Review, Medicine 8: 159 (May) 1929.

(5) Cooke, R. A.: Paroxysmal Hemoglobinuria, Am. J. M. Sc. 144: 203 (Aug.) 1912.

(6) Kolmer, J. A.: Personal communication to the author.

(7) Burmeister, cited by Purvis (9).

(8) Smith, Cited by Purvis (9).

(9) Purvis, A. M.: Paroxysmal Hemoglobinuria, Am. J. Dis. Child. 37: 1027 (May) 1929.

(10) Joltrain, E.: A propos de deux cas d'hémglobinurie paroxystique après transfusion sanguine, Sang 6: 455 (No. 4) 1932.

(11) Donath and Landsteiner, cited by Mackenzie (4).

(12) Kumagai and Namba, cited by Mackenzie (4).

(13) Mackenzie, G. M.: Paroxysmal Hemoglobinuria, in Cecil's Text Book of Medicine, ed. 3, Phila., W. B. Saunders Co., 1934, p. 964.

(14) Inamori, S.: Paroxysmal Hemoglobinuria Cured by Inoculation of Spirochaeta Morsus-Muris, Am. J. Dis. Child. 44: 379 (Aug.) 1932.

(15) Note in later article by Ito (16).

(16) Ito, N.: Paroxysmal Hemoglobinuria. Treatment by Inoculation of Spirochaeta Mosus-Muris, Am. J. Dis. Child. 46: 1062 (Nov., pt. 1) 1933.

THE CIVILIAN CONSERVATION CORPS: RÉSUMÉ OF A TOUR OF DUTY

By R. A. Bell, Lieutenant, Medical Corps, United States Navy.

The Act to establish the Civilian Conservation Corps, conceived in the mind of President Franklin D. Roosevelt and created by act of Congress (*1*), gives the President of the United States wide authority. He appointed Robert Fechner as Director of Emergency Conservation Work to execute it in his name. The Director is assisted by an advisory council consisting of one representative from each of the following Federal Departments: Labor, War, Agriculture, and Interior.

This relief project has four main functions: (1) Relief of unemployment, (2) provision of gainful employment for needy American youths, (3) conservation of natural resources, (4) education of the Civilian Conservation Corps enrollees.

The Labor Department has charge of the selection and certification of the enrollees.

The Agriculture and Interior Departments have charge of the conservation and resources. They establish work projects and have complete supervision of the enrollees while engaged thereon.

The War Department function is to—

(*a*) Accept the men selected by the Department of Labor and the Veterans' Administration.

(*b*) Examine physically and to enroll qualified selectees into the Civilian Conservation Corps.

(*c*) Command the Civilian Conservation Corps, from the time of acceptance of the man until his final discharge, embracing all the functions of reconditioning organization, administration, transportation, supply, sanitation, medical care, hospitalization, discipline, welfare, and education.

(*d*) Construct work camps and all new installations necessary to fulfill the functions listed under (*c*) above.

(*e*) Furnish work details from work companies to project superintendents.

(*f*) Demobilize the Civilian Conservation Corps in accordance with instructions to be issued at the proper time.

These functions have been decentralized through corps areas, districts, subdistricts, and, finally, camps. The camp commander exercises the normal functions of the company commander in the Army, omitting only that of military training, and he must temper all his actions to conform to the civilian status of the enrollee.

Each camp commander has assigned to his staff one or two line officers and one medical officer. As originally contemplated the companies were organized and located in the field under command of Regular Army officers and certain naval line and marine officers, most of whom were largely replaced by Reserve Army officers by Christmas of 1933. Since that time Regular Army personnel have been assigned to C. C. C. work only in a supervisory capacity.

At the very outset of the Civilian Conservation Corps program the lack of a sufficient number of available Regular and Reserve Army medical officers to properly staff the field companies became apparent. To meet this demand the President attached certain naval medical officers to the War Department (*2*) for duty with the Civilian Conservation Corps. These officers were distributed throughout the country under the jurisdiction of the various corps areas of the Army. The duties they were called upon to perform included, in addition to those required of a medical officer on field duty with troops, an extensive series of health lectures, training of enrollees in various phases of camp life, instruction of enrollees in connection with the C. C. C. educational program, public relations activities in connection with camp sanitary installations, provisions, and hospitalization of enrollees, and at times the administration of the camp mess or the camp exchange. This work as it pertained to the individual camp surgeon was reported upon by Buddington (*3*).

The subdistrict organization as established in the Third Corps Area, originally designed to function in an advisory capacity, was composed of a Regular Army field officer and a Regular Army medical officer. The Army field officers were soon replaced by Regular Army captains, the Army medical officers by naval medical officers and a Reserve chaplain was detailed to the staff.

In this position the medical officer, as the field representative of the corps area surgeon, served in an advisory capacity to the subdistrict commander on all matters pertaining to hygiene and sanita-

tion, advised and instructed camp surgeons as to their duties, and submitted routine and special reports to the corps area surgeon. The subdistrict staff covered from 6 to 14 C. C. C. camps, visited them weekly, and more often if special conditions warranted. The outgrowth of this advisory leadership was a loosely knit, individualistic, work camp organization with not too high standards. In May 1934 headquarters, Third Corps Area, issued a letter (*4*) of instruction to all district and subdistrict commanders in the Third Corps Area stating in part, "the C. C. C. camps will cease to exist as a mere series of work camps and hereafter will conform to the standards of Regular Army organizations." The subdistrict office became an office of record and the duties of the subdistrict commander were changed from advisory to commanding. This change resulted in a decided improvement in all phases of camp activity. Standards were raised, morale among officers and enrollee personnel improved and the C. C. C. became a more smoothly functioning organization. The work of the subdistrict staff was greatly increased, and, to effectively administer his many duties, the subdistrict commander necessarily detailed much work to the medical officer, with authority (*5*) to issue orders relative to the correction of sanitary deficiencies. Thus the surgeon became responsible for general supervision of the sanitation in all of the camps in his subdistrict.

To properly interpret the significance of these duties, it is necessary to study the personnel, officers, and enrollees, of the Civilian Conservation Corps in the field. This is no reflection upon the ability of the Reserve officers who have done and are doing an excellent job. They were suddenly called upon to fill a position which is not their life work and for which they have had very little specific training—the older group, in many cases, forced out of gaintul employment in civilian life by the depression and the younger group recently graduated from colleges. With standards being raised and policies established it became necessary to "show the way" in many cases. The rapid turnover in enrolled personnel, originally allowed to remain for only 6 months, necessitated the continual training of keymen. The C. C. C. enrollees, coming from families on relief, were largely unqualified for the special duties incident to camp administration. Recognition of this problem of special duty training resulted in the gradual liberalization of mandatory discharge regulations exempting keymen in company and administrative organizations from mandatory discharge due to length of service. Finally in July 1935 in order to more adequately serve the relief needs of the country the President directed the revision of regulations requiring mandatory discharge for length of service. An example of the confusion resulting from untrained clerks is worth reciting. One company commander, repeatedly called upon by corps area for copies of various correspond-

ence which should have been received over a period of several months, knowing that he had signed these communications presumed the mail delivery was at fault and forwarded duplicate copies in each case. In going through the company files sometime later he discovered that the new clerk had filed all copies of the various letters.

In molding the camp personnel into an efficient smoothly functioning organization it was necessary to teach each individual, commissioned and enrolled, the chain of command, individual scope of authority, and individual duties. Most difficult of all was making leaders out of enrollees who, unaccustomed to acting as leaders and hampered by the opinions of their fellows, were hesitant in accepting the authority to enforce regulations upon their respective sections and squads. Once this chain of command was established and the personnel trained, very little difficulty was experienced with the camp, whereas without an efficient organization reaching down to the individual enrollee, the camp was always backward. The responsibility of establishing such a chain of command rested with the subdistrict commander but much of it was delegated to other officers.

In the inspection of a camp, deficiencies noted were called to the attention of the commanding officer, means of correction were discussed, responsibility fixed and a time limit for completion established. Upon our return to the office a list of the deficiencies and proposed improvements was mailed to the camp commander, thus giving him a definite list of defects and providing a status file on the camp. To demonstrate the need of such minute supervision let me relate a few of the sanitary deficiencies noted in a subdistrict to which I was transferred. In one case three C. C. C. companies received their water from a pipe line which extended some 5 miles back into the hills, to an artificial pond, about the shores of which were dotted the outhouses of various mountaineers. The unfiltered water, ostensibly chlorinated at the source, showed heavy contamination of samples taken in the camp and from the pond. The chlorine gas was bubbling into the lake and not into the pipe line. In another camp we were vigorously assured that the water supply was excellent, "for look, it is piped into camp from a mountain spring up yonder." Investigation proved the "spring" to be a cattle watering hole in a gulley. In one camp the enrollees were in the habit of pitching cigarette stubs into the aisle. On my first inspection, about two dozen smouldering stubs and hundreds of charred spots were noted on the floor of a barracks.

These experiences could be related indefinitely. In many cases proposed changes were opposed by local dealers of foodstuffs. Only pasteurized milk and only inspected and approved meats were served.

The educational aspect of this work has been designed to impart such instruction as will foster the returning to the normal work-a-day

world, upon completion of the emergency relief project, citizens better equipped mentally and morally for their duties as such and with a better knowledge of the Government under which they live, and of all that that Government means (*6*).

This program as taught in the Third Corps Area, known as the five-point program, embraces: (1) Health, individual, family, and group. The courses, including personal hygiene, first aid, food, posture, contagious diseases, home and community sanitation and health principles and responsibilities, have been largely handled by the medical officers. (2) Everyday affairs—a study of newspapers, magazines, radio broadcasts and motion pictures with an interpretation of the meaning of the "news." This has been impartial, nonpolitical, and nonsectarian. (3) Human relationships—an appreciation of social relationship, inculcation of sense of responsibility to duly constituted authority, codes of ethics, courtesy, tact, initiative, cooperation, leadership, team play. (4) Instruction, academic and vocational elimination of illiteracy, classes in basic subjects and practical instruction in such subjects as woodcraft or motor mechanics. (5) Recreation and leisure time—the development of avocations, importance of reading hours, athletics and discussion groups. With the acceptance of shorter hours for the laboring classes this rightly takes cognizance of a national social problem.

It has been the goal to raise the educational level of each enrollee at least one grade during an enrollment period of 6 months. Arrangements have been made with schools throughout the corps area by which a discharged enrollee, upon presentation of his certificate, may receive credit for work accomplished (*7*). That this program has functioned effectively is well illustrated by the fact that private industry is giving preference to C. C. C. trained men. In June 1935 over 17,000 enrollees left the C. C. C. to accept jobs with private industry.[1]

Many observers are inclined to feel that the educational program of the Civilian Conservation Corps will pay greater dividends to the Nation than the conservation program which up to this time has received more attention and financial encouragement. A properly instituted educational program, thoughtfully executed, should perpetuate itself and pay dividends through encouragement of private initiative in conservation of resources, slum clearance, playground and park development, and the like. Such a program should have far-reaching effects upon the national psychology and initiative in addition to relieving distress among the indigent and furnishing an occupation for the Nation's youth.

[1] These figures should be accepted with reservations, for certain enrollees resign in order to return to their homes. The summer harvest beginning in June recalled a large number for temporary employment and data relative to the total number of employees taken on the national wage roll in June should be compared.

The purpose of this paper has been to present, in a general way, the organization of the Civilian Conservation Corps and the relation of the medical officers to the development and growth of this relief measure. With the detailing in February 1935 of one Reserve officer to each subdistrict staff, to function as an adjutant, the medical officer has discontinued the performance of any duties he may have been doing which are normally handled by the adjutant. On this tour of duty the job has been, in no small measure, just what we chose to make it. Far from being a poor assignment it has furnished a training and experience in (1) field hygiene and sanitation, (2) contagious diseases and preventive medicine, (3) administrative medicine, (4) detached duty, (5) post-exchange administration, (6) Army administration, (7) official channels of communication, (8) the relation of the medical officer to the line officer, (9) civilian relationships, (10) public speaking, (11) social problems and planning, (12) relief problems, (13) leadership, (14) group welfare, and (15) menu planning.

In closing, a word of appreciation for the Army personnel, splendid gentlemen and efficient officers. No more adequate tribute can be paid than to point to the success of the Civilian Conservation Corps, the administration of which has been largely responsible for its universal public esteem.

REFERENCES

(1) Entitled "An Act for the Relief of Unemployment Through the Performance of Useful Public Works, and for Other Purposes", approved March 31, 1933 (48 Stat. 22; U. S. C. 16; 585).

(2) Executive Order No. 6331–A, dated May 13, 1933.

(3) U. S. Naval Medical Bulletin, Vol. 32, 3rd issue, 1934, page 283.

(4) Letter, headquarters, Third Corps Area, U. S. A., Baltimore, Md., dated May 14, 1934, File No. 354–CCC (Camp Conditions) 20 (5–14–34).

(5) War Department Regulations, Relief of Unemployment, Civilian Conservation Corps, dated May 15, 1935, Section VIII, Par. 103 c, page 86.

(6) War Department Regulations, Relief of Unemployment, Civilian Conservation Corps, dated May 15, 1935, Section IX, Par. 141, page 114.

(7) Personal communication from Dr. Thomas Bennett, Educational Adviser for the Third Corps Area, U. S. Army.

SURFACE DECOMPRESSION OF DIVERS [1] [2]

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The decompression of divers following a period of work on the bottom is usually accomplished by bringing the diver to the surface

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[2] We wish to express our appreciation for the suggestions and advice by the Advisory Committee: Capt. E. W. Brown, Medical Corps, U. S. Navy; Comdr. E. L. Gayhart, Construction Corps, U. S. Navy; and Comdr. H. E. Saunders, Construction Corps, U. S. Navy, formerly a member of the committee.

We also recognize the assistance rendered by Lt. R. W. Clark, U. S. Navy; and Lt. R. A. Hansen, U. S. Navy, former officers in charge of the Experimental Diving Unit.

at the rate of 50 feet per minute with stops at various depths in accordance with the regular rules of decompression, for the depth and time of exposure, laid down by the United States Navy Diving Manual (*1*).

Frequently, however, in cases of emergency, divers have been brought immediately to the surface and placed in a recompression chamber where the pressure is raised to the equivalent of the first stop in the water and the diver is given the decompression he would have received if in the water. Heretofore this procedure has been referred to as "water-stage elimination decompression", but due to the awkwardness and ambiguity of the expression, the opportunity is taken to suggest a simpler term, i. e., surface decompression.

In the United States naval service on the occasion of the salvage of the U. S. S. *S–4* (*2*) partial as well as complete surface decompression was used. In partial surface decompression the diver makes his regular stops at the deeper depths by remaining on the stage and then is brought directly to the surface and placed in the recompression chamber where pressure is raised to the point of the last stage completed in the water and decompression is completed according to schedule. This practice was carried out for some time but finally complete surface decompression was adopted because of the coldness of the water. In this work, however, the regular diving table, as laid down by the Diving Manual (*1*), was modified and lengthened.

The practice of surface decompression is desirable, for (*a*) it gets the diver out of the cold water and tides into a warm chamber where he can be more easily cared for; and (*b*) it releases tenders and diving gear so another diver can be sent down sooner. Thus diving operations can be materially speeded up.

Because of the lack of previously recorded data a series of experiments were undertaken at the Experimental Diving Unit, Department of Construction and Repair, Navy Yard, Washington, D. C., to demonstrate conclusively the practicability of the procedure of surface decompression.

Apparatus.—Standard diving suits were worn by the men. The diving operations were carried out in a diving tank described by Hawkins, Shilling, and Hansen (*3*) and the recompression was carried out in the recompression chamber also described by Hawkins, Shilling, and Hansen (*3*).

Method.—The method may best be described by following one run completely through: i. e., run no. 15–A on November 2, 1933, at 150-foot depth with length of exposure of 50 minutes. The two divers were dressed and lowered into the diving tank, the hatch closed, and pressure applied at zero time. Reached bottom in 3 minutes. While on the bottom for the remaining 47 minutes of the 50-minute total exposure the men worked putting in and taking out bolts on a sheet

of iron clamped to an iron workbench. At 50 minutes the air pressure on the diving tank was released at such a rate that the two divers reached 100 feet at 51, 50 feet at 51:58 and 10 feet at 52:40. The hatch opened at 53:03 and the men were brought out of the diving tank as quickly as possible; the helmet, belt, and shoes were removed and then the men were placed in the recompression chamber. In the present case, the first man was in the chamber at 54:58 and the second at 55:15. The door of the recompression chamber was closed and the pressure was applied at 55:22. Thus the time from the opening of the diving tank hatch until both men were under pressure again was only 2 minutes 19 seconds. The pressure was raised to 50 feet in 1 minute 22 seconds, reaching there at 56:44. The time from leaving 50 feet in the water (the first stop in decompression for this depth and time of exposure) until the divers were at 50 feet in the recompression chamber was 4 minutes 46 seconds. They were then given the regular Diving Manual (*1*) decompression of 7 minutes at 50 feet, 10 minutes at 40 feet, 15 minutes at 30 feet, 30 minutes at 20 feet, and 30 minutes at 10 feet. This procedure was used for all dives at various depths and times of exposure.

Results.—At a depth of 100 feet 21 runs were made with exposure time starting with 9 minutes and increasing by 3-minute increments to a 30-minute exposure and then increasing by 5-minute increments to a 90-minute exposure with which exposure the series was ended. Six subjects made dives at each time of exposure, which makes a total of 126 man-dives at the 100-foot depth.

No *compressed-air illness* (commonly called caisson disease or "bends") was encountered except in one man who noticed a mild itch following the 27-, 35-, and 46-minute exposures, with a slight rash on the right arm following the last two exposures. This man was excluded in subsequent tests but it was not considered as of sufficient importance to terminate the series which was continued to an exposure of 90 minutes with no symptoms of compressed-air illness developing in any of the other divers.

At a depth of 150 feet 19 runs were made with an exposure time of 5 minutes to start and with increasing time increments of 3 minutes each run to a total exposure of 50 minutes. Nine subjects made dives at each time of exposure, making a total of 114 man-dives at the 150-foot depth.

Following the 25-minute exposure one subject had severe compressed-air illness and one had a mild attack. These men were not allowed to further participate in the series of tests. A third subject had a very mild attack following the 34-minute exposure, but the remaining six subjects continued to the 50-minute total exposure with no symptoms.

At a depth of 167 feet, 11 runs were made with an exposure time of 12 minutes for the first run with increasing increments of 3 minutes each run to a total exposure time of 42 minutes. Nine subjects made dives at each time of exposure except the last, making in all a total of 92 man-dives at the 167-foot depth.

Following the 30-minute exposure, one subject had compressed-air illness but this subject repeated the dive and continued the series to the end with no further symptoms. A serious case of compressed-air illness developed in one of the two subjects making the only dive of the 42-minute exposure time, thus necessitating the termination of the series.

SUMMARY

Surface decompression was tried out at 100 feet for exposures up to and including 90 minutes and no compressed-air illness was encountered.

Surface decompression was tried out at 150 feet for exposures up to and including 50 minutes. Three cases of compressed-air illness developed, two following the 25-minute exposure and one following the 34-minute exposure.

Surface decompression was also tried out at 167 feet for exposures up to and including 42 minutes. Two cases of compressed-air illness developed, one following the 30-minute exposure (subject repeated this run and continued to the end of the series), and one severe case following the only run at 42-minute exposure, thereby terminating the series.

It is planned to check these results further in actual diving operations on a salvage vessel.

REFERENCES

(1) Diving Manual, Navy Department, Bureau of Construction and Repair, section XII, 3670, 1924.

(2) Technical Bulletin, Navy Department, Bureau of Construction and Repair, Salvage Report of U. S. S. *S–4*, 1928.

(3) Hawkins, Shilling, and Hansen, Suggested Change in Calculating Decompression Tables for Divers, U. S. Naval Medical Bulletin, 33: 327–338; 1935.

PART II. SERVICE TESTS OF SURFACE DECOMPRESSION[1]

During the month of July 1935 additional dives were made under service conditions from the U. S. S. *Falcon*.

The method was the same as described in part I of this paper except that the men actually went down a descending line to the bottom in the open sea, and that the ascent was made by climbing up

[1] We wish to express our appreciation to Lt. R. E. Hawes, U. S. Navy and the officers and men of the U. S. S. *Falcon* for their cooperation and aid in accomplishing this work.

We also wish to thank Lt. J. A. Hollowell, U. S. Navy, officer-in charge, Experimental Diving Unit, who directed this work and assisted in the analysis of the data, and Commander F. S. Johnson, Medical Corps, U. S. Navy, for helpful suggestions.

the descending line onto the stage 30 feet from the bottom and then being brought to the surface at the rate of 50 feet per minute.

Results.—Fifty man-dives were made at depths rangings from 100 to 105 feet and for exposure times of from 50 to 92 minutes. One case of compressed-air illness of a very mild nature occurred following a 70-minute exposure at 102 feet and another case, also of a mild nature, occurred following a 91-minute exposure at 102 feet. Both of these cases occurred at the site of old injuries.

Thirty-eight man-dives were made at depths ranging from 148 to 156 feet and for exposure times of from 21 to 54 minutes. One case of compressed-air illness occurred following an exposure of 33 minutes at 156 feet. It was also a mild case.

Twenty-four man-dives were made at 162 feet and for exposures ranging from 20 to 46 minutes. No case of compressed-air illness was encountered in this series.

The decompression used for these dives was governed by the United States Navy Diving Manual (*1*) decompression table for each depth and time of exposure.

Table 1 gives the additional information concerning these dives.

TABLE 1

[Figures denoting time are averages for all dives at given depths]

Depth	Number man-dives	Descent time	Left bottom	Reached stage	Stage started up	Reached surface	Reached first stop in chamber
		Minutes	*Minutes*	*Minutes*	*Minutes*	*Minutes*	*Minutes*
100±.....	50	2:15	0:00	2:01	3:04	4:44	7:31
150±.....	38	2:29	0:00	4:12	6:06	8:53	12:32
162.....	24	2:43	0:00	2:51	5:19	7:54	10:50

Discussion.—Safe accomplishment of surface decompression depends upon the ability of the body fluids to hold nitrogen in supersaturation.

As an example of this supersaturation we calculated, by the method of Boycott, Damant, and Haldane (*2*), the tissues of a diver upon reaching the surface (ascent decompression, 50 feet per minute) following a 50-minute exposure at a depth of 150 feet (gage). This calculation shows the theoretical tissues to be saturated as follows: 5-minute tissues were saturated as if at a depth of 157 feet (absolute), 10-minute tissues at 167 feet, 20-minute tissues at 152 feet, 40-minute tissues at 119 feet, and the 75-minute tissues were saturated as if at a depth of 89 feet (absolute).

In computing the time of exposure it was found advisable in these tests to record from the time the diver left the surface until the stage was started up. This procedure was necessary because of the great variation in time required for the divers to get on the stage and unshackle it preparatory to ascent.

In calculating the various runs actually made, it became apparent that when the 20-minute tissues did not exceed a saturation of 145 feet (absolute) no compressed-air illness developed. It was then easy to calculate a theoretical maximum time limit of exposure for safe surface decompression at any depth. Table 2 gives these maximum time limits for certain depths corresponding to the United States Navy Diving Manual (*1*) decompression table. Of course, these theoretical figures must be subjected to experimental tests.

TABLE 2.—*Maximum time limit surface decompression*

Depth (feet gage)	108	132	150	156	168	180	192	204	225	250
Time (minutes)	90	54	45	42	35	28	24	23	20	17

Another factor of prime importance is the length of time that the body fluids can hold nitrogen in supersaturation before bubble formation commences. In our open sea confirmatory tests accomplished under service conditions the average time from the bottom to the first decompression stop in the chamber, for the 150-foot series, was 12 minutes 32 seconds. (See table 1.) For every dive this time must be kept as short as possible in order to minimize the occurrence of compressed-air illness. Therefore it is essential to have an adequate crew of well-trained men to handle the raising of the stage and the undressing of the divers.

A gas in supersaturation in a liquid may be suddenly released upon being jarred or agitated. Although this may not occur in the living body, it is advisable that the diver undergo as little exertion as possible during the ascent from the bottom to the recompression chamber. For this reason it is necessary to have the stage lowered to within at least 30 feet of the bottom so the diver will not have the exertion of climbing onto it near the surface where the supersaturation of his tissues is greater.

SUMMARY

In the diving tank series reported in part I, surface decompression was carried out for exposures up to and including 90 minutes at 100 feet (this and all subsequent depths in feet gage), 50 minutes at 150 feet, and 42 minutes at 167 feet.

In the service test series reported in part II, surface decompression was carried out for exposures up to and including 92 minutes at 100 to 105 feet, 53 minutes at 148 to 156 feet and 46 minutes at 162 feet.

It was shown from the tank tests and the service tests that surface decompression is safe for exposures up to and including 90 minutes at 100 feet.

Surface decompression can also be safely used for exposures up to and including 45 and 39 minutes at 150 and 162 feet respectively.

The theoretical safe length of exposure, calculated according to table 2, is 90 minutes at 100 feet, 45 minutes at 150 feet, 42 minutes at 156 feet and 39 minutes at 162 feet. Both tank and service tests sustain these theoretical figures.

It is necessary in the practice of surface decompression that (*a*) the time from leaving the surface until the stage starts up be considered the exposure time, and limited in accordance with table 2, (*b*) the stage be not more than 30 feet from the bottom, and (*c*) the stage be brought to the surface at a rate of 50 feet per minute and the diver be placed in the recompression chamber as quickly as possible with him undergoing a minimum of exertion.

Until further experimental tests are conducted with surface decompression, it is considered inadvisable to exceed either the depths or times of exposure as reported in this paper. Furthermore this work is based on results obtained in water of moderate temperature and whether the procedure is equally efficient for work in warm water still has to be determined.

BIBLIOGRAPHY

(1) Diving Manual, Navy Department, Bureau of Construction and Repair, section XII, 3670, 1924.

(2) Boycott, Damant, and Haldane, The Prevention of Compressed-Air Illness, J. Hyg. (Cambridge) 8:342–443; 1908.

DUODENAL ULCER [1]

By JAMES F. FINNEGAN, Lieutenant Commander, Medical Corps, United States Navy

In presenting this paper an endeavor shall be made to avoid the stereotyped and the obvious. In general it is culled from observation of stomach cases at the United States Naval Hospital, San Diego. We will, as far as our ability permits, give the reasoning for all which is stated as fact.

The predominance of this condition in males is accepted. The reason usually given is that males are in the great majority in the use of tobacco and alcoholic beverages. If this were at any time true, it surely has not been so for the last decade. Certainly not to the degree that the male sex predominates in the duodenal ulcer picture. There is an anatomical difference which favors the occurrence in the male; the duodenum ascends sharply in the first portion, then descends in the second portion to the bile ducts, whereas in the female the first portion is nearly transverse and has free access to the bile, therefore offering less opportunity to the effect of the acid

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stomach secretion. It is believed, however, that the main reason for this predominance in males is that they are to an overwhelming proportion more exposed to the stress and strain of life in general. It was noticeable that the great majority of ulcer cases occurred in unemployed veterans or in those in straitened financial conditions.

As to the question of racial predilections, it is noticeably absent in orientals and those of northern European stock. Here it would seem that diet possibly might be a factor. However, the staple of the oriental diet is carbohydrate which tends to produce a low hydrochloric acid stomach content, while the reverse is true of the northern European stock whose main article of diet is meat. The reason for this similar lack of incidence must be looked for elsewhere. These races have a marked similarity of existence, though on opposite sides of the world. Nature has not been lavish with either. The climate is harsh; the achievement of even the necessities is difficult, requiring much labor and facing of many natural disasters. Generations of such existence has bred in them a phlegmatic temperament, a stable nervous system and emotional fatalism. Adversities of life do not overwhelm them as readily as they do those to whom even a comfortable existence is readily obtainable. This would seem to be the reason for the striking absence of ulcer among these two such widely divergent races.

Age incidence: 30 to 40 years average, with a range of from 20 to 50. The reasons for its nonprevalence below the age of 20 are probably these facts: The ability to secrete a gastric juice of high hydrochloric content is not present in the gastric glands, and the nervous factor of stress and strain is absent. A diagnosis of duodenal ulcer after the fortieth year, unless the symptoms have extended over a long period of years, should be looked upon with suspicion, as in a large proportion of cases the so-called ulcer will be found to be carcinoma of the pylorus.

Diet has been indicted; poor habits of eating unsuitable food. It is felt that diet plays a very small, if any, part in the causation of ulcer, because duodenal ulcer occurs frequently in Navy personnel. No other group of individuals has its food so carefully selected, inspected and balanced as does the Navy. As we know from outbreaks of food poisoning, if food affects any of the personnel it will affect all the ship's complement. If diet was a factor of consequence in the production of ulcer we should have ulcer ships. The occurrence of ulcer in the Navy is a very potent argument against diet being a factor of any consequence.

Family predisposition; apparently there is no family tendency to ulcer; per se the heritage rather is that of an unstable nervous system which is characterized by the statement that the father or mother had all sorts of trouble with the stomach or bowels and that a brother or sister had the same symptoms.

The excretion of toxins through the liver, as in burn cases causes an acute ulcer. Trophic disturbances such as vasoconstriction, edema of mucosa with cutting off of the circulation, herpetiform lesions, endarteritis, focal infections producing bacterial emboli, lack of an antiferment preventing autodigestion of the mucosa have been mentioned as playing a part in the etiology.

In all cases of ulcer there exists a striking concomitant of nervous disturbances, ranging from a mild psychoneurosis to well-defined mental aberrations. Seeking to explain this finding, it was recalled that in periods preceding physical combat in athletics and in positions of danger or stress and strain, certain gastric and general reactions were noted. These are a feeling of goneness in the epigastrium, abdominal tightness, symptoms of hyperacidity, and intestinal hypermotility This is believed to be an acute situation neurosis. A conception which seems to fit the symptoms of duodenal ulcer and explain their origin is offered; that is a chronic situation neurosis resulting in dysfunction of the stomach and intestines. This is not conceived as a fear mechanism, but rather a fear defense mechanism characterized by a hypersecretion of pressor substances in the blood, a hyperstimulation of the organism as a whole to carry on increased burdens which are expected. Hypersecretion of hydrochloric acid for its intestinal antiseptic value and inherent need for food for bodily repair or possibly because of the inherited custom of feasting following victory. The excessive pressor substances are reduced in a physiological manner by strain of physical combat or by physical action in situations of danger or stress and strain. In the chronic form there exists an excess of pressor substances continuously with concomitants of hypersecretion and hypermotility of the intestinal tract. The pylorus is in a continuous state of excess tone. There is no rhythmical relaxation to allow neutralization of the pyloric end of the stomach by the alkaline duodenal secretion. The strongly acid stomach contents further tend to pyloric spasticity. The pylorus opens because of fatigue, and a gush of highly acid content is discharged against the duodenal mucosa. The continuous insult to the mucous membrane of the duodenum which is not suited to an acid environment, results in its destruction and ulcer formation.

Symptomatology.—The earliest manifestations are best described as a very indefinite picture of gastric disturbance such as distention with a vaguely indicated discomfort, burning or occasional dull pain. These symptoms cannot be localized to a very definite area of the abdomen, usually the whole epigastrium is indicated. There is only one outstanding feature of the early symptomatology, and that is periodicity.

Gradually this indefinite picture gives way to a definite clinical sequence which is characterized by the irregularity of its occurrence,

the chronicity of its course, localization to the region about the umbilicus, and regularity of one symptom, hunger pain.

The hunger pain is probably due to one or a combination of several factors, hyperchlorhydria, pylorospasm, or the gush of the strongly acid content against the duodenal mucosa. Its earliest manifestation is of a burning, acid sensation, followed by a soreness of the entire epigastric region. In the fully developed syndrome the pain is sharp, gnawing in character, regular in its relation to meals, 1 to 1½ hours after eating. However, variations of from 30 minutes to 4 hours after meals occur, due to variations in motility, ferment activity, stimulative or neutralization power of food, and various types of medication. A striking feature of the pain is the regularity with which it awakens patients from sleep at about 2 o'clock in the morning. Here again we find variations dependent in the main upon the hour of the evening meal. The location of the pain is usually low down in the epigastrium, in the region of the umbilicus. It is generally not of the finger type, that is, the patient can rarely localize the maximum intensity with the tip of one finger. While the localization of the pain is quite constant, the radiation is variable. The majority of cases describe the radiation downward toward the left iliac fossa. The next most frequent is downward and to the right iliac fossa; seldom to the gall bladder region, rarely upward into the chest behind the sternum. A companion pain in the right lumbar region posteriorly and right lower chest about the junction of the tenth or eleventh rib with the spine is of relative frequency. The pain is relieved almost instantly by food, milk or alkalies, in fact almost anything that is put into the stomach, not of acid character.

Another type of painful disturbance found in many ulcer cases is a continuous soreness across the lower abdomen, at times cramping in character.

A rare painful disturbance which usually accompanies an ulcer of penetrating character is so severe in its intensity that it doubles the patient up.

Nausea is present in almost every case at some period in the course of the disease. Vomiting is not a constant finding and is usually of the voluntarily induced type, that is, the patient comes to the knowledge that food causes pain and he seeks to relieve his symptoms by emptying the stomach. Vomiting is caused by reflex stimulation of the center in the medulla, from local irritation of the extremely high hydrochloric acid content of the stomach. When vomiting is a prominent symptom it usually denotes pyloric obstruction. In some few cases vomiting extends over a long period without any obstruction to the pylorus. These are believed to be due to an acidosis of starvation origin.

Vomiting of blood appears to be the initial symptom of ulcer in many cases. However, a careful questioning will bring out the fact that there have been some suggestive stomach disturbances, but not of sufficient severity to impress the patient, which have been lost sight of in the hemorrhage crisis.

The blood of the emesis is usually dark with many clots due to regurgitation into the stomach. If the hemorrhage is long continued the pylorus becomes patulous and bright bleeding does occur.

The usual hemorrhage picture is that the individual suddenly feels weak and faint; slight nausea coupled with a desire to move the bowels causes them to seek a toilet. This is due to the fact that the hemorrhage has occurred sometime before this. The symptomatology is induced by the anemia and the stimulus of the distention of the bowel by blood and clots. The nausea becomes aggravated and a large amount of black liquid vomitus mixed with clots is ejected. Frequently the patient loses consciousness and is found lying on the floor surrounded by a pool of dark vomitus. This episode is usually followed by several bowel movements of tarry character.

The appetite is generally very good; in cases when it is not the reason is given as being afraid to eat, knowing that food will bring on pain.

Nutrition is generally very well preserved except in cases of pyloric obstruction, where losses of up to 50 pounds are common.

Constipation is the rule, due to the combination of overdigestion of food from excess hydrochloric acid and enterospasm of neurogenic origin. Other evidences of this intestinal dysfunction are flatulence, borborygmi, colicky pains in lower abdomen.

Heartburn is a very common symptom caused by regurgitation of the highly acid stomach contents through the cardia.

Anemia of mild secondary type is present in nearly every case. Frequently paradoxical blood counts are seen in cases where there has been a recent blood loss by bowel, not severe enough for the patient to have had the clinical picture of hemorrhage. Such count occasionally runs up as high as 5,700,000 R. B. C. with an average of 5,500,000. The hemoglobin, however, is low, averaging from 60 to 70 percent.

Melena is of frequent occurrence. All cases show occult blood on a meat-free regimen.

Neurasthenia is present in nearly all cases, headaches for which no cause can be found, vasomotor disturbances, coldness and numbness of extremities, dizziness and visual disturbances, cardiac irregularities, usually tachycardia, pain in region of the heart, and a sense of oppression in the chest. Blood pressure is low in many cases, but the pulse pressure is within normal limits.

Gastric content.—A total hyperacidity and hypersecretion is a constant finding.

Perforation which is fortunately not a common complication, occurs usually after some physical effort such as shoveling, lifting, any exertion which increases intraabdominal pressure. The onset is ushered in by a sudden sharp pain as though the patient had been stabbed in the right upper abdomen, a consciousness that something very serious has happened, that something has let go inside the abdomen. The patient doubles up, with his hands pressed against the epigastrium, and usually lies on the floor. If he attempts walking he is unable to stand erect. His face shows marked pallor, with beads of sweat on his forehead. His expression is anxious, general signs of shock are present. There is retching if the stomach is empty; vomiting if the stomach contains food. The pulse is rapid and hard. The abdomen presents boardlike rigidity of the right upper rectus, later generalized. Once this has been felt by the palpating hand it is unforgettable. The abdomen feels like a table or desk top.

Variations from the usual are seen occasionally as a typical history, with the pain localized in the right iliac fossa but with rigidity of the upper right rectus and tenderness only in the right iliac fossa. Typical history and physical signs but not much increase in pulse rate and no signs of shock occurring in cases where the duodenum is high placed and closely approximated to the liver. Perforation occurring when the patient was absolutely at rest on the fourth day of an ulcer dietetic regimen. Subacute cases; pain not very severe but enough to cause the patient to seek hospitalization; signs of sepsis, temperature, chills, increased white and poly counts, abscess of lesser peritoneal sac. Cases with signs and symptoms. X-ray findings of subdiaphragmatic abscess. Rarely cases simulating an appendiceal abscess. Where there is any doubt as to the diagnosis an X-ray with the patient in the upright position will reveal an air bubble over the liver between this organ and the diaphragm.

The X-ray findings in a positive ulcer case need not be repeated, but it is well to recall that X-ray is one of the most helpful aids to diagnosis, but that the X-ray machine is also not a clinician and the findings reported from the X-ray should be interpreted in the light of the history, physical examinations, laboratory findings, and reaction to treatment. It also must be borne in mind that it is possible to have negative X-ray reports in known cases of ulcer.

The physical examination usually reveals a fairly well nourished male of mid-European stock, a high strung type. His attitude is apprehensive, heart is overacting, pulse is quickened. Sweat runs down the side of his chest. The abdomen generally shows the scar of an appendectomy performed for chronic appendicitis. Finger point tenderness is found within a radius of a 2-inch circle about the umbilicus. In the great majority it is 1 inch to the left of the umbilicus, occasionally above and to the right. Infrequently to the right

and below; seldom between the umbilicus and xiphoid. In many cases a palpable, tender, spastic descending and iliac colon is noted. Proctoscopy usually reveals a markedly spastic sigmoid.

Differential diagnosis.—Dysfunctions of no organic basis are characterized by lack of pain; variability of symptons and no regular relation to meals, occur generally in the mornings and are traceable to dietary indiscretions, poor habits of eating, greasy and poor cooking. These do not respond well to an ulcer regimen. Laboratory and X-ray findings are negative.

Visceroptosis.—The asthenic habitus with variability and multiplicity of symptoms. Obstinate constipation. Relief to a certain degree by small multiple meals and posture. Typical X-ray findings. Gall bladder disease.

Type of individual fat and phlegmatic. History of typhoid, pneumonia, cholangitis. Definite finger tip localization of pain to region of tip of ninth rib. Splinting of muscles and tenderness on pressure is localized to gall bladder region. Ulcer diets which are of high fat content markedly aggravate symptoms. Gall bladder visualization is a most helpful adjunct to diagnosis. Here again it is well to remember that the findings should be interpreted as part of the clinical sequence.

In early cases in youthful individuals the differential diagnosis clinically is often difficult. The gall bladder visualization and G. I. series usually give the diagnosis. In one case, however, the patient went to operation with a diagnosis of gall bladder disease and operation revealed a duodenal ulcer.

Differentiation from gallstone colic usually presents little difficulty. The pain of gallstones is colicky, radiates downward toward the umbilicus and upward toward the right scapula. The maximum intensity is localized in the gall bladder region. Shock is not as marked.

Renal colic; pain located in lumbar region radiating downward into penis testicle or right thigh.

Appendicitis acute; history and typical physical findings blood count differentiate. The so-called "chronic appendicitis" or subacute appendicitis may produce many of the symptoms of ulcer reflexly. The history may be of help. X-ray, however, is of the utmost importance in this situation. If the findings are not conclusive, exploratory laparotomy is justifiable.

Cirrhosis of liver presents enlargement of that organ, the Van den Berg being of the greatest diagnostic help.

Carcinoma of pylorus; age principally. Many early carcinomatousinvolvements of the pylorus will be found operable if all ulcer cases over 40 who do not respond readily and rapidly to dietetic

treatment are subjected to operation. It is felt that it is wiser to err on the side of radicalism in these cases than to be ultraconservative.

Ulcers of the intestine, amoebic, are revealed by stool examinations and proctoscopy. Ulcerative colitis, idiopathic or Bargens proctoscopy and marked bloody diarrhea, T. B. C., ulcerations, X-ray, proctoscopy, focus in lungs. Gastric crisis. Distribution of pain, girdle type. Argyll Robertson pupils, absent knee jerks, history of syphilis, blood and spinal fluid.

Arterial hypertension, with intestinal hemorrhage and gastric symptoms. Proctoscopy, X-ray if possible. However, any case of intestinal or gastric hemorrhage with blood pressure over 200 with concomitant urinary findings is probably bleeding from a rupture of an intestinal vessel.

Chronic gastritis presents no difficulty. The morning vomiting of ropy mucus, low hydrochloric acid content.

Preventative treatment.—Avoid oversalting of food, moderation in smoking. Endeavor to reduce gastric effects of neurosis by psychotherapy and rest.

General medicinal and dietetic treatments directed toward the following ends. To reduce free hydrochloric acid, to remove irritation, to correct undernourishment and anemia.

Specific procedures directed toward preparation for surgery. Postoperative treatment, to control vomiting, to stop hemorrhage.

The reduction of hydrochloric acid is accomplished by first reducing the psychological stimulus to a minimum. The method used is a diet which is not appealing to the special senses yet has a high caloric value. The main dietary component, the egg-cream-milk mixture, is not flavored. The ground meat is used raw at first, later but lightly seared. Rice is used plain boiled with unsalted butter. The salt-free factor renders the diet unpalatable and strikes at the source of the hydrochloric acid in the body.

Upon admission the patient is given a cleansing enema and made an absolute bed patient; later given head priveleges when deemed advisable. This continues for 18 days.

The diet which is used is termed a Lenhartz for convenience only as it is a modification of that diet as a basis.

LENHARTZ DIET

First to ninth days, 7 a. m. to 7 p. m.: Basic mixt. ½ oz. every hour.
Second day: Basic mixt. 1 oz. every hour.
Third day: Basic mixt. 1½ oz. every hour. Powder grs. XVQ ½ hour.
Fourth day: Basic mixt. 2 oz. every hour.
Fifth day: Basic mixt. 2½ oz. every hour.
Sixth day: Basic mixt. 3 oz. every hour.
Seventh day: Basic mixt. 3½ oz. every hour.
Eighth day: Basic mixt. 4 oz. every hour.

Ninth, tenth, eleventh, and twelfth days: Basic mixt. 4 oz. every quarter hour; 11 a. m. and 4 p. m. scraped beef, 35 grams.

Thirteenth, fourteenth, fifteenth days: Basic mixt. 4 oz. every quarter hour; 7 a. m. 1 soft egg, 2 tablespoons cereal and butter, cream and sugar; 11 a. m. 2 tablespoons rice, scraped beef, 35 grams; 7 p. m. 1 soft egg, 2 tablespoons cereal, cream and sugar.

Sixteenth, seventeenth, eighteenth, nineteenth, twentieth, and twenty-first days: 7 a. m. 2 soft eggs, 2 oz. strained oatmeal, 2 slices buttered toast; 9 a. m. 4 oz. basic mixt.; 11 a. m. ice cream or jello, minced chicken or beef, 4 oz. eggnog; 12 noon 4 oz. basic mixt.; 1 p. m. 4 oz. eggnog; 3 p. m. 4 oz. basic mixt.; 4 p. m. 2 soft eggs, 2 slices toast, 2 oz. strained cereal; 5 p. m. 4 oz. basic mixt.; 7 p. m. 4 oz. eggnog. Powders grains XXX, 1½ hours after each meal.

Formula for basic mixture.—1 egg, 1½ oz. cream, 4 oz. milk.

In the observance of ulcer cases over a period of years several facts were noted. The progressive increase in the amount of soda bicarbonate (the usual popular antacid) needed to relieve symptoms, indeed to a point where the daily amount used was expressed in terms of ounces. That the symptoms for control of which it was used did not proportionately increase in severity. This was interpreted to mean that the bicarbonate gave rise to a vicious cycle. This agent which was so effective in the early stages in combating the symptoms due to hyperchlorhydria, served to aggravate them as time went on. The conclusion was that carbonates should be removed from any regimen directed toward the medical cure of ulcer.

This was proved a move in the right direction by the findings in two achlorhydria cases which did not show any free or combined hydrochloric acid in the gastric contents after Histamine. These patients were placed on regular diet, given no medication except soda bicarbonate, this was given until their urine remained alkaline to litmus for a week. The gastric contents were then removed and revealed a free HCl of 16° and combined of 24° in one case, a free HCl of 18° and combined of 32° in the other. This experiment was repeated combining sodium chloride with the soda bicarbonate and higher percentages were obtained.

As a reduction of free HCl was believed to be the paramount factor in the treatment of ulcer, an alkaline powder which would do this and not be open to the objections that accompany the use of carbonates, was sought. Such a powder must have the following properties. It must not enter into the body chemistry, have two components, one rapidly ionizable; the other a complex radicle which disassociates slowly. The first to neutralize the immediate hydrochloric acid secretion, the latter to counteract the secondary rise.

In addition to the powder another method was used to effect HCl reduction, that of removing the main source of supply from the diet, sodium chloride. While it may be true that as long as HCl is present, enough chloride is absorbed from the intestine to continue its secretion and is the objection advanced to negative the effect of a salt-

free diet. This resorption of chloride cannot occur if the HCl is bound into an unabsorbable combination. It does not occur with the salt-free diet and an alkaline powder which can be recovered quantitatively in the feces.

That this chloride retention does not occur in patients treated with the method of treatment described in this paper is evidenced by the fact that any free HCl in the gastric contents of a patient after being on the ulcer regimen over a period of 10 days is an extreme rarity, the combined acidity is proportionately reduced also. It is also worthy of note that patients who have been on this diet and have faithfully carried it out desire salty and acid substances such as pickles and vinegar.

The alkaline powder has the following composition and method of administration. Magnesium oxide 5 parts, bismuth subnitrate 2 parts, calcium tribasic phosphate 7.5 parts, magnesium tribasic phosphate 7.5 parts. Approximately 1 gram of this powder is given every half hour between feeding of basic mixture, 2 grams at bedtime, until the sixteenth day, then it is given in 2-gram doses 1½ hours after meals and at bedtime.

The rationale of the powder is as follows: Bismuth protective; magnesium oxide rapidly dissociable for the primary hydrochloric secretion. The tribasic phosphate dissociates slowly and controls the secondary rise. As the calcium salt is constipating but not enough to balance the laxative action of the magnesiums, they are given in equal proportion. This serves in most cases to produce normal daily bowel movements. Occasionally the proportions of the two must be varied because of the preponderance of laxative effect.

No cathartics are allowed in the first 3 weeks. If there is need for measures other than the powder, soapsuds enemas are used. If gas in the large bowel which is manifested in most cases to some degree, due to lack of the antiseptic action of the hydrochloric acid, becomes excessive, turpentine enemas are indicated and are successful. As the patient becomes an up-patient, spirits of turpentine on sugar proves effective.

Removal of irritation is produced by absolute bed rest which reduces nervous and physical activity to a minimum. Bland diet which removes local irritation. Barbital and bromides where indicated. Psychological treatment directed toward removal of sources of mental stress and strain.

Correction of anemia by use of 30 grains of Blauds pills, pulverized and added to basic mixture daily.

Preparation for surgery when operation has definitely been decided upon. As a routine patients were placed on forced fluids, glucose 5 percent, soda bicarbonate 5 percent, Murphy drip 3 hours on and 3 hours off for 9 hours daily. Where nutrition was markedly impaired

the basic mixture was forced to limit of tolerance supplemented by glucose intravenously.

Vomiting of the type that causes a patient to seek admission to the hospital is conceived as of starvation acidosis origin and readily responds to Murphy drip of glucose and soda bicarbonate. Five percent Murphy drip, 40 drops to the minute continuously with nothing by mouth for 12 hours then albumin water ad libitum only for 48 hours.

Treatment of hemorrhage.—Absolute rest, morphine in sufficiently large doses as to produce maximum results. Continuous ice bag to the abdomen. Take blood pressure immediately, to rule out circulatory cause. Continue blood pressure readings once every hour, as blood pressure below 100 systolic is of favorable portent as to the probability of coagulation in the bleeding vessel. A rise of blood pressure to normal after the first 12 to 24 hours indicates that the blood vessel has thrombosed and further hemorrhage will not be expected. Red blood cell count, hemoglobin estimation; bleeding time; smear for abnormal cells is done immediately to rule out blood abnormalities. Nothing by mouth for 24 hours. Murphy drip of glucose and soda bicarbonate 5 percent continuously. Ice may be given to suck to prevent drying of mouth. Chewing gum is also useful, if patient is not too weak. After 24 hours ice cold albumin water in dram doses every 30 minutes. After 48 hours Lenhartz basic diet mixture ice cold, half ounce doses every half hour. The alkaline powders are added after 72 hours when the mixture is increased and placed on an hourly basis. Pulverized Blauds pills 15 grains XV are added to the mixture t. i. d. Thrombo plastin has been used ice cold by mouth with some success in continued bleeding cases.

A reticulocyte count is made about the third or fourth day to determine the response of the bone marrow. Liver extract has been found of no value. If the blood pressure is well above 100 systolic upon admission and active bleeding continues, nitroglycerine is helpful. Transfusion or infusion has not been done on any case, as it is believed that in the presence of active bleeding these measures will simply raise the blood pressure and volume, thereby continuing the hemorrhage. These cases will developd an effective thrombosis in the vessel involved when the local arterial flow is lessened sufficiently by the diminished blood pressure. The rare cases which continue to bleed after the systolic pressure is below 100 are probably cases where there is a localized arteriosclerosis of the involved vessel. The liquid diet is continued for 6 weeks. A very unusual fact has been noticed, that the X-ray usually shows a patulous pylorus without any evidence of ulcer in the duodenum at the end of this period of treatment. The significance of this, however, cannot fully be appraised until the patients have been observed over a long period of

time. Three cases have been symptomless and in a state of well being for a year past hemorrhage.

In the management of postoperative cases they were first informed by diagram and instruction that the digestive powers of the stomach were reduced and to chew the food more thoroughly, than they were accustomed to do. Keep on a low sodium chloride intake. Use the alkaline powder. That the ulcer was not cured and a continuance of the diet and powder was necessary for at least a year. That thorough mastication must continue for many years, as that part of the intestine into which their food was now going was not naturally fitted to accept large masses of food. All of these cases promptly experienced relief of symptoms, gained weight, and left the hospital markedly relieved.

In all ulcer cases before leaving the hospital the fact that the ulcer is not cured is impressed upon them and that their future lies in their own hands, a studious observation and attention to the past hospital routine must be followed for a year to a year and a half before medical cure can be hoped for.

In service men unless nearing the 16 or 20 year Fleet Reserve transfer period, survey from the service is indicated. It is wise to adopt this attitude as a general procedure, due to the fact that nearly 100 percent of ulcer cases sent to duty return to the hospital, as it is impossible to obtain proper diets, spend much time on the sick list and have to be surveyed eventually.

The hospital routine is as follows: (1) Do not use alcohol, tea, or coffee. (2) Reduce or abolish use of tobacco. (3) Do not add salt to food; avoid other strong seasoning. (4) Do not eat when tired or when emotionally upset. (5) Chew food thoroughly. (6) Use plenty of water, 8 to 10 glasses daily. (7) Milk may be used as much as desired. (8) Keep bowels free. If cathartic necessary use mineral oil, petrolagar, soap suds enema. (9) Continue use of stomach powder. (10) Do not use extremely hot or cold beverages. (11) Avoid all coarse foods, meats, or vegetables. The safest procedure is to advise that all red meats be run through a food chopper before cooking. Vegetables to be thoroughly mashed and coarse fibers removed. All acid fruits and fruits with skins and seeds such as grapes must not be used. Return to the hospital if any ulcer symptoms recur and persist under this regimen.

These methods have been in use at this hospital for a period of about 2 years with good clinical results.

ERRORS IN SIX CONSECUTIVE CASES OF "APPENDICITIS"[1]

By W. H. MICHAEL, Commander, Medical Corps, United States Navy

An interesting and instructive collection of material has been discovered in the study of six patients consecutively admitted with the diagnosis of appendicitis acute. Though it is the established rule at this hospital to operate at once all acute appendicitis cases and all probable appendicitis cases (unless serious counterindication exists) the first four cases were not operated and recovered from four widely different maladies.

The error in the fifth case was not in diagnosis, but in preoperative treatment. Everything went astray in the sixth case and the writer removed an almost normal appendix. From a retrospective point of view this case received both the wrong diagnosis and incorrect treatment.

The high points of the six case histories follow, and, though it is freely admitted that criticism is easy but art is difficult, an attempt will be made to discover the reason for each mistake.

Case 1.—White male about 23 years old admitted to the hospital on September 4 with the diagnosis of appendicitis acute, undetermined. Onset occurred that morning with a dull generalized pain in the abdomen which became worse following breakfast, with a tendency to radiate to the right lumbar region. Patient vomited several times without relief and an enema was given without decreasing the pain. On admission the pain was most intense in the right hypochondriac region. There was slight nausea. Patient admitted that urination was painful and frequent, and that there had been a persistent morning tear since his gonorrhea 5 years before. Physical examination revealed: A general abdominal tenderness which was most marked in the upper right quadrant, a thin muco-purulent discharge from the urethra, and a firm, irregular, moderately tender prostate, a smear from which was positive for gonococci.

Symptoms subsided though there was a slight recurrence of the abdominal pain on September 29 (25 days after). An X-ray taken on October 1 showed a distinct shadow at the level of the uretero-cystic junction on the right side. However, this small stone was not discovered in the X-ray until after it had actually been passed on October 29, 28 days after the X-ray. The patient went to duty well on November 19.

The mistake here lay in not taking a more careful history and coupling that history with the fact that the physical findings were distinctly referable to the urinary tract. However, it is to be noted that the diagnosis was given as undetermined, indicating that some doubt existed in the mind of the physician making it. And he was right, when confronted by two diagnoses, to choose the more urgent; for by so choosing, the case was certain to receive immediate study rather than morphine.

Case 2.—A young white male was admitted with the diagnosis of appendicitis acute, on September 6, 1935. The same morning about 9 a. m., after the patient

[1] Read before a meeting of the Escambia County Medical Society, at the United States Naval Hospital, Pensacola, Fla., Dec. 10, 1935.

had finished drinking a bottle of beer, he was suddenly seized with abdominal cramplike pains which were most severe in the epigastrium. Nausea but no vomiting followed. His bowels had moved the day before. There was no urinary disturbance. On admission there was a constant nauseating pain in the epigastrium with a tendency to radiate to the right hypochondrium and the right lower quadrant. The pulse was 106. Temperature and respiration were normal. Routine blood and urine examinations showed nothing of interest.

Physical examination was negative except as follows: Tenderness in the epigastrium and the right lower quadrant, injected sclera, and, what proved to be most important of all, a marked odor of alcohol on the breath. After this discovery the patient admitted that he had done considerable drinking the night before. Bogen's test for alcohol in the urine showed 2.5 mg or more per cubic centimeter. The diagnosis was changed to alcoholism acute, and recovery in 24 hours on appropriate treatment followed.

Here the error is more obvious than in the first case. Patients with acute appendicitis do not drink beer for their breakfasts, nor do they have such a sudden onset of symptoms accompanied by the signs of alcoholism.

Case 3.—A young white male was admitted to the hospital on September 7 with the diagnosis of appendicitis acute. The patient woke at 5 a. m. that day with cramplike abdominal pain which was most severe in the epigastrium and left hypochondrium, but radiated to the entire abdomen. Bowels had moved regularly, no nausea vomiting, nor urinary disturbance. Pain rapidly became more severe making breathing painful. The pain radiated to the left shoulder. On admission the patient was in moderate shock, temperature subnormal, skin pale and cold, mucous membranes cyanotic, pupils equal, but reacted to light and distance. Respiratory movements had greatly diminished. Abdomen was painful and hard, almost to boardlike rigidity. Dullness was present in the left flank, which dullness turned attention again to the history. Patient admitted that he was struck in the left chest by a pitched baseball 6 days before admission, but had thought nothing of it. Urine showed a trace of albumin but no blood. X-ray was negative, showing no pneumoperitoneum, thus, it was hoped, excluding ruptured ulcer. The white blood count was 25,000 with 90 percent polymorphonuclear leukocytes. The diagnosis of ruptured spleen was made and treatment for shock instituted. The patient recovered under expectant treatment. His red blood count decreased from 4.2 million on admission, to 2.9 million a week after, and then rapidly returned to 4.3 million on September 25. He went to duty, apparently well, on September 27.

Abdominal pain seems the one point in this case on which to base the diagnosis of appendicitis. Failure to elicit the important history of trauma led the doctor astray. The pain and symptoms were more intense in the northeast abdomen, and the condition of shock pointed to hemorrhage or perforation rather than appendicitis. Nevertheless, right or wrong diagnosis, the doctor did the really essential thing—sent the man to the hospital without delay.

Case 4.—A young white man of the C. C. C. was admitted September 9, 1935, with the diagnosis of appendicitis acute. Onset occurred 2 days before with moderate cramplike pain in the abdomen. No nausea, vomiting, nor fever. Pain subsided the next day, but returned again after supper when the patient vomited. He gave a history of tape worm accompanied by similar symptoms

in 1929. The physical examination revealed only slight tenderness in the epigastrium. Routine laboratory findings were normal, as were the temperature, pulse, and respiration. All pain subsided following a high enema which returned considerable hard dry fecal matter. Stools were positive for the ova of hookworm. The diagnosis was changed to hookworm disease and constipation was believed to have aggravated the symptoms of that disease. The patient went to duty on September 17.

Indefinite abdominal symptoms are common in hookworm disease, and, since approximately 80 percent of the members of the C. C. C. organization of this region have hookworms, the diagnosis of appendicitis acute in the presence of such mild symptoms seems unjustified.

Case 5.—The patient, a young white man, was admitted on September 16, 1935, with the diagnosis of acute appendicitis. Symptoms appeared at 3 a. m. the same day when the patient was awakened by a severe generalized abdominal pain. He reported to the camp infirmary where he was given a dose of salts which failed to relieve the pain. Toward noon the pain began to localize in the lower right quadrant and the patient became nauseated and vomited without relief. The one physical finding of interest was localized tenderness in the right lower quadrant. An appendectomy was done without awaiting the results of laboratory work. A necrotic appendix covered with diptheritic membrane was removed. There was considerable cloudy fluid in the abdomen. The case recovered without incident or drainage.

The case is included because it exemplifies the worst of this series of errors: The administration of a purge to a case presenting acute abdominal symptoms. Though the case was sent in with the proper diagnosis, this error in treatment was inexcusable and outweighs in potentially bad results any of the other mistakes.

Case 6.—A young man was admitted on the 19th of September. A dull pain appeared at about 9 a. m. the previous day. The pain began in the lower right quadrant radiating toward the umbilicus. The morning of admission the pain became more localized in the lower right quadrant and the patient vomited at 3 p. m. Patient's bowels moved several times in the afternoon. The last movement, about 6 p. m., consisted principally of mucus streaked with blood. The physical examination revealed a slight tenderness in the lower left quadrant of the abdomen and muscle spasm with marked tenderness in the lower right quadrant. Routine laboratory findings shed no light on the diagnosis. Though it was realized that this was not a clear case, it came well within the realm of probability, and operation was decided upon. An appendectomy was done under spinal anesthesia and an apparently normal appendix removed. The next day hookworm ovae were found in the stool. The case, in retrospective, was considered as an acute, though mild, entero-colitis plus hookworm disease. The patient made an uneventful recovery.

In review: The first case proved to be a stone in the right ureter; the second, acute alcoholism; the third, a ruptured spleen; the fourth, hookworm disease and constipation; the fifth was correctly diagnosed after the unforgivable administration of a purgative; and the sixth, which was operated upon, was probably mild entero-colitis plus hookworm disease.

Tice's Practice of Medicine states that the following abdominal conditions must be kept in mind in making the diagnosis of appendicitis acute in males: Dietl's crises, stone in the right kidney or ureter, the abdominal crises of locomotor ataxia, acute obstruction of the bowels, gallstones, typhoid fever, acute hemorrhagic pancreatitis, Meckel's diverticulum, diaphragmatic pleurisy, and Henoch's purpura. Dean Lewis' Practice of Surgery makes these additions to the above list: Pinworms in children, spinal arthritis, herpes zoster, right-sided epididymitis, pneumonia, pyelitis, tuberculosis, and pneumococcus peritonitis, acute enteritis in children, and gastric or duodenal ulcer.

It is interesting that of the five cases of this series sent to the hospital by well-trained physicians with the incorrect diagnosis of such a common condition as acute appendicitis, only one—the stone in the ureter—falls directly under any of this long textbook list of conditions.

But the potentially tragic crime in this series of mistakes was that of giving a purge to the fifth case. The physician in that case may be proud of making the right diagnosis, but this error in treatment which occurred under him is worse than all the others taken together. For though the others were all improperly diagnosed, they all, including the case which was operated, received safe treatment. It is believed that it was due to this safe, if not correct, treatment that this series proved itself to be an unmarred comedy of errors.

INFLUENZA LYMPHATICA—THE PENTATYPE OF INFLUENZA[1]

By Roger A. Nolan, Commander, Medical Corps, United States Navy

In the year 426 B. C., Hippocrates for the first time described the gross picture of the disease now known to us as influenza. Centuries later Pfeiffer tells us of the bacillus which bears his name and thought by him and others to be the etiological factor of influenza. He further writes and describes a recognized entity which he calls glandular fever. This we are to speak of later. And now the remarkable Osler, whose classical subdivision of influenza made its vast complex symptomatology most understandable. He accomplished this by taking four of the major systems of the body and fitting them separately into the gross symptomatic and clinical picture, resulting in four types—no. 1, the respiratory type, affecting the respiratory system; no. 2, the gastro-intestinal type, affecting the gastro-intestinal system; no. 3, the rheumatoid type, affecting the voluntary muscular system, and type no. 4, affecting the nervous system and then he stopped—yet he too writes of a glandular fever, describing it in his masterly but brief manner, and now because of my observations, data, etc., to follow, I herein add to Osler's classification of types of influenza and introduce to you the belated pentad, namely, influenza lymphatica, affecting primarily the lymphoid system and tissues of the body.

[1] Named by the author, in a paper read at the sixth annual meeting of the American Public Health Association held at Helena, Mont.

Report of an epidemic of glandular fever, so-called because of the glandular manifestations; infectious mononucleosis, so-called because of the blood picture; monocytic angina, so called because of the blood picture and pain in the neck. Place, Coronado, Calif.; population, 8,700; time, beginning November 1934 and lasting until June 1935. Number of cases over 2,000.

This paper consists of two reports—one written on December 30, 1934, 6 weeks after the first cases were noted and the second report herein submitted. Early recognition of this disease and my reason for anticipating an epidemic was based on my experience while stationed at the United States naval powder factory at Indian Head, Md., some 7 years ago, 1928.

In November of that year the U. S. S. *Nitro* called to take on 1 million pounds of powder for distribution to ships of the fleet. Her last port of call had been Panama. On her departure it was noticed that a mild influenza, respiratory in type, appeared amongst the 700 civilian employees. A little later in the public school, whose registration was about 900 pupils, symptoms of so called glandular fever appeared. As I recollect, I saw and diagnosed about 250 cases of this school's enrollment. I called this matter to the attention of the State public health authorities, who sent two investigators and they concurred with my findings. We three agreed that we had never observed a similar clinical picture in children before. At this time my diagnosis was made on the information obtained from half of a printed page in Osler, in which he described this condition now known as glandular fever.

Note of facts—no. 1, arrival of the ship from Panama, and the contact of its crew with mass population ashore had resulted in the appearance of a mild influenza epidemic, respiratory in type, within 10 days after departure of the ship; no. 2, appearance of so-called glandular fever in juvenile population.

DECEMBER 30, 1934—FIRST REPORT

1. The early cases were first noted in Coronado 10 days after the arrival of the fleet from the east coast on November 9, 1934, in the homes of officers and men who were given leave. Many were suffering from head colds, etc., and 500 cases of mild influenza were noted in the fleet on their trip from Panama to the Coronado-San Diego area.

2. The symptom complex noted in the present epidemic is distinctive. In the cases of children from 6 months to 5 years of age, the onset was noted by the parent; in those between the ages of 5 to 13 years the onset was noted by the parent or school teacher, and was aptly termed "a change in the personal characteristics." Among the younger children some were cranky, others fretful—not wanting to

play and preferring to lie about; this sign in most cases was constant. The changed mannerisms lasted from 3 to 5 days and then a series of symptoms developed which were of such moment that a doctor was called to the home.

3. The symptomatology varied but little in the 220 cases:

(*a*) Fever was constant symptom 100 percent, ranging from 100° to 104°.

(*b*) Pain in back of neck, 80 percent.

(*c*) Vomiting, 10 percent.

(*d*) Abdominal discomfort, 20 percent.

(*e*) Blood counts, total, red, white, and differential were made on over 50 cases. The persistent picture was this:

(1) Red counts normal or nearly so, 100 percent.

(2) Hyperleucocytosis, 40 percent, as high as 28,000, with polymorphonuclears occasionally predominating.

(3) Leucopenia, 60 percent, as low as 4,000.

(4) Startling increase of abnormal lymphocytes in the blood in all cases, at some period during the course of the disease.

(5) Presence of abnormal monocytes seen in 80 percent.

(6) Lymphoblasts in abnormal amounts in 35 percent.

SYMPTOMS

(*a*) *Fever.*—The temperature was taken in many cases by the parents, who became alarmed at the sudden rise. In 95 percent of all cases this initial rise of temperature always occurred at about 4 p. m. No chill preceded the rise in temperature. The patients became flushed, were lackadaisical for 3 days prior to the onset, initial rise of temperature was always over 100°, and in many cases reached 104°. The patients were "dopey" at this time, sallowness and a pinched expression of the face was noted in those over 4 years of age. Dreaming was constant with mild delirium.

(*b*) *Pain in back of neck.*—The symptom of pain in the back of the neck was investigated and in all of the cases it was due to cervical myositis shown by the exaggeration of pain when patients voluntarily or involuntarily rotated the head on the neck. The reason for this myositis is easily traceable to the intallible diagnostic symptom, namely, a particular type of adenopathy of the posterior cervical and glands of the neck. In differentiating this adenopathy from other types, canary bird, feathery palpation is essential so that the small, shoe-button glands may be made out bilaterally, numbering from 15 to 40, by running the finger over the mastoid process down to the acromio-clavicular joint. These glands are identical in feel with those of the glands felt in lues with the exception that they are mildly painful. This finding is now further checked by extending the lower limbs of the children and finding the same glandular condi-

tion in both groins and axilla. This glandular condition is persistent for a period of 1 month or more after the onset of convalescence.

(*c*) *Vomiting.*—The symptom of vomiting occurred in about 10 percent of all the cases and followed the rise in temperature. It was not severe and the withdrawal of cold drinks and solid foods, giving no cathartics, appeared to control this symptom and differentiated it from the vomiting seen in enteritis. There was no pain, tenderness, or rigidity of the abdominal musculature. The abdominal discomfort may be due to the involvement of the mesenteric glands and spleen and is apparently reflex in character. A constant search for enlarged spleens has failed to reveal any.

OBSERVATIONS

(*a*) Recovery of all cases to an apparently normal health status within 3 weeks has been noted. The blood picture, agglutination test, and noticeable specific adenopathy, all modified, remain however for an undetermined time.

(*b*) It is highly infectious for all children under 13 years of age. It must be differentiated from the exanthemata, catarrhal fever, acute, meningitis, influenza, tonsillitis, and diphtheria.

(*c*) Glandular fever in a baby, child, or adult seen for the first time is difficult to diagnose. When seen in epidemic form it is easily noted and is a pleasant condition to treat, for the aftermaths are few if any. In severe adult cases it must be differentiated from typhoid, diphtheria, lymphatic leukemia, common cold, tuberculosis, syphilis, influenza, and acute catarrhal fever.

Examples of maltreatment.—(*a*) A child, 5 years of age, developed a toothache. The mother put a hot-water bag to the child's jaw. The cervical glands on that side were enlarged and became extremely painful. The blood count jumped and the temperature rose.

(*b*) A child while on 5th day of convalescence was compelled by the mother to attend her acrobatic dancing school. Hand stands, etc., were the order of the day. That night all the glands in her neck were enlarged and painful. Temperature and blood count rose and the patient suffered a definite set-back.

(*c*) Because some 18 cases complained of pain in the back of the neck, mothers rubbed that part of the area with salve, ointments, etc., and in all of these cases rise in temperature, enlargement of glands, and rise in white count was noted.

(*d*) High temperatures in the rooms, 75° to 82° is considered harmful. In these cases convalescence was protracted, also in some few cases where solicitous mothers have given children hot tub baths.

Is epidemic grandular fever in children a blessing in disguise? Does it mean a percentage of immunity against the dreaded future pandemic of influenza vera?

GENERAL STATEMENTS

(*a*) Etiology to date unknown.
(*b*) Pathology indefinite.
(*c*) Apparent sequellae, poor resistant blood picture.
(*d*) Death rate nil if uncomplicated.
(*e*) Blood picture in epidemics definite.
(*f*) Introduction of this disease into a juvenile community is preceded by a mild epidemic of influenza in contact adult population.
(*g*) Influenza may demonstrate itself in the adult household following a case of glandular fever in the child.

Paul and Bunnel agglutination test.—If this simple agglutination test backs up the clinical picture of epidemic glandular fever and proves it to be a definite entity, then hematological investigation in the so-called milk epidemic of catarrhal fever, acute, and influenza is to be considered and encouraged, for it is a quick step in differentiation.

JULY 1, 1935—SECOND REPORT

THE THOUSANDTH CASE OF SO-CALLED EPIDEMIC OF GLANDULAR FEVER (INFECTIOUS MONONUCLEOSIS)

1. Its infectiousness is most manifest. It is now definitely established in this epidemic that the incubation period is about 5 days and no one living in the same apartment or house, be they infant, juvenile, or adult, if contact is daily, can escape infection. Because of this proven infectiousness and because it is now making itself manifest in the enlisted personnel of this area, if early recognition is made possible aboard a ship or station, it would seem advisable to quarantine.

2. Its classical picture is complete. Having seen from November 19 to July 1, 1935, not less than 2,000 cases in children, ages 1 month to 18 years, and not less than 55 adults up to the present writing (many of these cases in conjunction with other medical officers), I can clearly state that its clinical picture is constant and its symptomology the same as mentioned in prior article, dated December 30, 1934, or so-called first report.

3. Error in diagnosis almost impossible. Since November 1, in Coronado, whose total population is about 8,700, its juvenile population about 1,800, all attending one major public school—daily attendance averages 800 normally. There is a permanent school nurse in daily attendance at this school. Four civilian doctors practice in the afore-mentioned population. There is a public health nurse and a public health doctor located here also. In 7 months not a single case of scarlet fever, mumps, measles, whooping cough, diphtheria, influenza, pneumonia, septic sore throat, or tuberculosis has been reported by any physcian in this area. This is strange when we note the

fact that the daily absentees from the public school has averaged 100 cases daily since November 19. The cause of this absence has been illness and is epidemic in character, all cases similar clinically and hematologically.

4. Hematological findings are most helpful in backing up the clinical picture. Daily total white count which may vary from three thousand to forty thousand in adults or children. Those with the introductory high white count will drop to the low white count in 2 weeks' time. Those who start with a leukopinia of three to four thousand will rise to normal in some indefinite time.

Differential blood diagnosis at least three times a week to note shift of cells and sudden rise in total counts is essential.

Anomalies in differential—in the large lymphs enormous granules are noted in most cases. All the white blood cells appear to be fragile, and on stained smears will lose much of their shape and character. Lymphs show lymphoblasts and turk cells at intervals.

(*a*) The presence of monocytes in abnormal amounts at some time.
(*b*) Blood picture, if studied, is constant.
(*c*) All blood counts are taken in the morning.

INTERESTING COMPLICATIONS AND OBSERVATIONS

(*a*) Rash (dermatological manifestations).
(*b*) Earache.
(*c*) Giant local adenopathy.
(*d*) Stomatitis.
(*e*) Acetone in the urine.
(*f*) Unexplainable asthenia.
(*g*) Tracheo bronchitis.

The rashes we have seen during this epidemic in infants, children, and adults have been so complex that to classify them would be difficult. The cases numbered about 50. These rashes were first punctate, resembling the rash of scarlet. It was noted early in this condition that the rash appeared over the back and chest, then appeared on the arms, but never below the wrists—hardly ever on the face. (See picture no. 1.) The same may be said about the lower limbs—the rash stopped bilaterally at the middle of each thigh. (See picture no. 2.) Tape one showing line of demarcation, and tape number two sight of glandular enlargement. (See picture no. 3.) Appearance of rash showing tape over post cervical glands. The punctate rash resembling scarlet, then changing to a maculopapular form like that of German measles. It was most confusing.

The records in the case of W. W. E., seaman first class, U. S. N., is submitted to demonstrate differences of opinion by four medical officers:

February 12, 1935, admitted to the sick list, naval air station, diagnosis undetermined (scarlet fever); February 12, 1935, transferred to

naval hospital, where he was readmitted same date with diagnosis of scarlet fever (Schultz-Carlton test negative). February 13, 1935, diagnosis changed to German measles; February 19, 1935, to duty, recovered from German measles; March 3, 1935, readmitted naval air station, diagnosis German measles. This case was observed for a period of 40 days and the maculopapular rash was still present on the 40th day of the disease. At all times during these 40 days a generalized adenopathy was most apparent.

Acetone in the urine.—In 100 patients whose urine was examined daily, acetone was found to be present at some time during the acute onset, or early stage of this condition.

Unexplainable asthenia.—The two following cases I was in daily contact with:

Case no. 1.—A pharmacist's mate (note graph) (note leucopenia). This man had worked with me daily over a period of 5 months. Prior to this his infant child, age 3 months, developed so-called glandular fever. While the baby was convalescing, the father started to complain. He noticed the glands in the back of his neck and groins were enlarged and painful. Stiffness of both sides of his neck and a feeling of pressure on flexing the head on chest were other complaints. He called my attention to this. I placed him under quarantine, taking daily blood counts, etc. (See graph.) A rash like that shown in photograph, resembling German measles, appeared with lines of demarkation 1 week after onset, lasting over a period of 3 weeks. He was drowsy, suffered from a noticeable asthenia, and on exertion complained on three different occasions of feeling so faint that he had to lie down. He remained in quarantine only 4 days; then I returned him to work. The asthenia persisted for 1 month. His appetite at all times was excellent. He had a mild stomatitis. His sleep over a period of 2 weeks was not restful, and he stated that he dreamed nightly—all of his so-called dreams were pleasant. This case was considered one of protracted convalescence due to the fact that he was willing to cooperate with me to determine if activity prolonged the outstanding symptom of asthenia.

Case no. 2.—Active aviator, lieutenant, United States Navy, initials, W. B. W. This officer had two children, both of whom I treated for so-called glandular fever. On March 26, while this aviator, who is able and experienced, was flying out at sea over the airplane carrier *Saratoga*, he reported by radio that he was unable to make a landing after four attempts because of intense unexplainable weakness complicated by a feeling of indefinite faintness. Because of this message, he received radio orders from the commanding officer of the *Saratoga* to fly south some 100 miles to the San Diego base, where he landed with some difficulty. He was hardly able to stand when he climbed out of the cockpit. He went home exhausted and reported

to the sick bay the next morning. He was examined by two flight surgeons, who gave him the Schneider index (a test of circulatory efficiency) score of naught. This method of determining physical fitness is based on pulse and blood-pressure response to prone, standing, and after exercise. Arbitrary values are assigned to pulse and blood-pressure readings and their changes when posture is changed, and to pulse responses and return following exercise.

Because of his statement by radio to the flagship, plus his symptoms, plus the Schneider, he was immediately grounded and turned over to me for study and observation. As he arrived at the stage of convalescence, succeeding Schneiders were taken by aviation doctors with the following results:

March 26, 1935	zero.
March 27, 1935	plus 2.
April 2, 1935	plus 15.
April 5, 1935	plus 8.

Index values: 7, unsatisfactory; 8, poor; 9, satisfactory; 12 to 18, very good.

Acetone was present on two occasions. On April 5, against orders, he took a plane and made a short hop to determine if the asthenia returned. He flew for 10 minutes, returned, made a good landing, but again complained of asthenia and faintness while in the air. The fleet sailed on April 29. This lieutenant went aboard, apparently well, from a clinical and hematological standpoint. The following is his statement:

On March 26 I first noticed a loss of appetite, indigestion. I did not notice any fever. I flew that day, making two bombing hops of a total duration of 3½ hours. On the night of the 26th I took off from the deck of the *Saratoga* and was in the air 1 hour. I noticed a depressed feeling come over me while flying. I made four attempts to land, but because of the way I felt, decided it was too dangerous. I then requested radio permission to fly to the shore base. On this trip I noticed that my eyes were weak and tired. After landing, I went home and to bed, exhausted. The following morning I reported to the squadron at 9 a. m. I felt weak and finally reported to the chief flight surgeon.

He was examined and turned over to me as mentioned previously.

Tracheo bronchitis was noticed in about 5 percent of the cases. The duration of this condition was about 5 days.

SUMMARY

In 1928 on the east coast, and in 1934 on the west coast, on two occasions where mass adult influenza infection into controllable supervised communities were followed by so-called glandular fever, epidemics in the juvenile population—last port of call in both instances being the Canal Zone.

The individual or household observation in which the lymphatic influenza or glandular fever in the children of the family appeared,

the adults in the house became ill, manifesting symptoms of true mild influenza as we know it.

Vice versa, the adult officer who arrived at his home with mild respiratory type influenza infecting his children; they invariably responded with the clinical and hematological picture I have attempted to describe—this I saw in over 200 instances.

The fact that it was described by Pfeiffer while he was studying the influenza bacillus is of interest.

The clinical symptomatic picture in the youngsters, leaving out the adenopathy and blood picture, is the same as mild adult influenza.

Its contagiosity, asthenia, leucopenia, clouded etiology, known yet unknown pathology, its sporadic and epidemic nature, its variance in virulence as to individual age incident, time of year and geographic location, its complications, the rash seen in some epidemics of influenza being identical with that of so-called glandular fever, making for half-hearted diagnoses of scarlet fever and German measles, the difficulty attending the prevention of its spread, its recurrent and recrudescent phases all permit me to say "know influenza" and all its types, and the recognition of sporadic and epidemic influenza lymphatica will be diagnosed more often and we will more likely find that so-called German measles is but a dermatological manifestation of the present so-called glandular fever, but which I prefer to call influenza lymphatica. German measles in sporadic form is often diagnosed when the physician knows the case is not scarlet fever or true measles; therefore, it must be German measles because of the presence of a papular rash and an adenopathy. This is ordinarily the basis on which the diagnosis is attained. Now in the future let us investigate our so-called German measles by using the means at hand in a laboratory, and think in terms of influenza or so-called glandular fever. If this is done fewer diagnoses of German measles will be made.

CONCLUSIONS

(1) The early clinical picture in cases of high or low total white count are the same.

(2) One hundred and twenty-five bloods of adults and children with the typical clinical picture and blood counts were tested, using the Paul and Bunnell agglutination method. Twenty-five proved to be positive, 16 doubtful, and 74 negative.

(3) The severity of the case apparently does not enter into the picture.

(4) Close study of this test does not permit me to state at what period of the disease it makes its appearance.

(5) The test was positive, doubtful or negative early in high and low total white counts.

(6) Outstanding clinical cases, on repeated tests, refused to release the secret locked up in this Paul and Bunnell agglutination test.

(7) The test when positive is most picturesque and definite.

(8) It is hoped that this test will be used in all cases where epidemic influenza should appear, for in this way its relation to influenza, if any, will be brought to light.

(9) Cases diagnosed tentatively as mild scarlet and true German measles in 10 instances gave us a positive Bunnell.

(10) The type of adult that comes down with a lymphatica manifestation in this epidemic leaves the individual who is not susceptible to the common cold or seasonal grippe or catarrhal fever. It was also noted that their personalities were phlegmatic in type, generally.

(11) Osler, Hare, and others have observed that in epidemic influenza, regardless of type, contagious diseases of childhood seem to temporarily disappear. This fact was noted to a superlative degree in Coronado, from October 1934 up to and including May 1935.

WARNINGS

(1) In all cases convalescing: Any surgical procedure—this includes tonsillectomy and tooth extraction—repeated blood counts, and close observation will prevent embarrassment.

(2) On leaving Coronado to come here—many new officers' and enlisted men's families were arriving—it was noted that after 8 or 10 days the babies in these families were brought to the dispensary with what the parents stated was a cold in the head. These cases are now and will continue to be diagnosed influenza lymphatica and not glandular fever, monocytic angina, or infectious mononucleosis.

(3) A protracted lymphoid tissue convalescence is to be looked for if cases are mishandled.

ACROMIOCLAVICULAR DISLOCATION

By R. A. BENSON, Lieutenant, Medical Corps, United States Navy

Dislocation of the outer clavicular articulation is encountered not infrequently. Eliason cites 70 dislocations in 520 cases. Stimson found 69 in 1,527 cases. In the Navy for 3 years, 1931, 1932, and 1933 there were 43 admissions aggregating 1,958 sick days, an average of 48 days each.

The anatomy and symptomatology is so thoroughly discussed in current texts that it may be omitted here. Etiologically traumatism due to recreative sports is undoubtedly of major importance. Seventeen, or 40 percent of the Navy's 43 admissions were under this class. Football players are frequent subjects.

Treatment resolves itself to reduction and fixation. The former is accomplished with comparative ease, but fixation has been attended

with many difficulties as is evidenced by the numerous bandages and dressings that have been devised and recommended. The results from their use has not been altogether satisfactory, most writers indicating the necessity of open reduction in many cases.

The Velpeau and Desault bandages as well as Sayers dressing are only partially effective in that they fail to maintain fixation for more than a limited time. A simple type of dressing has been used by the writer which has proven very effective, is simple to apply and allows the patient a maximum amount of freedom (figs. 1 and 2). No originality is claimed for this method as it follows very closely a dressing suggested by Eliason. However, the attention is directed to certain features which if followed have been found to make this dressing valuable. The method is as follows: Two strips of adhesive plaster are cut, each 3 inches in width, one 26 inches and the other 48 inches in length. The shoulder is cleansed with ether, a pad is placed in the axilla, and another smaller one (2 by 3 inches) is placed over the outer end of the clavicle. With the patient sitting on a stool the first strip of adhesive is applied by grasping both ends and with firm pressure, placing it over the outer end of the clavicle down onto the back and chest. With an assistant supporting the arm upward at the elbow the second strip is started anterior to the clavicle, run backward over the outer end, then down and over the posterior surface of the arm, around the ulna, thence upward and over the outer end of the clavicle lifting upward as this is applied. The success of the dressing depends upon the placement of the plaster around the upper forearm. The lateral margin should be 2 inches from the tip of the olecranon. This then serves to give the forearm the role of a lever which directs its pull over the clavicular end. No sling or support is used for the hand, thereby allowing the forearm to act in the capacity of a lever at all times. This dressing should be continued for 4 weeks. Should the patient complain of the adhesive plaster cutting the inferior forearm surface a piece of basswood splint, padded on the upper side may be placed along the ulnar surface of the forearm with the adhesive plaster around same.

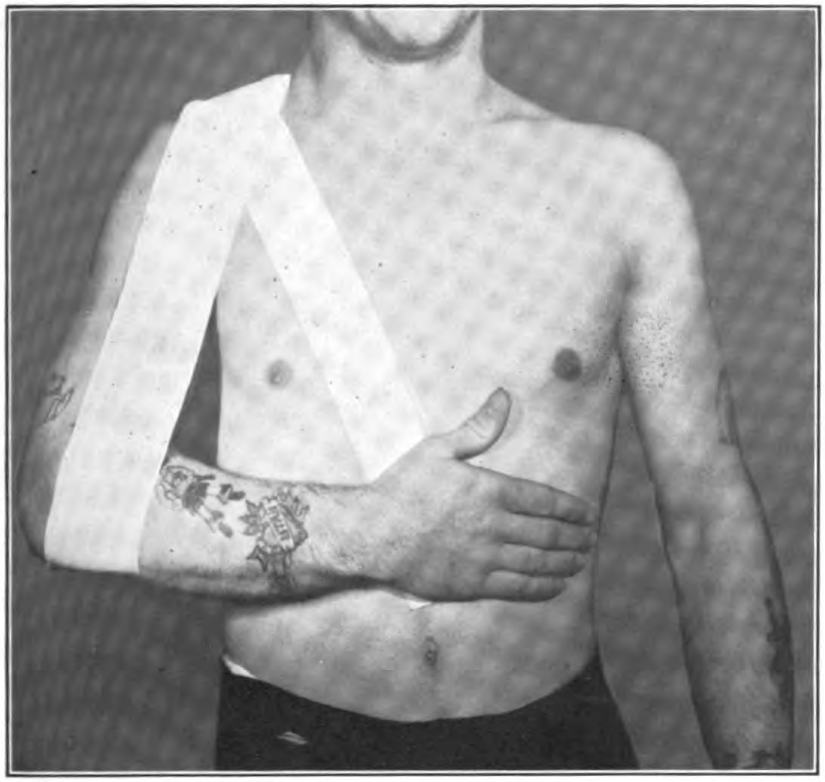
The patient is allowed free use of the hand, external and internal rotation of the shoulder joint. The axillary pad, being held loosely may be changed as often as necessary for comfort and cleanliness.

ENDOMETRIOSIS [1]

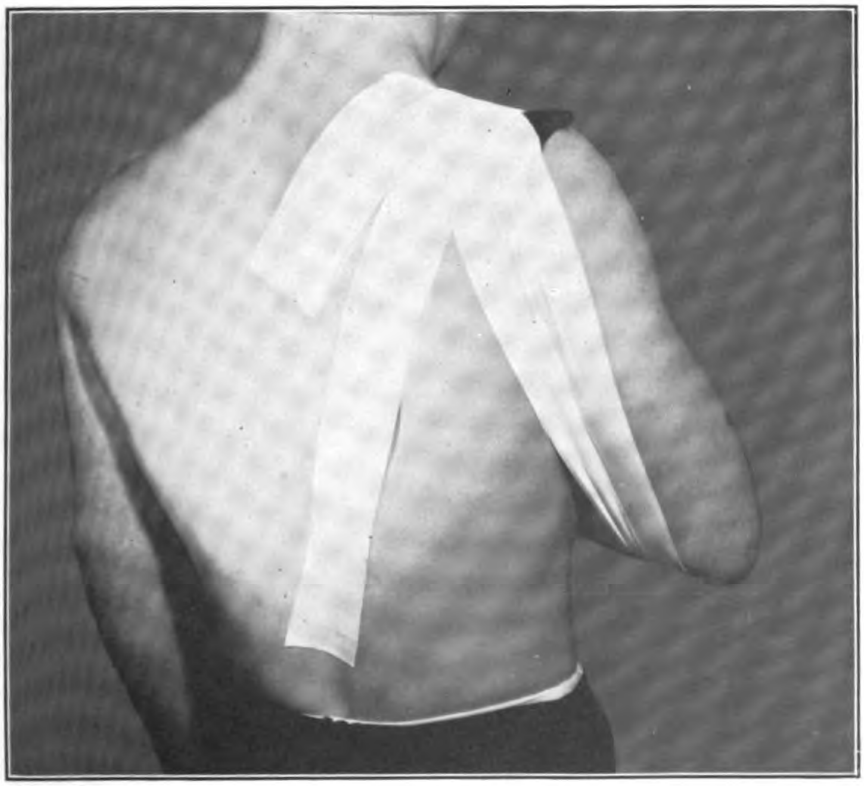
By ALBERT T. WALKER, Lieutenant, Medical Corps, United States Navy

Endometriosis may be defined as a pathological condition in the female characterized by the presence of ectopic implantations or

[1] Read before the section on obstetrics and gynecology of the Territorial Medical Society, Territory of Hawaii, Honolulu, Apr. 27, 1935.



Front view.



Back view.

DRESSING FOR ACROMIOCLAVICULAR DISLOCATION.

growths of endometrial tissue. It is also referred to as adenomyosis, endometriema, adenomyema, all of which are more or less synonyms for the same disease condition, but may place it anatomically a little more accurately than the general term endometriosis. However, endometriosis covers the range of possible areas involved and is as good a term as any to designate this condition.

The areas involved in this peculiar condition are usually divided into two classes, those with intrapelvic involvement and those with extra pelvic involvement. Practically any of the pelvic structures may be involved in this process and in addition certain of the abdominal viscera and certain areas of the abdominal wall may be involved. A simple classification of these tumors is as follows:

Intrapelvic: 1. Uterine:

(*a*) Central or diffuse involvement.

(*b*) Periphenal or circumscribed.

2. Extrauterine:

(*a*) Ovarian or peritoneal.

(*b*) Rectovaginal.

(*c*) Round ligaments.

(*d*) Sacral ligaments.

Extrapelvic:

1. Inguinal or round ligaments in abdominal wall.
2. Umbilical.
3. Abdominal wall scars.
4. Intestines.
5. Liver, gall bladder, kidney, etc.

By this classification we see that all pelvic structures and many extrapelvic structures may be the site of growth of these extopic pieces of endometrial tissue. It is evident, therefore, that the symptoms and the pathology of this disease will vary according to the areas involved.

An important, and probably the most interesting phase of this problem, particularly from a pathological standpoint, is the probable origin of this tissue. There is much discussion in the literature concerning this point, with Sampson ranging himself on the side of implantation and Iwanoff and Novak principally feeling that the growths may be the result of cellular changes in otherwise normal epithelium. Sampson was the first to report the presence of these chocolate cysts as probable implantations of normal uterine mucosa upon the ovaries and peritoneum and advances the theory and supported it with observations that the endometrium passed in retrograde fashion through the tubes into the pelvic cavaity with regurgitated menstrual blood. He believed that all endometrical transplants originated in this manner. Iwanoff, and more recently Novak, advanced the Mullerian theory inasmuch as they were unable to

account for some of the ectopic growths purely on a basis of migration through the fallopian tubes. They feel that certain tissues, particularly epithelium lining the peritoneum and covering its viscera, is able to undergo metaplastic changes in the presence of some unknown stimulus, possibly hermonal, and assume the characteristics of endometrial tissue. Robert Meyer also supports this theory and has made certain observations which tend to substantiate it.

The evidence at hand supports the view that these growths originate both from adult endometrial tissue, that is uterine mucosa, and also from embryonic Mullerian tissue. It has been definitely shown, and Sampson admits this possibility, that there is also some metaplasia of the mucosa of the fallopian tube which, undergoing epithelial changes, assumes the character of endometrial tissue. So we have evidence to prove that both schools of thought are correct and that these endometrial tumors arise both from abnormal development of Mullerian tissue and as a result of implantation of normal uterine mucosa which has migrated in retrograde fashion through the fallopian tubes. Another avenue of spread which will be just mentioned is direct extension from the endometrial lining of the uterus throughout the muscularis of the uterus to form the diffuse adenomyesis so frequently seen in connection with uterine fibroids and so often only recognized on microscopic section. Taussig summarizes our present knowledge of endometriosis as follows: The tubes are always open, menstrual blood passes out through the tubes in retrograde fashion, the endometrial tissue so carried to the cul de sac is implanted thereon in a pattern which can only mean direct implantation. We also have definite evidence of development of various forms of tubal, uterine and even squamous epithelium from the coelomic epithelium and there is also evidence of traumatic metaplasia at certain points, particularly at the fimbriated ends of the tubes. We can therefore not be content with accepting one theory as explaining all cases, we must agree there is definite metaplasia and we must agree there is also direct implantation and must explain the origin of ectopic endometrial tissue on this basis.

To come to the more practical aspects of the subject we must first consider diagnosis. The outstanding symptom of this condition is pain at the menstrual periods. And as important as the pain is the fact that it is acquired during the patient's menstrual life. The patient usually comes to the physician complaining that all her life her periods have been regular with a normal painless flow but that for the past few months she has had much co-menstrual pain and that she bleeds a few days longer than previously. The menorrhagia is particularly characteristic of the diffuse adenomyosis in which the uterine wall is involved. There are few symptoms present other than the above. Many women go on for years with endometrial

tumors until they assume large proportions and are extensively invasive before seeking medical care because the only symptom they have is acquired dysmenorrhoea. If the growth has invaded the intestinal tract the patient may complain of constipation and have the symptoms of obstruction. If the growth involves the rectovaginal septum there may be signs of obstruction at that point and also pain on defecation. No matter where the growth is located the symptoms complained of are enhanced at the menstrual period. This is due to the localized swelling incident to the extravasation of blood from the menstruating tissue. On examination the findings will depend also on the position of the growth. If the uterine wall is diffusely involved one finds enlargement of the uterus which is somewhat softer than that found with fibroids, the uterus will increase and decrease in size with the menstrual periods. Quite frequently in this condition the only enlargement of the uterus will be in the anterior posterior deameter. It is of paramount importance to examine the patient before and after her periods in order to note the changes in size and shape of the uterus with the menstrual congestion and post-menstrual involution. If the implants involve the ovaries they can be palpated as enlarged and cystic, but they differ from simple cysts in being painful and fixed.

There is usually puckering of the mucosa of the fornices, and particularly of the cul de sac, when this area is involved, as these implants are invasive and cause an inflammatory reaction which results in scar tissue formation. If situated in the rectovaginal septum, there will be felt a nodular mass in that region which may or may not be painful but which will fill and empty with each menstrual period and can be watched over a period of months, turning dark blue and filling with blood with the premenstrual changes going on elsewhere and subsequently regressing to a small punctate area after the period has ceased. Growths on the intestines may show no physical findings except by X-ray if they are extensive enough to cause deformity. Most of the lesions are found coincidentally during the course of pelvic operations for other conditions.

Development of this type of tumor depends on the presence of the follicular hermone of the ovary. Treatment therefore consists in the temporary or permanent cessation of ovarian activity. This is a difficult condition to treat if at all extensive, because adequate treatment of the lesions in young women demands the undesirable procedure of castration, either by surgical means or by radiation therapy. In small lesions in young women who have no symptoms, I believe the patient should be treated conservatively, the lesion observed occasionally, and active treatment instituted only when indicated by the appearance of unfavorable symptoms. When encountered during the course of pelvic operations the local growth, if not extensive, should be

excised. If situated on the ovaries and adjacent peritoneum, conservative ovarian surgery should be attempted with removal of only that portion of the ovary and parietal periteneum which are actually involved. In some cases, however, the pathology would be so extensive that radical surgery is necessary. It is well to emphasize that the growths are invasive, frequently involving ovaries, tubes, peritoneum, and intestines. In order to dissect this mass free from the intentines it may be necessary to injure the intestinal coats, cutting into them down to the mucosa in order to remove all of the endometrial tissue which has invaded the structures. In these instances it is well to keep in mind the fact, particularly with very extensive growth, that the tissue is kept active by ovarian hermones and that if it is surgically dangerous to excise the whole growth, its partial excision, with removal of all ovarian tissue, would be sufficient. The removal of the ovarian tissue removes the stimulating effect of the follicular apparatus and allows regression of the endometrial growth. In women past the child-bearing age or in whom the family is complete, panhysterectomy is the treatment of choice.

There are, of course, some women who are such poor surgical risks that extensive surgery is contraindicated. In these patients a sterilizing dose of radium or deep X-ray will usually cause regression of the tumors. This effect of course is based upon the production of an artificial menopause, much the same as that produced by excision of the ovaries.

In our practice in the past year we have had a number of cases of endometriosis involving many of the structures noted under the classification. The most common lesion with which we have had to deal is that caused by an invasion of the uterine muscle or a fibroid by these endometrial elements. These cases are all cured by either supravaginal or complete hysterectomy with conservation of the ovaries and offer no particular surgical problem. Other cases have shown extensive involvement of both ovaries together with the peritoneum and adjacent intestines and have presented rather serious surgical problems. In these patients it is necessary to free the intestines from the invading growth by sharp dissection, which, if done completely, may result in invading the intestinal canal. The removal of such an extensive growth results in the production of rather large surfaces denuded of peritoneum which must be thoroughly peritonealized if good postoperative results are expected. I might add that in this class of case with intestinal involvement much of the pain of which the patient complains is due to dragging on the intestines and to a fixed immovable spot which cannot move freely with peristalsis. The surgical removal of these fixed points with their proper peritonealization with allow normal intestinal movement with peristalsis and result in cessation of pain. It has been our experience that properly performed surgery

offers maximum benefits to patients with endometrial growths and that the proper surgical procedure depends upon a knowledge of the pathology and the physiology of these tumors.

TREATMENT OF LUNG ABSCESS

HOWARD L. PUCKETT, Lieutenant, Medical Corps, United States Navy

It is not the purpose in this discussion of lung abscess to reveal anything new or startling, but to point out some of the old and well-established procedures in the treatment of this disease. Because of its discouraging morbidity and mortality rate, there have been too many attempts to use untried and hypothetical therapeutic methods. Such attempts are often dangerous and sometimes even fatal.

Lung abscess is an uncommon disease; many physicians are in practice years or even a lifetime without seeing a single case. It is not surprising, then, that when a case does present itself to the average physician, there is not a little confusion regarding the treatment. There is nothing more gratifying than the results obtained from the thoughtful treatment of lung abscess, and with this in mind the following system of therapy is presented.

Prophylaxis.—Everything possible should be done to prevent lung abscess since it has such a long morbidity and high mortality rate. Aspiration of infection is the most frequent cause of this disease, accounting for almost 50 percent of the cases. Tonsillectomy is the greatest etiological factor in this category. Mouth and throat operations should be avoided under a third stage general anaesthesia, during an acute respiratory infection, and when the oral hygiene is poor. Local anaesthesia for throat and mouth operations is usually the safest procedure. Maintenance of the cough reflex cannot be overemphasized.

Aspiration of vomitus in the semiconscious patient, as sometimes occurs in lobar pneumonia, alcoholism, and general anaesthesia, frequently results in abscess of the lung. This could be prevented in most cases by turning the patient on his side, a problem of alert nursing. The following is a case of pulmonary abscess which probably had its origin in the aspiration of vomitus during an attack of lobar pneumonia.

Case 596. V. G., a young man, aged 18, suddenly developed a chill on December 31, 1934, which was followed in a few hours by a fever and general malaise. There was some pain in the right chest and a slight cough. The second day after the onset, general malaise, pain in the chest, and cough had become more marked. The respiratory rate was increased above normal, and there was some expectoration of rusty sputum.

The past history was essentially negative, except that he had been bothered with a slight cough prior to his present illness. There was no history of operation or recent dental work.

On examination, the face was flushed and the skin hot and moist. The lips became cyanotic about the sixth day. The respiratory rate was increased and ranged between 30 and 40 after the first week. The pulse rate varied between 110 and 150. The temperature was constantly maintained between 101° to 104° F. On the third day a marked jaundice appeared which continued until January 11. There was frequent vomiting during this period. The stools were clay colored and the urine was highly concentrated, containing a slight amount of albumin.

The chest revealed less movement on the right side. Tactile fremitus was increased over the right lower lobe. Over this area dullness was pronounced by the third day and the voice and breath sounds had a bronchial character. The sputum was typed and found to contain group four pneumococci.

A diagnosis of lobar pneumonia of the right lower lobe and toxic hepatitis was made.

January 9, the intercostal spaces were bulging; the percussion note was flat, and the voice and breath sounds were absent over the affected area. The patient had become much more toxic and all of his symptoms were aggravated. The last rights of the church were administered.

The condition was thought to be complicated by an empyema. A thoracentesis was done and repeated several times over a period of about 24 hours, but less than 10 cc of fluid was obtained. This fluid was pinkish in color and gelatinous in consistency, containing a few pus cells per high power field. The temperature, pulse, and respiration took on a septic character.

January 10, the patient suddenly coughed up about a pint of foul purulent sputum. He was placed on postural drainage for 3 minutes twice daily and, thereafter, from 800 to 1,500 cc of foul purulent sputum was obtained every 24 hours. The expectorated matter revealed pneumococci, but no acid fast organisms or spirilla. His condition was improved enough by January 12, 1935, to have the chest X-rayed. A lateral and anteroposterior röntgenogram was made and a diagnosis of acute lung abscess of the right middle and lower lobes was established.

Perspiration was profuse. There was an estimated fluid loss of more than 2,400 cc every 24 hours from sweat alone. To replenish the excessive loss from sweat and postural drainage, hypodermocylosis, veinocylisis, proctocylisis, and forced fluids by mouth were given daily. The hemoglobin was 54 percent. The patient was given 15 grams of iron and ammonium citrate four times daily to combat this. Brewer's yeast was given to increase his appetite, cod liver oil for its tonic effect, and a high caloric, high protein diet to restore the tissue loss.

The temperature, pulse, and respiration gradually decreased until February 3, 1935, when there was a sudden rise above the highest recent preceding level. However, by postural drainage an average of over 1,000 cc of purulent sputum every 24 hours was still obtained. Nevertheless, an X-ray of the chest on February 4, showed that the drainage from the abscess was inadequate. In order to promote further drainage it was decided to do a thoracotomy.

The first stage was done February 5 by resecting the ninth rib and exposing the pleura at the midscapular line under 1 percent local procaine anaesthesia. The wound was packed with gauze to promote the formation of adhesions between the parietal and visceral pleura. The second stage was completed February 8 by locating the abscess with a hypodermic needle and syringe, and then passing a grooved director along the course of the needle until a sinus was formed. The opening was dilated with a hemostat. A large amount of foul pus drained out and two soft rubber drains were inserted into the cavity.

The position of the drainage tubes was changed daily, and they were removed on the tenth day. Thereafter, the sinus was dilated with the finger as often as necessary to obtain sufficient drainage.

The patient gained weight; his appetite improved; his temperature, pulse, and respiration reached a normal level; and by March 7 he had sufficient strength to get into a wheel chair.

Although the patient vomited several times daily during the first week of his illness, there is no positive evidence that he aspirated any of his vomitus. However, he was extremely ill and semiconscious, and it is likely that he did aspirate some of this material. The lung was already damaged to such an extent that this added irritation produced the necrosis and actual destruction of the lung tissue. Perhaps, if this patient had received more careful and expert nursing attention during this acute phase of the pneumonia, the possible aspiration could have been prevented.

The etiological factor in the next case was probably the aspiration of vomitus. At least it was known that the patient was often in a condition to favor such an aspiration, since he was almost constantly under the influence of alcoholic beverages.

Case 2–F. A man, aged 42, was hospitalized on several occasions over a period of 18 months for a cough and pain in his chest. This was associated with ease of fatigue, night sweats, and the expectoration of 300 to 600 cc of purulent sputum daily. He was known to be a chronic alcoholic and had a diagnosis of lung abscess from the time of his first admission. He was treated by postural drainage with considerable improvement, but the abscess failed to heal. Operation was advised, but the patient refused for some time.

The patient appeared undernourished. He had marked dental caries, oral sepsis, and inflamed tonsils. His chest was slightly emphysematous in type, mobility was not impaired. Percussion note was slightly hyperresonant, except over the right lateral portion just below the axilla where there was some dullness and a friction rub. Coarse rales were present about the hilum.

The temperature, white blood count, and sedimentation rate were elevated. The temperature was septic in type. Repeated sputum examinations were negative for acid fast organisms. X-ray revealed an abscess in the right lower lobe.

The patient received a bronchoscopy March 4, 1932, without any apparent benefit. Finally, a thorocotomy was done, and after a long but uneventful convalescence, the patient was discharged symptom free.

The third case is an example of a lung abscess following the extraction of a large number of teeth under general anaesthesia, another common cause of aspiration.

Case 3–C. A man, 36 years of age, was admitted to the hospital complaining of weakness and loss of weight, which had been present 6 weeks. The condition came on a week after extraction of his teeth under ether anaesthesia. The onset was marked by chills and fever. Soon afterward he developed a productive cough and expectorated about 200 cc of purulent sputum daily. He was confined to bed 3 weeks and lost about 26 pounds of weight.

On physical examination, the patient appeared pale and undernourished. He was edentulous and had hypertrophic, inflamed tonsils. His chest was asthenic in type with impaired mobility on the left side. Fremitus was decreased over the left apical region and dullness was found from the seventh rib to the base on that side. Coarse rales could be heard about the hilum on the left. The breath sounds were greatly diminished over the lower left lobe. X-ray revealed an ab-

scess in the left lower lobe. The sputum was negative for acid fast organisms, spirilla and fusiform bacilli.

After a period of 3½ months on postural drainage and high caloric diet, the patient was discharged home asymptomic. X-ray failed to reveal any evidence of cavity at the time of discharge, but there was a certain amount of fibrosis at the old site of the abscess.

When foreign matter is known to exist in the lung, prompt bronchoscopic removal should be contemplated, notwithstanding the fact that in the healthy individual, such substance is frequently absorbed, coughed up, or encysted. If all patients who aspirated foreign material developed a lung abscess, it would be a very common disease. A tooth has been known to remain in the lung for years without causing any remarkable disability. Nevertheless, the majority of lung abscesses follow aspiration, and bronchoscopy offers the best means of preventing this disease by removal of the foreign substance from the lung.

Blood and lymph infections leading to lung abscess can often be traced to improper care of the primary infection. For the most part, the local lesion receiving adequate external drainage and physiological rest will heal without further extension. Infection about joints should receive special attention in regard to rest of the part. Hot hypertonic solutions of salt and incision of fluctuating areas will increase the evacuation of pus and promote healing.

Medical treatment.—The general principle underlying the medical treatment of lung abscess is the same as that of the surgical treatment. The only difference is in the method of approach. Both require a great amount of thought, judgment, and responsibility. The general principle is that adequate drainage must be obtained, and, at the same time, the general physiological processes of the patient must be carefully watched.

From a medical standpoint, postural drainage is the best established and most reliable method of obtaining evacuation of the septic matter from the abscess cavity. Obviously, the success of this procedure depends on the communication of the abscess with a bronchus, a condition usually present early in the process of the disease.

Postural drainage is often the only effective method of treating the very sick patient. In contradiction to Brunn (1), it is courting disaster to *fail* to apply this remedy when the patient is "cyanotic, with high fever, rapid pulse, and a low vital capacity." Remove the pus from the abscess cavity and the vital capacity will increase and the cyanosis will disappear. A full abscess cavity is the source of much of the toxicity and cardiac embarrassment, therefore, when it is emptied the fever and pulse will decrease. Postural drainage is safer than operation for such a patient, exhausted from coughing and drowning in his own pus. It is better to have 2 or 3 patients die from the use of gravity drainage than have 15 or 20 die from not using it. Failure

to use this well-established and tried remedy in the very sick lung abscess patient will not help to lower the high mortality rate.

On the other hand, if a routine method of postural drainage is established without consideration of the general condition of the patient and the specific underlying pathology, the results will be successful only by chance. The patient may be able to take 1 or more minutes of drainage with only the foot of the bed elevated, or he may be strong enough to hang his head and shoulders over the side of the bed. Enough drainage is often obtained from the very sick patient in 1 or 2 minutes to reduce the toxicity and give him some rest from excessive coughing. Occasionally, as much as 500 cc of purulent material may be obtained in this short period. First, the patient should lie on the healthy side to permit drainage from the cavity, and second, in order to prevent the infected material from lodging in the normal bronchi, it is important to remember to turn the patient on the diseased side to complete the process.

The patient loses a large amount of fluid from drainage of the abscess, from perspiration, and from an increased respiratory rate. This loss may be so great that a combined method of fluids orally, intravenously, rectally, and subcutaneously may be required in order to obtain a balance in water metabolism. Severe cases with large abscesses may lose as much as 7,000 cc of water every 24 hours. An estimate of the fluid loss can be roughly obtained by weighing the sweat absorbed in the bed clothing for a definite period, by measuring the urine and the sputum, and by calculating the normal vapor loss from the breath and skin. From 30 to 40 cc of water is lost every hour at basal conditions.

A high protein, high vitamin diet will aid in restoration of the destroyed tissue, increase the appetite, and probably the tissue resistance of the patient as well. A liberal amount of vitamins A, B, C, and D should be included, especially vitamin B. Eggnogs are good because of the protein, minerals, vitamins, and fluid contained in them. Liberal doses of iron are helpful in combating the secondary anemia. Iron and ammonium citrate is a good preparation to use because it is practically nonastringent, and may be given in doses as high as 90 grains daily without any appreciable gastro-intestinal irritation. In the extremely emaciated patient, care should be concentrated on the skin to prevent the development of bed sores. Daily back massage with alcohol will increase the circulation in the superficial tissues, and talcum following the massage will decrease the friction of the bedclothes against the body.

Persistent cough may be relieved by administering a half a grain of codeine every 4 hours, and hemorrhage may be combated by quarter grain doses of morphine. When adequate drainage of the abscess is obtained sedatives are usually unnecessary.

A careful study of the temperature, pulse, and respiration should be made daily. If there is a progressive decrease in these, medical treatment should not be abandoned for surgical intervention. These three physiological processes may remain within normal limits when the disease becomes chronic, providing there is adequate drainage. If there is an exacerbation of the temperature above the highest recent preceding level, further X-rays should be made and surgical intervention kept in mind. This change usually means that there is insufficient drainage from the abscess cavity, or that there has been an extension of the infection.

At least one lateral and one anteroposterior X-ray is advised in all rontgenological studies of the pulmonary abscess. The rontgenologist, no matter how extensive his training, who makes a hasty diagnosis of these films without taking into consideration the patient's clinical signs and symptoms is going to arrive at erroneous conclusions in a large percentage of the cases. X-ray interpretations of this order for the uninitiated are dangerous, since they may lead to the wrong method of treatment, and perhaps, the death of the patient.

Surgical treatment.—The most common form of operative treatment of lung abscess is thoracotomy. The operation in itself does not require a great amount of skill, but when and where to operate demand considerable judgment. The two following cases are illustrations of the danger in delaying surgery. Grave complications directly traceable to the abscess were present in both at the time operative interference was considered, and one of them was so dangerously ill that a thoracotomy was not deemed advisable. Death followed pulmonary hemorrhage in both patients.

Case 4–H. A man, aged 38, was admitted to the hospital complaining of a productive cough which had been present 7 months; the condition followed pneumonia. He expectorated from 100 to 300 cc of foul, purulent sputum daily. His physician prescribed postural drainage and the foul odor disappeared from the sputum, but the quantity expectorated did not decrease in amount.

On physical examination, the patient appeared chronically ill. His temperature was 100° F. and his pulse 102. His tonsils were enlarged and diseased. His chest was slightly emphysematous in shape. Tactile and vocal fremitus was increased over the lower, posterior right chest extending up to the third rib. The percussion note was hyperresonant over the same area. Breath sounds were increased over both sides posteriorly between the eighth and tenth ribs, and there was amphoric breathing on the right side over an area about the size of a silver dollar. Coarse rales and a friction rub could be heard in the same vicinity.

His sedimentation rate dropped the first hour to 14 mm. He showed a slight secondary anemia with 14,300 white cells and 70 percent polymorphonuclears. The sputum was negative for acid fast organisms, spirilla, and fusiform bacilli. X-ray revealed an abscess of the right lower lobe.

The patient was placed on postural drainage and immediate operation was advised. Finally, after 12 days in the hospital, the patient consented to a thoracotomy.

The operation was done in two stages. The second day after the last stage of the operation the patient developed a pulmonary hemorrhage and died. Autopsy revealed chronic pericarditis, chronic obliterative pleurisy, chronic multiple abscesses of the right lung, and chronic glomerular nephritis.

Case 5–K. A man, aged 43, had a productive cough and pain in the right half of his chest, which had been present 4 months. The condition started with a cold in the head and chest following exposure from living in a room without heat. He expectorated from 100 to 250 cc of foul, purulent sputum every 24 hours. There was a progressive weakness and loss of weight.

On physical examination, the patient was found to be extremely emaciated. The breath was foul, the teeth carious with a marked pyorrhea, and the tonsils were enlarged and inflamed. The chest had a flat shape with a slight lagging of the respiratory excursions on the right. Tactile fremitus was increased on the right anterior chest wall between the second and the fourth ribs; the same area was hyperresonant to percussion; and coarse rales could be heard throughout both lung fields.

The temperature was septic in type with a corresponding change in the pulse rate. There was a leucocytosis with an increase of the polymorphonuclears. The sedimentation dropped 30 mm in the first hour. Repeated sputum examinations were negative for acid fast organisms and spirochetes. X-ray revealed an abscess of the right middle and lower lobes.

The patient was placed on postural drainage and 200 to 300 cc of foul, purulent sputum was obtained daily. Operation was not deemed advisable due to the extreme toxic and emaciated condition of the patient. Thirteen days after admission to the hospital he died from pulmonary hemorrhage. Autopsy revealed abscess of the right lung with gangrene.

The abscess is definitely localized when possible by X-ray study and by physical findings. Rib resection is done over the most dependent point and the nearest wall of the abscess. The safest procedure is an operation in two stages. The first stage is to promote the formation of firm adhesion between the parietal and visceral pleura. A skin incision of 5 or 6 inches over the rib or ribs to be resected is made under 2 percent local procaine anesthesia. The intercostal nerves and periosteum are infiltrated with the anesthesia, otherwise there may be considerable pain. Four or five inches of the periosteum is stripped from the ribs before they are resected. The parietal pleura is then exposed, and the wound is packed with gauze, which is left in place for 3 to 5 days. If the abscess wall is near the periphery, the pleura may be so necrotic that the pus will break through when it is exposed. If this should occur, complete the operation in one stage by inserting into the cavity one or two soft rubber tubes one-quarter to one-half inch in diameter.

Before the second stage of the operation is started, a test for adhesions is made by inserting a hypodermic needle into the pleura. If the end oscillates with the respiratory excursions, the parietal and visceral pleura are not adherent, and the operation is deferred until ample adhesions have formed.

To locate the abscess insert an aspirating needle through the pleura in the direction indicated by X-ray and physical findings;

pus can be drawn through the needle when the abscess wall is penetrated. Enlarge the opening along the course of the needle with a grooved director or cautery until a sinus about one-half inch in diameter is formed. One or two soft rubber drains are then inserted into the cavity to complete the second stage.

Change the position of the drainage tubes daily to prevent pressure necrosis of the adjacent blood vessels and hemorrhage. The tubes are removed in 10 to 14 days, and the draining sinus is kept open by digital expansion. Frequently the sinus closes before the cavity is healed. Then the procedure is to repeat the operation by secondary resection of the ribs.

Pneumothorax is sometimes a valuable adjunct in the treatment of lung abscess. It is particularly helpful in the drainage of centrally located abscesses where medical treatment has failed and external surgery is not advisable. Large cavities with extensive adhesions and thick walls, which have failed to respond to more conservative surgical measures, are often treated successfully with thoracoplasty. This operation is sometimes necessary for extensive chronic multiple abscesses, and, should it fail in obtaining the desired results, lobectomy may be tried as a last resort. But lobectomy, even under the most favorable conditions, results in a high operative mortality.

CONCLUSION

To avoid the more common errors in the treatment of lung abscess, some of the well-established therapeutic procedures have been discussed. The application of these methods with judgment have been shown to reduce its mortality and morbidity rates (*2*).

REFERENCES

(1) Brunn, H.: Lung Abscess. J. A. M. A. 103: 2000 (Dec. 29) 1934.

(2) Halpin, J. A., Ellis, J. W., and Puckett, H. L.: Pulmonary Abscess. Am. J. Surg. 23: 547–550 (Mar.) 1934.

THE RELATIVE PROTECTIVE VALUE OF VARIOUS PROPHYLACTIC DRUGS AND METHODS FOR THE CONTROL OF VENEREAL DISEASES

By R. C. Boyden, Lieutenant, Medical Corps, United States Navy

During the early part of a tour of duty on the Asiatic station, the writer was forcibly impressed with the incidence and prevalence of venereal disease on the station. In the fall of 1932 the average number of weekly injections of neoarsphenamine on 1 ship alone was between 80 and 90, the total complement being about 500. The venereal restricted list for gonorrheal and chancroidal infections was a lengthy one. This in spite of periodical instruction in matters pertaining to the prevention of these infections, the availability of materials for prophylaxis, both aboard ship and at the gangway for liberty parties.

It was observed that the number of admissions for these conditions went through a fairly regular cycle, the curve rising and falling in general phase with the ports visited; i. e., the rate was always higher in Shanghai than in Chefoo, etc.

The sanitube had been, generally speaking, the mainstay for prevention on the station for a number of years. It was noted that the admission rate was higher in ports where the sanitube was the principal prophylactic agent used. In these ports first-aid stations were either lacking or extremely inaccessible. The writer has been unable to find any reports of research on the value of calomel ointment, where such research was conducted under reasonably surgical or aseptic conditions. The value of this and other methods had always been taken for granted.

The average number of venereal admissions per thousand for 1932 on the Asiatic station was 445, or more than 3 times the rate of the entire Navy.

With these facts in mind the writer was led to question the value of methods then in use, and determine if possible the existence of more effective ones. The study was made in Destroyer Squadron 5 embracing a personnel of about 2,400.

Generally speaking, all ships moved as a unit between Manila and the various China ports. This relatively homogeneous movement aided very materially in the proper interpretation of the reasons for the rise and fall in the admission rate from month to month as the ships moved to the various ports. Added to this was the fact that in Chefoo, during a portion of the time in which the study was made, all prostitutes were in a segregated controlled district and were subjected to regular medical examinations by local Chinese doctors. All men leaving this district were required to take prescribed prophylaxis. It is safe to assume that 95 percent of all exposures in the fleet during this period occurred in this district.

The first-aid station was situated inside the district adjacent to the only exit, and every man taking prophylaxis was given a numbered ticket which he surrendered to the patrol on his way out. If he fell more than five numbers behind he was directed to return to the station for another prophylaxis. This was done to prevent men returning to the houses for another exposure after prophylaxis had been administered.

Prophylaxis consisted of thorough washing with soap and water, an injection of one-half percent protargol retained for 5 minutes, and a liberal inunction of calomel ointment. This was done under the supervision of a chief pharmacist's mate. The individual's name, rate, and ship was recorded, along with the number of the house in which the exposure took place and the time elapsing subsequent to the exposure.

During the first year of the period embraced in the study emphasis was placed on the sanitube (calomel ointment), soap, and protargol. In cities where controlled districts did not exist (Shanghai, Hong Kong, and Manila) all men going over the gangway exhibited a sanitube as a part of their shoregoing equipment. It will be seen upon referring to figure 1 that, in general, the admission rate increased markedly where almost complete dependence was placed on the sanitube. There were relatively few first-aid stations in these ports, and they were poorly situated. A very large majority of the men exposed used a sanitube immediately following exposure and delayed further efforts at prophylaxis until they returned to the ship, usually the following morning. Many of these contracted venereal disease in spite of the calomel ointment.

Figure 1 depicts the number of original admissions in the entire squadron for the years 1932–34. The ports visited are chronologically listed. It will be noted that during the first year in Chefoo, under controlled prophylaxis, the number of admissions was lower than for the time spent in Shanghai that fall. This speaks well for immediate prophylaxis, which was available in Chefoo, but not in Shanghai. However, the admission rate per thousand for that year on the Asiatic station was more than three times the rate for the entire Navy, which leads to the conclusion that even controlled prophylaxis did not accomplish its purpose. It is believed further that, inasmuch as conditions were nearly ideal for prophylaxis in Chefoo, a good share of the responsibility for the high admission rate rested on the materials used. It was ascertained that the average time elapsing between exposure and prophylaxis during this time was about 15 minutes. Such a period is well within preconceived theories concerning the time-limit effectiveness of venereal preventative drugs. Nevertheless, the admission rate continued high under this system.

In October 1932 certain disciplinary measures were instituted for chronic offenders who were more or less continually on the sick list with venereal disease. "Repeaters" or those who had several admissions within 1 year were also subject to these measures, which will be described more fully in a subsequent paragraph. The full effect of these measures did not become apparent for 1 or 2 months, but the admission rate did eventually drop and remained consistently low for the next year.

Referring again to figure 1, it will be seen that the number of admissions during 1933 was considerably lower than in 1932. Prophylaxis and the segregated controlled district were essentially the same as in the previous year, so a share of the decline can rightly be laid at the door of the disciplinary measures instituted near the first of the year. There were, however, one or two other factors which were considered also to have had a part in the decreased number of admissions. Prin-

cipal among these was the fact that the pay of the men for this particular year was approximately 50 percent less than in 1932, due to economy law deductions, plus the great decline of the United States dollar on Chinese exchange after the United States went off the gold standard. The total number of exposures in the controlled district was approximately 35 percent less than in the previous year. Undoubtedly some of this decline was due to loss of pay. It is reasonable to assume that the number of infections would also be lower, if the number of exposures were decreased.

In the summer of 1934, while the fleet was in Chefoo, a different system was inaugurated. The controlled district was disintegrated, a number of the houses were placed out of bounds, the patrol was no longer maintained, and no effort was made to compel prophylaxis following exposure. At the time this seemed to be possibly a precarious step, and there were some who thought that the scattering of the prostitutes would result in an immediate increase in the number of venereal admissions. This did not prove to be the case.

Monthly venereal talks were continued on all of the ships and the use of the rubber condom was recommended. Plain soap and water was also advocated. Men were urged not to place reliance on the simple application of calomel ointment. On all ships condoms were furnished to the men free or at cost. The responsibility for the contraction of venereal disease was placed on the individual and the disciplinary measures were retained.

Referring to figure 1, it will be seen that the cessation of controlled prophylaxis did not result in an increased number of admissions. During these months of 1934, July to December, emphasis was only placed on condoms and soap and water. Calomel ointment was not recommended, and the number of tubes issued by the medical departments of the various ships declined to a marked degree.

It is believed that the decline in the number of admissions is definitely due, in a large part, to changed methods of prophylaxis. Several individual instances known to be authentic are related to show the relative unreliability of methods formerly in use.

1. An enlisted man while being exposed, felt the condom tear and reported within 5 minutes to the first-aid station, where he received complete calomel ointment and protargol prophylaxis. Five days later he developed a chancroid at the site of the small abrasion received at the time of exposure.

2. One case reported to the doctor within 1 hour after being exposed and received complete prophylaxis. A week later he developed gonorrhea.

3. A number of men were seen at venereal inspections with genitals covered with calomel ointment, which they had renewed constantly

since exposure. Their names were taken, and subsequently several of them developed penile lesions.

4. Close questioning of nearly 100 patients revealed the fact that almost invariably condoms had not been used, or had been torn during exposure. Calomel ointment failed to protect them.

Such cases indicate not only the frequent failure of the usual protective measures, but in addition they reveal that actual harm may result, due to the individual's belief that he could expose himself indiscriminately, relying on these measures for protection against the inevitable contact with venereal disease exhibited by practically all of the prostitutes on the Asiatic station.

Figure depicts by graph the number of admissions in the squadron for 1932–34.

The disciplinary measures referred to earlier, defined as chronic offenders those who had had several original admissions within a period of 1 year, or one enlistment, and indicated punishments ranging from liberty restrictions to a recommendation for undesirable discharge due to unclean habits. These measures were in force during 1933 and 1934. There were 115 less admissions in 1934 than in 1933, or a reduction of approximately 25 percent. From these figures the deduction can logically be made that disciplinary measures, while contributory, were not the major reasons for the decline in the number of admissions. It is believed that education of the personnel to the use of more positive protective measures has contributed very materially to the lower rate.

A further indication of the efficacy of the recently instituted prophylactic and disciplinary measures is revealed by the figures, given for the entire Asiatic station, of the number of venereal admissions per thousand personnel for the past 3 years:

	Admissions per thousand
1932	445
1933	226
1934	168

CONCLUSIONS

1. The routine use of heretofore generally accepted prophylactic methods, i. e., calomel ointment, etc., has proven unsatisfactory on the Asiatic station.

2. The use of the rubber condom and soap and water has proven more efficacious in the prevention of venereal disease.

3. Certain disciplinary measures tend to lower the number of admissions on this station.

EACH SQUARE REPRESENTS ONE ORIGINAL ADMISSION.

Numbers indicate total cases for month and year.

Total number of original admissions for venereal disease and ports visited

	January	February	March	April	May	June
1932						
Ports visited	Manila	Shanghai	Shanghai	Shanghai	China ports	China ports, Chefoo.
Number of admissions	82	75	64	134	162	109.
1933						
Ports visited	Manila	Manila	Manila	Shanghai	Shanghai and Chefoo.	Chefoo.
Number of admissions	59	49	40	46	102	33.
1934						
Ports visited	Manila	Manila	Manila	Japan	Shanghai and Chefoo.	Chefoo.
Number of admissions	28	34	21	36	36	52.

	July	August	September	October	November	December
1932						
Ports visited	Chefoo	Chefoo	Chefoo	Shanghai [1]	Hong Kong and Manila.	Manila.
Number of admissions	77	79	79	159	59	55.
1933						
Ports visited	Chefoo	Chefoo	Chefoo	Shanghai	Hong Kong and Manila.	Manila.
Number of admissions	15	22	13	17	30	54.
1934						
Ports visited	Chefoo [2]	Chefoo	Chefoo	Shanghai	Hong Kong and Manila.	Manila.
Number of admissions	21	30	21	19	37	30.

[1] Disciplinary measures instituted.
[2] Red-light district closed.

A METHOD USED IN THE TREATMENT OF 33 CASES OF ACUTE GONOCOCCUS URETHRITIS WITHOUT SICK DAYS

By R. A. VILAR, Lieutenant, Medical Corps, United States Navy

In dealing with the prophylaxis of gonorrhea in the service, men are instructed by lectures how to prevent infection. Like with any mass of people, these words of wisdom uttered by the lecturer are profitable to some but mean nothing to others. These last ones are those who later report to the medical officer that they are suffering from some venereal disease such as gonorrhea.

The problem that confronts the medical officer is how to treat these cases suffering from acute specific urethritis without relieving them from their duties. The author has always leaned toward conservatism with regard to time of starting intra-urethral injection and their number and strength; and from his experience feels that starting the injections in the acute stage is conducive to such complications as epididymitis, posterior urethritis, and prostatitis.

Treatment of gonorrheal urethritis by rest in bed and hygienic measures during the acute stage is a well-recognized procedure, but it is hard to convince the patient that he is receiving proper attention.

It is even more difficult to retain the patient's confidence and cooperation when he is kept on duty and given no local treatment. However, this was the method adopted for these 33 cases and proved satisfactory.

Bearing in mind that the services of the men were much needed, they were treated as ambulatory cases and on a duty status. They performed all of their duties in the usual manner.

The course of treatment in each case was carried out in the following manner: The patient was called into the doctor's office and talked with at great length in order to gain his confidence. He was strongly urged not to use any local treatment until advised to do so by the physician. It is a factor of the first importance to gain the patient's confidence, otherwise he will not follow the prescribed course of treatment. He will believe that the physician is doing nothing for him, and will consult someone else who will probably do him more harm than good. The nature of his disease must be explained to him as well as the complications that may develop if he does not follow the course of treatment. If the patient fails to keep faith with the physician, he is likely to supplement the prescribed treatment with that he will obtain from other sources.

Each man was asked to return at intervals of 3 to 4 days for inspection. By doing this, the urethral discharge was kept under observation and when this discharge had thinned out and decreased considerably, that is, when the acute stage had subsided, intra-urethral medication was started.

During the time that patient was waiting for the urethral discharge to subside and thin out, he was placed on some urinary antiseptic for oral administration, such as urotropin with sodium acid phosphate, 10 grains of each, three times a day. He was advised to keep his bowels opened, to abstain from any form of intoxicating liquor, from sexual intercourse, and from highly seasoned foods. His diet consisted of bland and easily digestible food. Every patient was told that by drinking great quantities of fluids, especially water, his urinary output would be increased, thus keeping the urethra as free of pus as it was possible. Local hygiene of the penis was also enforced. A "butterfly bandage" served the purpose very well and avoided soiling of underwear. In case the patient complained of nocturnal erections and emissions, sodium bromide in 30-grain doses, three times a day, was given.

When the time to begin intra-urethral medication had arrived, that is, when the acute symptoms had subsided, the patient was instructed in the proper use of the penile syringe and in the method of injection of solutions into the urethra. First, before attempting to inject the antiseptic into the urethra, he was told to urinate and thus clear away the pus discharged from the urethral canal; in this way the danger of pushing the infection farther into the urethra was diminished.

The penile syringe was filled only one-third full. With one hand the patient caused pressure to be exerted at the base of the penis in order to obliterate the urethral lumen, and with the syringe in the other hand he injected the antiseptic very slowly and without much pressure, into the urethra. Then he released the base of the penis and pressed the glans penis around the meatus and thus retained the antiseptic in the anterior urethra. The solution was retained for from 5 to 10 minutes, then allowed to run out.

During the early part of the intra-urethral treatment, one injection a day of one-fourth of 1 percent solution of protargol was found to be sufficient, preferably in the morning immediately after arising. Later the injections were increased to two and three a day according to the amount of urethral discharge present. The lesser the amount of pus the greater the number of daily urethral injections, and vice versa.

The treatment was continued until there was a total absence of urethral discharge and repeated urethral smears had been negative for gram negative intra- and extra-cellular diplococci. Then each case was considered as "cured"; but the patients were warned to report immediately any evidence of recurrence, which in this series of cases did not occur.

The 33 uncomplicated cases mentioned required an average of 53 days to effect a cure. The greatest number of days required was 113, and the least number was 24. None of these cases had to be admitted to the sick list, and every patient continued to perform his duties on board ship.

In addition to these cases there were nine other cases which were complicated and therefore had to be put to bed. The complications and their number were: paraphymosis, two; acute epididymitis, three; and posterior urethritis, four. The reasons for these complications were: treating self before reporting to the medical officer, beginning intra-urethral injections too early, and faulty technique in the method of urethral injection. The last two in disobedience to the doctor's instructions.

These complicated cases spent a total of 657 days in the sick list, or an average of 73 days per man, during which time their services were lost.

CONCLUSIONS

1. Acute gonorrheal urethritis can be treated and cured without relieving the patient from his regular duties, and there will be less chance of complications if urethral injections are delayed until the acute symptoms have subsided.

2. It is necessary to have the patient's confidence, otherwise he is likely to disregard the doctor's instructions and warning against supplementary treatments advised by others.

POLYMASTIA, WITH SPECIAL REFERENCE TO SUPERNUMERARY AXILLARY BREASTS: BRIEF REVIEW WITH CASE REPORT

By C. F. Storey, Lieutenant, Medical Corps, United States Navy

Polymastia is one of the most interesting developmental anomalies with which physicians come in contact. That it has been known since ancient times is pointed out by Gill (*1*), and the antiquity of the condition is also emphasized by Rebeiro (*2*). In spite of early recognition by the profession, polymastia seems to have been somewhat neglected by the medical writers and information on the subject is not easily obtainable. De Lee (*3*) and other authors of standard textbooks of obstetrics discuss it only briefly. French (*4*), Cabot (*5*), and other authors of other recognized works on differential diagnosis fail to mention it. In at least some of the medical schools the subject gains scant attention.

I thoroughly agree with Gill (*1*) in his statement that "information concerning the characteristic differential diagnostic points as well as the treatment of complications arising in supernumerary mammae is surprisingly scarce in the medical literature" and, as a result, when one encounters his first case he is apt to be puzzled as to the diagnosis and uncertain in his treatment. It is for this reason, together with the fact that I desire to report a somewhat rare case of triple supernumerary breasts in the right axilla with a single supernumerary breast in the left axilla, all actively functioning during lactation, that I felt this paper to be worth while. The case reported is also of interest in that, I believe, it illustrates the upper limit of the milk-line in the human.

Definition.—Polymastia is defined as a condition in which, in the human, more than one pair of breasts are present (Stedman) (*6*). Formerly, as Klinkerfuss (*7*) has pointed out, physicians were at great pains to differentiate between true supernumerary breasts and aberrant lobes or lobules of the normal breast, and they did not include the latter group in the term polymastia. However, since the difference between the two is purely one of scientific interest, the tendency has grown to include all cases presenting mammary tissue definitely outside the normal gland under the heading of polymastia. Present usage therefore seems to dictate the inclusion not only of supernumerary breasts and aberrant lobules of the normal breasts, but also of ectopic breasts under the heading of polymastia.

Synonyms.—From the foregoing it is obvious that the following terms are accepted as synonymous with polymastia: Supernumerary breasts or mammary glands, accessory breasts or mammary glands; aberrant breasts or mammary glands, and true ectopic breasts or mammary glands.

Frequency of occurrence.—The relative frequency with which this condition is observed seems to be a matter of dispute. Gill (*1*) states

that it is rare in the human, and De Lee (*3*) and McFarland (*8*), among others, are in agreement with his opinion. Mason (*9*) refers to it as uncommon and expresses the opinion that one in every several hundred women exhibit some form of polymastia. On the contrary, Stockard (*10*), Dixon (*11*), and others speak of it as a not infrequent occurrence. Stockard (*10*) declares that it has been observed as often as from 1 to 7 percent of cases examined by different examiners. If we are to include all anomalies of the breast for consideration, such as polythelia, a developmental abnormality in which there is an accessory nipple present but only vestigial remains of the gland itself; unimastia, in which there is only one breast present, an extremely rare finding; amastia, in which no breasts are present, likewise very unusual; and true ectopic breasts, our percentage occurrence would probably run fairly high. Since breast anomalies are more often seen in males than in females, the inclusion of both sexes in a study of polymastia would boost the figures considerably. However, if we limit our cases to those *in the female* with supernumerary breasts only, much fewer cases are encountered. Going further, and including only those supernumerary breasts which assume a functioning status during lactation, and our percentage is further reduced; and finally, if we consider only functioning supernumerary breasts that are located in the axilla, we are dealing with a condition that is undeniably rare. Just how rare remains a matter of doubt. I have been unable, in a rather comprehensive though admittedly incomplete review of the literature, to find statistics that would serve to clarify this question, and until more cases are reported and a long series compiled and analyzed, it is my opinion that it is best left open to discussion. A definite familial tendency toward polymastism has long been recognized.

Embryology.—In a consideration of polymastia it is essential to briefly review the embryonic development of the mammary gland in order to make clear the developmental abnormality responsible for the polymastism.

The tubular mammary glands peculiar to mammals are accepted as modified sweat glands. They are derived from the primitive milk-line (crista lactea), a linear ectodermal thickening which extends between the upper and lower limb buds on either side of the midline. This ectodermal crest is first noted at the end of the fourth week of intra-uterine life (9 mm embryo), and early in the second month, in the future pectoral region, there is normally a thickening and down-growth of the epidermis, and the mammary area becomes flat and eventually depressed below the level of the surrounding epidermis. Later there is an evagination at this site, resulting in a papilla-like formation, the anlage of the mammary gland (Arey) (*12*).

In the lower animals, such as the dog and pig, there is a series of the papillae or "mounds" (Gill) (*1*) produced by cellular proliferation,

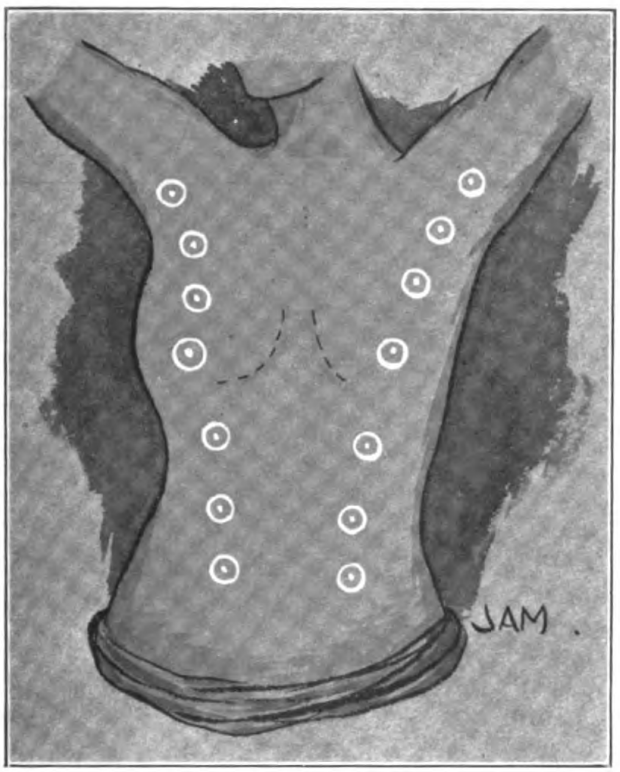
and in these animals the portions of the milk ridge between these "mounds" is absorbed, while the "mounds" go on to develop into mammary glands. A similar series of these papillae are sometimes formed in the human embryo, but ordinarily in the human any mammary points in excess of the normal two undergo developmental arrest, atrophic change, and eventually disappear. In such a case, when the retrogressive changes just mentioned fail to take place, growth and development continue to greater or lesser extent, finally resulting in the presence in the adult of accessory mammae varying from mere vestigial remains to glands approaching the normal in every respect.

Later in the second embryonic month buds appear from the epithelial anlage, elongate, and form solid cords, varying in number from 15 to 20, which in the third month invade the underlying mesodermal tissue, which has been compressed by the downgrowth of the epidermis, and which, with its elements arranged in concentric layers, forms the forerunner of the gland stroma. The down-growing cords are the precursors of the milk ducts (lactiferous tubules) and their tributaries, and by dividing and branching give rise to the future lobes and lobules of the fully developed mammae, while, as previously pointed out, the mesodermal tissues which have been invaded by the downgrowth of the cellular projections later forms the supporting connective tissue framework of the mature gland. These cordlike cellular projections form a lumen only at birth, at which time they not infrequently secrete a milk-like material. This secretion is said to be noticed more frequently in male than in female infants.

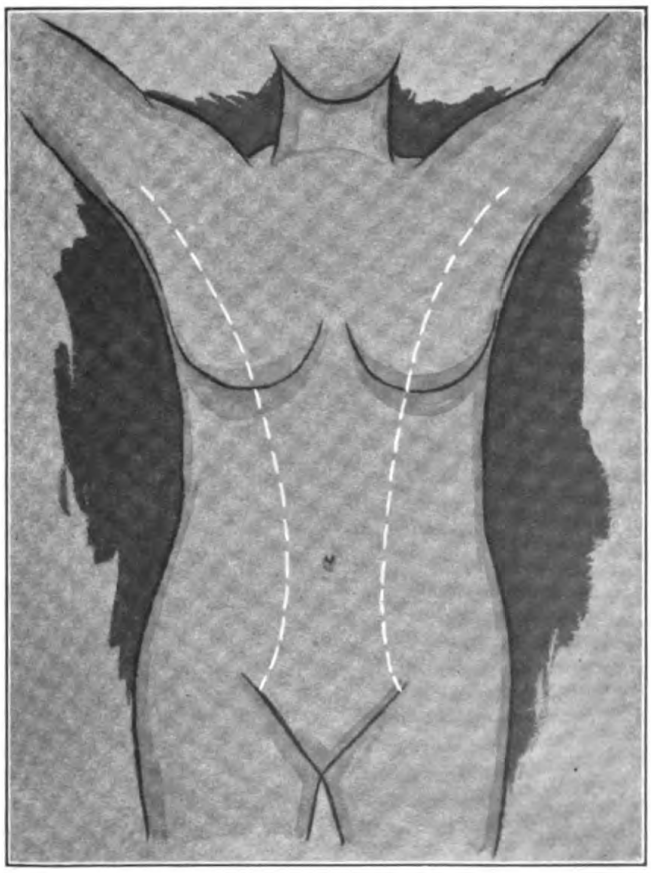
The beginning formation of the nipple is first evidenced in the sixth embryonic month by an elevation at the apex of the mammary area, and from the cellular projections constituting the anlage of the lactiferous tubules, bulbous-ended processes have branched out and formed the forerunners of the true glandular tissue. Somewhat later the epidermis in the area where the galactophorous ducts open on the surface is evaginated about the mammary points marking their surface terminations, enveloping them in its substance and forming the nipples.

It is clearly indicated that two conditions must be fulfilled before polymastism will be observed in the adult: namely, the papilla-like formations which constitute the anlage of the mammary glands must be present in the embryonic milk line in numbers exceeding the normal two; and, secondly, retrograde metamorphosis must fail to take place in these excessive formations, allowing them to grow to more or less maturity.

Since arrest in development may occur at any stage of intra-uterine life, we may expect to find varying degrees of development in the supernumerary breasts detected in adults, and this expectation is



MAMMARY GLAND GROUPING ALONG THE MILK LINE.



THE EMBRYOLOGICAL MILK LINE.

borne out by clinical observations. The following general classification of true supernumerary breasts as a whole is proposed:

I. Small accessory breasts which are never functionally active have no nipple or dilated pore as an outlet, and no areola.

II. Accessory breasts, usually somewhat larger and more definitely defined than the above, which have no nipple or dilated pore, and no areola, but which are functionally active during lactation.

III. Accessory breasts similar to those in group II but ordinarily showing better general development, being functionally active at the time of lactation, and exhibiting at least a rudimentary nipple or dilated pore through which milk is excreted or can be expressed, but which have no areola.

IV. Accessory breasts approaching the normal type of mammary gland in their development, possessing a well-developed nipple, a distinct areola, and lactating freely during the nursing period.

It should be remembered that this proposed grouping is intended merely to divide the types of supernumerary breasts encountered into rough general classes and that all gradations between the various groups may be discovered. It is felt, however, that some such classification may be advantageously used as a guide by those who may in the future desire to report cases of supernumerary breasts, and that its use will facilitate the gathering of statistics on this interesting condition.

Location.—If we bear in mind the embryological development of the mammary gland it becomes evident that we may expect to discover supernumerary breasts from time to time at any particular point along the course of the milk line in the adult (see fig. 2); that is, on either of the midline on the ventral surface of the body from the axillary to the inguinal region, and as a matter of fact, that is exactly what we do find. True ectopic breasts, of course, may occur at almost any location on the body, but it is not the purpose of this paper to include a detailed discussion of that condition.

In general it may be said that more accessory mammae are noted on the left side of the body than on the right, and that a great many more are observed below the normal breasts than above them. Those superior to the true mammae are usually also lateral to them, whereas those below the normal mammary glands are usually mesial to them. A pronounced majority of all supernumerary mammary glands are situated in the pectoral region. Laloy (*13*) gives the following percentage distribution for the various areas:

	Percent
1. In the thoracic region of the mammary crest above the normal breast	4. 1
2. In the thoracic of the mammary crest below the normal breast	92. 9
3. In the abdominal portion of the mammary crest	3. 0

Considering not only supernumerary breasts but erratic or ectopic breasts as well, the following figures obtain (Laloy) (*13*):

	Percent
1. On the anterior thoracic wall	91.8
2. In the axilla	4.6
3. On the back	1.8
4. Over the acromium process	.9

The location of the remaining 0.9 percent is not stated, but it is assumed that they are scattered over various and sundry eccentric sites on different parts of the body. According to Graves (*14*) there are four cases in the literature of supernumerary or ectopic breasts in the vulva, and McFarlane (*15*) has reported one such case.

Numerical distribution.—Since polymastia in the final analysis is believed to represent a reversion to the ancestral condition (Dixon) (*11*), in which more than two mammary glands were normal, and in which many young were probably produced at each birth, it is not hard to vizualize the production of many pairs of accessory breasts, assuming that the reversion was more or less complete. In a consideration of the question of reversion, it is interesting to note that accessory breasts in the human are usually observed in the positions normally occupied by mammae in the lower animals (pig, dog, cat, etc.) (see fig. 3). In these lower animals, the embryonic crista lactea converge as they travel posteriorly, almost meeting in the midline at their posterior termination. Conversely they tend to diverge in the cephalad direction. In this connection the following interesting points are brought to mind: (1) Supernumerary breasts in the human are seen much more often below than above the normal breasts (the lower animals have no breasts cephalad to the anterior extremities); (2) those occurring above the normal breast in man are ordinarily lateral to the normal breast, whereas those inferior to the normal breast are also usually medial to it. These thoughts lead us to ponder as to whether the aberrant breasts located in the labium majora referred to by Graves (*14*), McFarlane (*15*), Bell (*15*), Rebeiro (*2*), and others are ectopic mammae or whether they are supernumerary mammae representing a reversion to the type in which the crista lactea almost met in the midline in the caudad direction.

In line with what might be expected from the above considerations, supernumerary mammae of considerable multiplicity have been reported from time to time. Dixon (*11*) mentions the finding of three extra pairs, and states that cases in which they were probably more numerous than this have been recorded. Neugebauer (*17*) reported a patient with 10 breasts, all functionally active.

Hauseman (*18*) gives the following percentage occurrence of the various numbers of supernumerary breasts:

	Percent
Single	63. 5
Double	32. 7
Triple	2. 7
Quadruple	. 8

Though he makes no specific reference to the occurrence of more than four accessory breasts, it is assumed that the remaining three-tenths of 1 percent includes all cases presenting more than that number.

Supernumerary axillary mammary glands.—In their excellent monograph upon "The Breast: Its Anomalies, Its Diseases, and Their Treatment", Deaver and McFarland (*19*) proposed the following classification of axillary mammary tissue:

I. Cases in which lumps of painful character appear in the axilla during pregnancy or at the beginning of lactation, either where small painless lumps have existed since puberty or where the patient was unaware of the existence of any abnormality.

II. Cases in which "axillary lumps" similar to those mentioned, occur under like conditions, but discharge a milky secretion through openings of varying size and number.

III. Cases in which "axillary lumps" are surmounted by a more or less distinct areola with one or more rudimentary nipples.

IV. Cases in which there is a distinct mammary gland in the axilla, upon which there is a distinct areola, and a fairly well-formed nipple.

This classification serves as a useful basis for studying supernumerary axillary breasts, being especially helpful in grouping the cases reported in the literature.

Signs and symptoms.—Except as they are altered by their peculiar location, axillary supernumerary mammary glands give rise to physical signs and symptoms similar to those occasioned by these anomalies when situated elsewhere. They are rarely noted prior to puberty. If there is a nipple present the patient usually thinks of it as a mole and pays no great attention to it. Ordinarily they do not give rise to symptoms until after the patient becomes pregnant, but Anderson (*20*), McFarland (*8*), and others have reported cases in which the patient first became aware of the abnormality at the time of the beginning of the menses, noting an axillary fullness or distinct swelling, at times mildly painful, occurring in association with the monthly periods. Symptoms at this time are very rarely of a disturbing nature, consisting in most cases merely of the recognition of the mass.

In the typical case, as described by Anderson (*20*), Klinkerfuss (*7*), Pankow (*21*), Hindbaugh (*22*), Harner (*23*), and numerous others, the patient first becomes aware of a mass or swelling in the axilla in about the fourth month of pregnancy (although a tumor of slowly

increasing size may be detected by a patient previously unaware of its existence as early as the second month (Klinkerfuss) (*7*)), or not until after delivery (Gill, et al.) (*1*). The mass may or may not be painful when first observed. When seen early in pregnancy and kept under observation the supernumerary breasts are seen to undergo a slow progressive enlargement as gestation proceeds, maintaining a comparative pace with the normal breast in this respect. However, as is likewise true of the normal mammae, accessory breasts undergo the major part of their enlargement post-partum coincident with the establishment of lactation. It is at this time that the patient usually begins to complain of pain in the gland, and not infrequently this is the first time that its existence is called to the attention of the physician. The pain is described as similar to that felt in an overdistended normal breast. In the majority of cases it is relieved by allowing the infant to nurse, either by simultaneous spontaneous "leakage" through a nipple or dilated pore of the accessory breast, or by the actual emptying of it through an elongated duct connecting with the normal breast. When the accessory breasts can be so emptied, the patient suffers only from the temporary discomfort of these breasts when full, and from the inconvenience caused by the wetting and soiling of the bedding or clothing when they empty. If the proper precautions are not taken this leakage is apt to result in excoriations of the skin which is at times a source of no little trouble.

The physical signs consist simply of a round or ovoid mass of variable size, located at almost any point in the axilla, ordinarily near the lateral border of the pectoralis major muscle. The tumor is intimately connected with the overlying skin, but free from attachment to the underlying structures. It is more or less freely movable within reasonable limits, tender when full of milky secretion, and is perhaps surmounted by a nipple, either with or without an areola, or presenting instead one or more dilated pores through which milk escapes, or in other cases, no opening at all. A cordlike connection with the normal breast may be visible or palpable. The mass imparts to the palpating hand a sensation similar to, or identical with, that of the normal mammary gland. As the infant nurses milk is seen to escape from those accessory breasts which possess a surface opening or openings, and at other times milk can be expressed from them by gentle pressure.

The normal breasts are usually larger or more engorged than those ordinarily seen in patients not possessing supernumerary breasts, a finding commented upon by Wu (*24*) and others.

Careful examination may reveal other developmental defects in the patient, a point which has been repeatedly emphasized. It is not extraordinary to observe masculine characteristics in these cases.

Lactation may continue in the supernumerary breasts for a period of time similar to that of the normal breast or it may cease somewhat earlier. It appears from a review of the literature that in the average case lactation in the supernumerary breasts ceases in about 3 or 4 weeks time, in spite of its continuing unimpaired in the normal mammary glands. This phenomenon can probably best be explained on the basis of disuse.

Histological considerations.—Since lactiferous glands are looked upon as exaggerated and specialized homologues of the sudoriparous glands, it is not surprising to find them similar histologically. Each sudoriparous gland seems to consist of a single tubule, perhaps in the larger glands giving off one branch, while a mammary gland consists of a highly complex system of branching tubules. Each gland is composed of a secretory portion and a conducting portion, with the structure varying with the function. The theoretical fundamental structure of the two glands are the same, consisting of a membrana propria surmounted by two layers of cells, the outer less well-defined layer, known as "basket" cells and regarded as ectodermal muscle cells, and the definite inner layer of large cuboidal cells, referred to as the "chief" or secretory cells.

As the ever-increasing quantity of secretion in the lactiferous glands requires larger and larger tubules to accommodate it, we find the tubules in this gland uniting as they approach the nipple, eventuating in large columnar epithelium-lined ducts and ending in the ampulla.

Since the sudoriparous glands receive no tributaries, they maintain a more or less constant diameter throughout, except that they are widely dilated during active secretion. In contradistinction to the lactiferous glands, when the secretory portion of the sudoriparous glands leave off and the conducting part begins, instead of expanding, the tubule diminishes to approximately one-half its former diameter, loses its muscle cells, and develops a double or triple cell layer with a homogeneous zone or cuticle about the lumen. Since the sweat glands, while not constantly active, must always be ready to assume their function, their structure is more or less constant and does not change from time to time. The lactiferous glands, on the other hand, are only rarely called upon to exercise their function, and for this they must make elaborate preparations. In these glands, therefore, a marked structural variation is seen, depending on whether they are in the stage of resting or inactivity, or in the process of involution or evolution.

Prior to the onset of menstruation, little change is seen in the lactiferous glands, but with the onset of this phenomenon, probably as a result of hormonal stimulation, arising in the ovary and derived from the liquor folliculi or secreted by the corpora lutea (Rosenberg) (*25*), remarkable changes take place, previously quiescent tubules extending and branching until definite mammary lobules are found.

If pregnancy does not supervene, the useless glandular tissue undergoes atrophic change and the gland returns to its resting stage.

Applying this knowledge to those rare cases in which lactiferous tissue is found in the axilla, the clinical phenomena which take place is made clear. Prior to the advent of the menses the patient is unaware of any anomaly. At this time she may or may not note an axillary fullness at the time of her monthly periods. Whereas the gland has previously been in the resting stage, it is now in the stage of evolution preparing to exercise its function following the anticipated impregnation. Should impregnation fail to take place, through the influence of the hormone by which it is controlled, the gland goes through an involutionary stage, resulting clinically in the disappearence of the axillary fullness. However, should the patient become pregnant, the stage of evolution or preparation continues, eventually resulting ln the assumption of a functioning status by this anomalous lactiferous tissue. When lactation sets in, these accessory breasts become engorged with milk, and it is at this time that the annoying symptoms make their appearance. Histologically, the tissues in supernumerary breasts at this time differ in no way from those of the normal breast.

Diagnosis.—If supernumerary breasts are borne in mind when one encounters an axillary tumor which first gives rise to symptoms at the beginning of lactation, the diagnosis is usually not difficult. In atypical cases, particularly those presenting no opening through milk escapes, it is not so easy to arrive at a definite conclusion. In such cases biopsy which microscopic examination of sections of the tissue removed will result in clearing the question up. Following are the usual diagnostic criteria:

1. Axillary tumor, increasing in size at pregnancy and at each pregnancy.
2. Marked increase in the size of the tumor during lactation.
3. Immediate relief after suckling infant.
4. Ability to express milk from the masses.
5. Subsidence upon being treated as engorged breasts.
6. History of similar condition in other members of the family.
7. Unusually marked engorgement of the normal breasts.
8. Discovery of an areola and nipple, nipple alone, enlarged pore or pores, or palpation of a cord-like connection with the normal breast.
9. History of swelling of the masses at the menstrual periods.
10. Spontaneous subsidence in about 3 weeks following the establishment of lactation.
11. Biopsy, with the finding of the characteristic histological structure and a collection of milk.

12. Gland puncture resulting in the withdrawal of a fluid chemically and microscopically indentical with milk. (This procedure is indicated, of course, only in those supernumerary mammae which possess no outlet through which milk escapes to the surface. Although I have seen no reference in the literature to this means of diagnosis, I have used it successfully myself.

Differential diagnosis.—Any condition that will give rise to a tumor mass in the axilla must at least be considered in the differential diagnosis of axillary supernumerary breasts. The subject cannot be considered unimportant. Errors may lead to improper treatment, resulting in "discomfort for both the patient and the physician", as Klinkerfuss (*7*) has so aptly put it in citing a case incorrectly diagnosed as acute adentitis and treated by incision. On the other hand, when correctly diagnosed, the patient is considerably relieved to learn that she suffers from nothing more serious.

Among the more prominent conditions to be considered in the differential diagnosis, the following are included:

1. Hypertrophied sudoriparous and sebaceous glands.
2. Lipomata.
3. Acute axillary lymphadenitis.
4. Chronic axillary lymphadenopathy (lues, tuberculosis, Hodgkins disease, lymphosarcoma, etc.).
5. Metastatic carcinoma.
6. Aberrant lobule of a normal breast.

It is not my intention to describe fully the differential diagnosis of these more or less well-recognized conditions, but rather to merely call attention to them. As De Lee (*3*) has pointed out, hypertrophy of the sudoriparous glands in the axilla at the time of lactation is not unusual, and this condition is not always easily ruled out. However, the history of the case, the size and feel of the mass, the relief following nursing the infant, and the discovery of a nipple or dilated pore will usually make the diagnosis clear. In other cases the obtainance and examination of the milky secretion may be necessary, or finally, biospy.

The other condition apt to cause difficulty is an aberrant lobule of a normal breast. This subject can be disposed of by the statement that the differential diagnosis here is of no practical importance, but merely one of scientific interest, as the histology, anatomy, function, and symptomatology of the two are identically similar, and the treatment of both is the same.

Treatment.—In this country the principle of conservative treatment in supernumerary axillary breasts is almost unanimously endorsed, and surgical intervention is only rarely considered necessary. In the usual case relief is obtained by allowing the infant to nurse. The modus operandi by which this results is either through spontaneous

leakage of the accessory breast coincident with nursing the normal breast, or via actual drainage of the anomalous organ through a duct connecting with the normal breast. Where leakage occurs some absorbent material must be placed over the point where the fluid escapes, and the surrounding skin dried thoroughly and covered with a dusting powder after nursing is completed to prevent excoriations of the skin. It is at time advisable to cover the overlying skin with petrolatum before putting the baby to breast.

In uncomplicated cases the only direct treatment indicated is the application of an axillary pad and binder. An ice bag is sometimes beneficial where there is an excessive secretion of milk and resultant engorgement. Fluids may be restricted for a few days as these mothers usually produce an overabundance of milk. In rare cases the temporary use of sedatives may be necessary.

Under the above treatment the supernumerary breasts usually cease lactating in 3 or 4 weeks' time, and those which do not do so ordinarily become painless and are considered a noxious feature only because of the soiling of the mother's clothing when the baby nurses.

The most difficult cases are those in which there is no patent duct connecting with the normal breast, and no nipple or dilated pore to serve as an outlet for the secreted milk. Ordinarily these breasts will soon subside under palliative treatment as outlined above, but if the pain is too great to be borne without too much discomfort, or if the glands do not subside in a reasonable length of time, excision is advisable.

Complications such as abscess, galactocele, etc., are treated in the same manner as when they occur in the normal breast.

Patients almost invariably inquire as to the predisposition of these tumors to cancer. On this question there are definitely two schools of thought. In general, it may be said that most authorities in this country are of the opinion that carcinomatous change in supernumerary breasts occurs rarely, if at all. Miller (*23*) states that he found no report in the literature of malignant change in accessory mammae. Mason (*9*) states that it is not to be feared. Klinkerfuss (*5*) does not look upon neoplastic degeneration in these anomalies as even a remote possibility, and other American writers seem to hold to the same view. This accounts for the tendency to the most conservative of treatment which is advised in this country.

On the other hand, generally speaking, foreign authors take a radically different attitude of these anomalies, viewing them with more or less alarm. Rebeiro (*2*) made a very complete study of the subject in 1931 with a thorough review of the literature, and he found numerous references to the tendency of the tumors to undergo malignant change. He quotes from the work of Williams (*26*), Goepel (*27*), Bizar (*28*), Martin (*29*), Cameron (*30*), des Barres (*31*), Morestin

(*32*), Portier (*33*), Pauvie and Porter (*34*), Foerster (*35*), and others, all of whom referred to the occurrence of neoplasms in supernumerary mammae. Bizar (*28*) collected some 70-odd such cases. Williams (*26*) reported an incidence of malignant new growths in these tumors of 14 percent.

According to these authors the malignant degeneration usually assumes the form of adeno-lipo-, adeno-fibro-lipo-, or other varieties of adenomatous carcinoma. It should be noted, however, that it appears that malignancy occurs in a proportionately larger number of cases of true ectopic mammae, than is seen in the case of supernumerary breasts.

Bizar (*28*) is unequivocal in his statement that there is a definite tendency of these tumors to cancer and Rebeiro's (*2*) study leads him to the same conclusion. Their views may be said to be fairly representative of the foreign school of thought.

As a result of their fear of malignant change and their belief that it is a probable development in supernumerary mammae, the profession abroad leans toward radical treatment. Bizar (*28*) states unreservedly that prompt and complete extirpation is indicated, and Rebeiro (*2*) and others in South America, Japan, and on the continent agree that surgery is the treatment of choice. The English are an exception, most authorities in that country recommending conservatism.

CASE REPORT

Mrs. J. C. C., para iii, first seen in the second month of third pregnancy. Two previous pregnancies ran a normal course with delivery at term of a living child on each occasion. Neither child showed any developmental abnormalities. The two children, aged 6 years and 18 months, respectively, are living and well. Two days after delivery of her first child patient became aware of a large mass in her left axilla and three somewhat smaller masses in the right axilla. On the third day of the puerperium these masses became tense and painful. They were relieved when the infant nursed. They were brought to the attention of the attending physician who examined them and made a diagnosis of *axillary varicose veins*(*!*). No treatment was prescribed. The pain and swelling remained present but showed a gradual progressive diminution from day to day, until the pain was no longer present at the end of three weeks. The swelling was noticable until the end of the fifth month. There was no leakage from the masses.

The history of the second pregnancy was identical except that the patient first noted the slowly enlarging masses at the beginning of the seventh month of gestation, but as before the major portion of the enlargement was coincident with the establishment of lactation. She was under a different physician on this occasion, who made a diagnosis of acute axillary lymphadenitis. Incision was considered, but the patient refused operation because of her knowledge and belief that the acute symptoms would subside as they had on a previous occasion, and that is exactly what happened.

There were no remarkable physical findings when I first examined the patient other than those associated with pregnancy of 3 months' duration. Because of the history the axillae were subject to close scrutiny but no nipple was in evidence. Palpation revealed no abnormal mass in either axilla. The patient was seen at

monthly intervals, through the seventh month of her pregnancy and bimonthly during the last 2 months. A slight fullness was noted in each axillae at the sixth month. By the seventh month a definite palpable tumor mass was present in the left axillae and three such tumors in the right axillae. The three masses in the right axilla were arranged in a line, extending from below upward and laterally, with the first mass naturally superior and lateral to the normal breast, the second tumor above and lateral to the first, and the third situated superiolaterally in reference to the second. All three of them were definitely in the axilla, the most medial and inferior one being posterior and lateral to the lateral margin of the pectoralis major muscle. They were not painful and underwent a slow progressive enlargement as gestation progressed. At the time the patient was admitted to the hospital in labor the mass in the left axilla was about the size of a medium sized lemon, while the larger of those in the right axilla was the size of a small lemon, the second of the masses on this side about as large as a small lime, and the third or smallest one was about the size of a small English walnut. The masses were slightly tender at the time of admission. There were no remarkable physical finding at this time except that of a patient at term in labor, other than the fact that the patient's normal breasts were enlarged far beyond that usually observed in a patient of her type. The right breast was larger than the left. Delivery was uneventful.

On the second day of the puerperium the axillary masses underwent a rapid and startling enlargement and became very tense and exquisitely painful. The breasts became engorged at the same time. The pain was so severe that large doses of codeine had to be given for relief. The pain was relieved when the baby was put to breast. The masses appeared to decrease slightly in size after the baby nursed, though the diminution was not marked. There was no spontaneous leakage from these tumors, but a small dilated pore was observed over the eminence of each mass, from which a fluid grossly and microscopically identical with the breast milk could be expressed by moderate pressure on the masses. No sign of acute inflammation was present in the tumors. Repeated examination failed to reveal any connection between the tumors on either side and the normal breasts. (See figs. 4, 5, and 6.)

The treatment prescribed was conservative. An axillary pad with binder was applied, with cold applications, and codeine by mouth, when the pain became too severe to be borne comfortably. By the tenth day the masses showed a slight decrease in size and were only slightly painful when the breasts were engorged. The patient was discharged at this time and experienced no difficulty requiring treatment after leaving the hospital.

She was seen again 6 weeks later. At this time the masses were approximately one-half as large as they were at the beginning of lactation but were no longer painful or tender. It was still possible to express milk from the dilated pores of each accessory breast with little difficulty. The normal breasts were functionally active.

The patient was unable to say whether her mother or any other member of her family had had a similar anomaly.

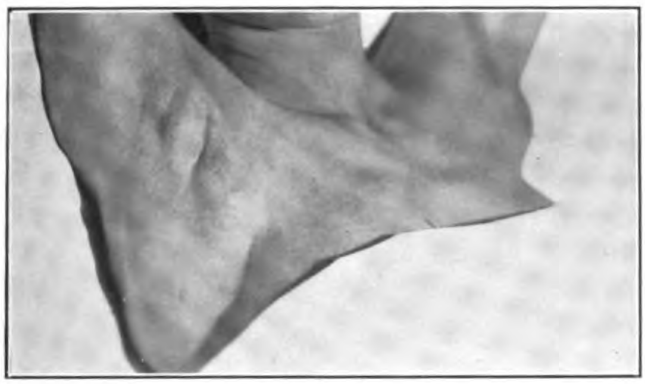
The accessory breasts were measured on the fourth post-partum day and the following measurements obtained:

In the left axilla: 5.5 cm in the long diameter, 4 cm in the short diameter, and approximately 3.5 cm in height.

In the right axilla: Lower mass, 5 by 3 by 2 cm; middle mass, 4.5 by 3 by 2.5 cm; upper mass, 3 by 1.75 by 1 cm.

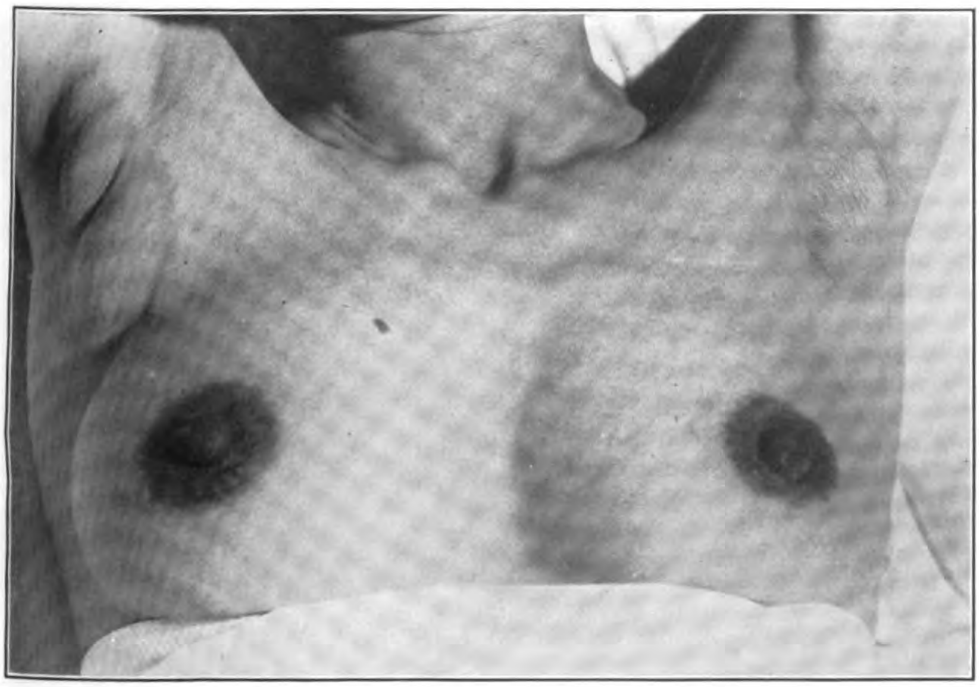
It is believed that this is a true case of accessory breasts, and the following findings support this diagnosis:

1. Axillary tumors increasing in size at pregnancy and at each of three pregnancies.



FRONT VIEW OF THE THREE ACCESSORY BREASTS IN THE RIGHT AXILLA.

Note the grooves between them. They are not connected with each other or with the normal breast. The silhouette of the tumor in the left axilla illustrates the height of these masses quite well.



FRONT VIEW OF PATIENT BOTH NORMAL AND ACCESSORY BREASTS.

Light shining directly on the left axilla prevented the supernumerary breast in this location, which in reality was much larger than the largest one on the right, from showing up well. Note engorgement of the normal breasts, and also that the right breast is considerably larger than the left.

2. Marked and rapid increase in size with the establishment of lactation.

3. Immediate relief and decrease in the size of the tumor after suckling the infant.

4. The ability to express milk through a dilated pore discovered at the apex of each of the four masses.

5. The subsidence of the symptoms following the treatment as engorged breasts.

6. Very marked engorgement of the normal breasts.

7. Progressive decrease in the size of the masses after delivery.

It is believed that this case is extremely rare. According to the best figures obtainable quadruple supernumerary breasts in the axilla occur approximately once in 656,000,000 patients. In most such cases there are two such breasts in each axilla. I have found no reference in the literature to a case presenting three accessory breasts in one axilla and only one in the other.

This case would fall in class II of the classification of Deaver and McFarland (*19*) and class III of the classification which I have proposed. The case probably illustrates the very upper limits of the milk line in the adult.

SUMMARY

1. Polymastia is discussed, with particular reference to supernumerary axillary mammary glands.

2. A classification of accessory breasts is proposed.

3. The importance of the recognition of supernumerary breasts in the axilla is emphasized.

4. The difference of opinion as to the question of malignant change in these tumors between American and foreign physicians is pointed out.

5. A case presenting four well-defined functionally active accessory breasts in the axillae is reported.

6. Gland puncture as a diagnostic aid is suggested.

CONCLUSIONS

1. It appears that accessory breasts as a whole, while not rare, are not commonly encountered, although just how frequently they occur is still a matter of doubt.

2. Supernumerary breasts occur less often in the axilla than elsewhere along the milk line.

3. The diagnosis of the condition is not difficult if it is kept in mind when encountering an axillary tumor which makes its appearance at the beginning of lactation.

4. Carcinomatous degeneration probably occurs more frequently than most authors in this country seem to believe.

61137—36——7

SHORT-WAVE RADIOTHERAPY IN VINCENT'S INFECTION

By C. E. ALLEN, Lieutenant, Dental Corps, United States Navy

There have been numerous recognized therapeutic agents for the treatment of acute inflammatory conditions in the oral cavity; there have also been many failures, especially in the treatment of the ulcerated type of gingivitis, Vincent's infection and periodontitis. Most of the failures are due to the use of caustic drugs which cause great tissue destruction of the gingivae, interproximal and peridental tissues, truly the main support and upon whose health the life of the tooth depends.

For the treatment of Vincent's infection or any other form of suppuration the first factor is to increase tissue resistance. It cannot be stated that any therapy is specific for Vincent's infection and can meet all demands for a cure, however caustic drugs head the list for treatment but cause great damage to the oral mucosa. The reason no specific therapy exists is that we know no specific etiology. Numerous drugs may penetrate the mucosa and kill the fusiform bacillus and Vincent's spirochetes that have entered the tissues, still no one has proven these bacteria cause this disease.

Plaut and Vincent found that spirochetes and fusiform bacilli are found in symbiosis in the ulceromembranous disease of the mucosa. The mere finding of the bacteria from a smear does not help in establishing the etiology, diagnosis, treatment, or prognosis of the disease. On the inflamed surface of any mucosa these bacteria may be found. In arsenical, radium, mercurial, and phosphoric stomatitis, and in scorbutic diseases of the oral cavity these bacteria are also found in abundance. Rarely is a smear made in inflammatory conditions of the mouth for Staphylococci or Streptococci or many other bacteria which cause tissue necrosis and which perhaps are the originators of this disease. This condition would afford a favorable medium for the Vincent's spirochete and fusiform bacillus to grow in, and if they are of significantly low virulence they will produce no pathogenicity.

Vincent's infection is a serious disease which may be transmitted by contact and the common use of eating utensils. It may start as a slight trauma by too vigorous brushing of the teeth in the presence of infection.

Tissue resistance may be lowered by systemic and local causes. Under the first can be mentioned general debility, tuberculosis, diphtheria, diabetes, avitaminosis, gastro-intestinal disorders, diseases of the blood, and metallic poisoning. Among the local predisposing causes are the presence of calculus, partially unerupted and carious teeth, the excessive use of alcohol, tobacco, and spicy foods.

At the onset of the infection one or more areas of inflammation may be noted along the gums, on the soft palate, the pillars of the

tonsils or on the tonsils. As the disease progresses the areas of inflammation become covered with deep craterlike ulcers having a punctate appearance, covered with a dirty grayish membrane which is not firmly adherent and bleeds freely on removal. If this condition is present, there is usually a foul odor to the breath. The patient complains of pain around the teeth rarely increased on swallowing and which does not radiate. The temperature seldom rises above 100° F. Lymphatitis of the gums exists as the disease progresses eventually producing a cervical adenitis.

The drugs which have been advocated for treatment are many. The scientific rational of treatment are classified by three methods:

1. The topical use of oxygen liberating compounds in order to inhibit the growth of anaerobic organisms.
2. Intravenous and topical use of spirocheticides.
3. The topical use of caustic drugs.

In my opinion the therapeutic value of oxidizing agents such as chromic acid, hydrogen peroxide, and sodium perborate is overrated. Chromic acid is an oxidizing agent and a caustic which precipitates albumin. The failure of these drugs may be due to the fact that the micro-organisms causing the disease change their oxygen tension. Application of chromic acid and other caustic drugs must in some measure cause necrosis of the tissue which in turn is an excellent pabulum for the growth of bacteria. Hirschfield has reported several cases of "chemical burns" from the improper use of sodium perborate. Buckley states that organisms may find entrance to any tissue or organ, but unless they grow, multiply, and produce some deleterious action on the host their presence is without special significance. Intravenous injections of arsphenamine or bismuth are not specific, for syphilitics being treated by these drugs often develop Vincent's infection.

Since 1914, the beginning of the World War, there has been a gradual increase in the number and virulence of cases, and 40 to 60 percent of the cases of today are chronic. The transferring from man to man may be increasing the virulence of these organisms. This proves the inefficiency of drugs and some form of treatment other than drug therapy must be resorted to.

Treatment as used at this dental clinic is as follows:

1. The first treatment of the patient consists of removing the local irritants, such as calculus and debris, from the teeth and gum tissue.

2. Through the courtesy of the "outpatient clinic", which is equipped with a short-wave radio emitter, we have been able to use this radiotherapy in conjunction with our other treatment. The patient is given a short-wave radio diathermy treatment of 15 minutes duration and advised not to use tobacco, alcohol, spices, or any irritants. A diet of milk, beef broth, eggs, fish, raw and cooked

leafy vegetables, and 8 ounces of orange juice daily is prescribed. A mild saline cathartic is ordered. Tooth brush and water, no dentrifice, to cleanse the teeth after each meal is advised. Eating utensils must be isolated and sterilized after each meal. The tooth brushes are brought to the office each day for sterilization, and warning against kissing or spreading the disease in other ways is given.

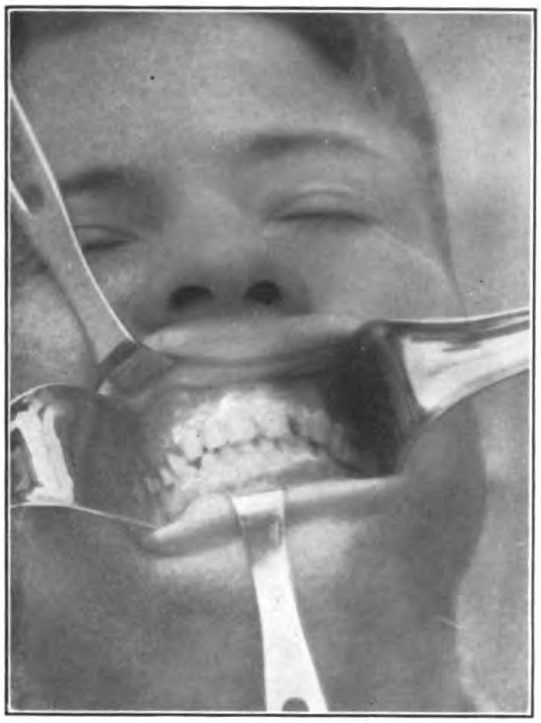
The patient is given two short-wave radio treatments daily over a period of 1 to 3 weeks, depending on the initial progress of the disease. About 12 hours after the first treatment the patient is comfortable and free from pain in the gums and cervical lymph glands. After the second treatment corrective measures such as filling carious teeth, removing overhanging margins on fillings, balancing occlusion and a thorough scaling and polishing of the teeth can be accomplished. Within a few days the marginal slough has disappeared from the gums and bleeding has ceased, and in a week the patient is greatly improved and well on the way to recovery.

In the six cases that have been treated at this clinic with short-wave diathermy, none will require treatment for peridontoclasia.

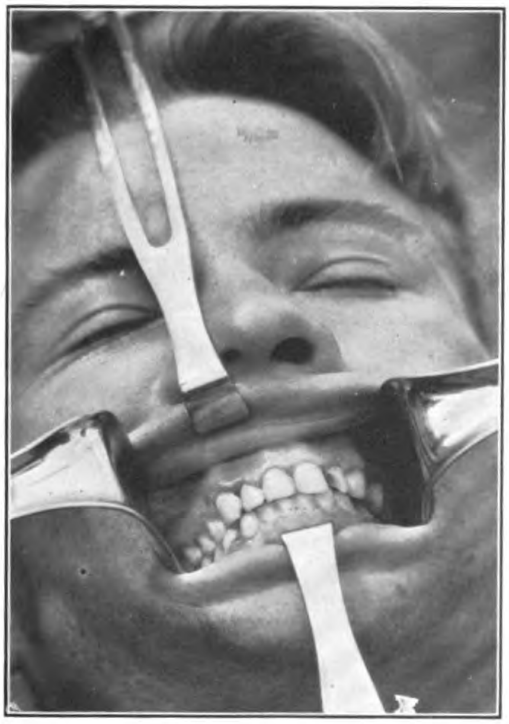
It is alleged the therapeutic radio short wave is a stimulant, dilator, anodyne, and bactericide; a stimulant, for it stimulates the blood flow to the exposed area. As the blood is drawn into the area, the capillaries gradually restore the blood flow to normal, carry off the congested blood from the infected area and thus relieve the pressure of the congestion on the nerve filaments. By the return of the blood flow to normal the pressure is removed and pain subsides.

Physicists and physiologists theoretically believe this ray produces a deep ultra microscopic massage of great tissue depth. Nolan assumes the tissue under treatment undoubtedly retains the internal heat produced by this radio short wave and develops radio fever, or an artificial fever. They contend that a temperature of 106° F. is easily developed in the tissues, which is sufficient to destroy or incapacitate bacteria. Tissues will safely stand this temperature, as it requires about 120° F. to destroy tissue cells. Where pus is confined, drainage should be instituted before treatment begins as the pus might be scattered through the tissues and cause pyemia.

Since frequency and wave length are in reciprocal relationship, the expression "short wave" is synonymous with high frequency. Since the length of the wave sent off from an electrode is dependent to an extent upon the medium through which it must pass, theoretically fat, bone, pus, and soft tissues will cause friction or resistance to the passage of the waves, and resistance means heat. The resistance of the waves' entrance into the tissue sets up a cellular frictional activity thereby producing a high temperature. The wave prevents accumulation of the blood and lymph from the vessels into the tissue, breaks up the exudations and hastens their absorption. The greatest factor



BEFORE TREATMENT.



AFTER TREATMENT.

in all of this treatment as mentioned early in this article is the saving and returning to normal of this very important tooth supporting tissue without great destruction by the use of caustic drugs and oxidizing agents. In the final analysis the treatment of any infection resolves itself into a matter of effecting immunity against invading organisms.

Considering the results obtained, would not the short-wave radiotherapy machine be a valuable addition to our armamentarium in the treatment of Vincent's infection?

REFERENCES

Clinical Incidents: Diagnosis and Treatment; J. A. D. A., 21: 768, May 1934.

Codel, Martin: Radio and Its Future; Harper's, 1930.

Taylor, A. Hoyt: Short Waves, 1930; pp. 275–284.

Donson, S. D.: Vincent's Infection Contracted During Luetic Treatment; September 1933; Dental Cosmos.

Grossman, Louis I: Results of Diathermy Treatment of Periapically Infected Teeth; September 1933; Dental Cosmos.

CLINICAL NOTES

A CASE OF PERIPHERAL NEURITIS DUE TO LEAD

By W. H. Funk, Lieutenant Commander, Medical Corps, United States Navy

A case of peripheral neuritis due to lead is reported which was diagnosed by biospectrometric examination of a skin biopsy when other routine tests for lead were negative. Patient was demineralized by the metal free diet and therapy as outlined above. At the conclusion of this treatment, there was no lead retention by skin biopsy and there had been a remarkable improvement in the patients condition.

S. H. K., chief printer, F. N. R., entered the United States Naval Hospital, Great Lakes, Ill., on August 1, 1935 as a stretcher case complaining of inability to use his hands and feet, to such an extent that he could not walk or feed himself. Patient is 44 years old and retired from the Navy in 1934 after 17 years' service, most of which time was spent in the printing trade. In this work he often melted up lead and was exposed to the fumes of lead as well as handling type at all times. He was also exposed at various times to aluminum at this work. After his Navy service he continued at the printing trade, but was not exposed to molten lead.

In 1934, at the time of his retirement, he was in good health except for "pins and needles" feeling in his right hand. His disability progressed, involving both hands, then his right leg and finally his left leg. By February 1935 he was unable to walk at all or feed himself.

His condition had been diagnosed as multiple sclerosis and also myasthenia gravis. He had been treated with glycine and had all his teeth extracted without improvement. He was thoroughly studied at the Mayo Clinic, where blood and urine studies were negative for lead poisoning. There a diagnosis of neuronitis was made and treatment with pilocarpine given.

On admission physical examination showed both hands atrophic, with limited movements. Feet could not be moved. There were areas of diminished sensation over arms and legs with loss of reflexes of the extremities. Physical examination was otherwise essentially negative. Laboratory examination showed negative Kahn, negative urine, slight anemia, but no stippling seen.

A skin biopsy was sent to Dr. L. Edward Gaul, of New York City, who very kindly did a biospectrometric (quantative spectrographic

anaylsis for metallic constituents) examination and made the following report:

Metallic elements were present in normal quantities except for 0.000001 gram lead retention and high aluminum retention. This is four plus lead retention, or the highest that they get.

The patient was treated with the metal-free diet and therapy as suggested by Dr. Gaul and used in the Neurological Institute of New York City. This is briefly as follows:

Medication, ammonium chloride, 1 gram t. i. d., increasing the dose 1 gram every other day until a maximum dose of 18 grams a day is given.

Ammonium phosphate, same as above on alternate days.

Intramuscular sulphur colloidal (Doak), one injection every third day for 10 injections.

Patient to drink 14 to 16 glasses (3,500 to 4,000 cc) of metal-free water every day.

Diet consisted of fresh fruit, fresh vegetables, butter, meat or fowl, eggs, bread, and honey, with the following restrictions:

All food seasoned with chemically pure salt.

Milk, cream, and butter to be metal free.

No cooking fat of any kind allowed; only butter used.

No sugar of any kind; honey was used in its place.

No sea food or food canned, dried, or preserved in glass.

All food kept, prepared, and cooked in iron, glass, pyrex, or enameled cooking utensils.

Bread was made with unsifted wheat flour.

The patient was kept on this treatment for 6 weeks. Twice during this time gastro-intestinal upsets necessitated discontinuing the ammonium drugs for several days and then starting again on the 1-gram dose with daily increases. The water was provided by the laboratory still. Metal-free butter could not be secured in Chicago, so ordinary butter had to be used. Otherwise the diet was strictly adhered to.

At the conclusion of the course of treatment, the patient started to improve rapidly and his improvement has been continuous ever since. At the conclusion of the treatment another skin biopsy was sent to Dr. Gaul, who reported that metallic elements were present in normal quantities.

The patient at the present time feeds himself, shaves himself, lights and smokes cigarettes, and walks with a typical steppage gait. He still requires support when walking.

UNILATERAL ACQUIRED SYPHILITIC INTERSTITIAL KERATITIS: REPORT OF CASE

By J. A. MILLSPAUGH, Lieutenant, Medical Corps, United States Navy

Interstitial or parenchymatous keratitis is a chronic inflammation with cellular infiltration of the middle and posterior layers of the cornea which becomes hazy throughout and has a ground-glass appearance.

The disease occurs most commonly between the fifth and the twentieth years; it is exceptional after 30. Females are more often affected. Generally both eyes are attacked, usually in succession. It is said the companion organ scarcely ever escapes involvement. Relapse occurs. Prognosis is guarded; 2 decades ago it was well nigh hopeless.

Congenital syphilis is the etiology in the great preponderance of cases. Approximately 50 percent of congenital luetics present an interstitial keratitis. Tuberculosis is the next but far less frequent cause. It is said to occur very rarely in acquired syphilis.

Duane states:

> By far the greatest number of cases of parenchymatous keratitis are to be referred to hereditary syphilis. In a few cases this form of keratitis is also observed in acquired syphilis.

May says:

> It [interstitial keratitis] is rarely the result of acquired syphilis.

Stokes concedes that it may occur with the secondary outbreak, but that it is an extremely rare complication. He further concedes that he has never seen an example. This observation is perhaps justification for presentation of such a case:

The patient is a white male aviation machinist's mate, first class, United States Navy, aged 32, height 69 inches, weight 154 pounds, of German descent, general physical appearance good. There are no congenital syphilitic stigmata. The frontal and vertical hair is thinned. This was noted many years ago and it has not progressed. It is thought to be a familial trait, as father and brothers are similarly affected.

Family history.—Nothing is known of the patient's grandparents. His father and mother are living and well, each aged 78. There are seven siblings, of whom six are living and well; one died at the age of 6 months from an unknown cause. A wife and one child (the only pregnancy) are apparently normal; their serology is Kahn negative.

The patient's preservice medical history is insignificant. He has more than 13 years' naval service. In 1923 he had 5 sick days with a diagnosis of bronchitis acute. In 1925 he had 15 sick days with a diagnosis, "Wound, incised, right thigh." Until development of his present disability the patient had no other admission to the sick list—a notably clear health record. He has two good-conduct medals.

Present illness.—August 17, 1935, patient reported to the dispensary, Naval Air Station, Pensacola, Fla., complaining of irritation of the right eye. He thought there was a foreign body present. None could be found. Two days later the irritation of the right eye persisted and was more pronounced. He was

admitted to the dispensary ward for hot wet boric-acid compresses followed by silvol drops. Two days later the eye was unimproved and he was transferred to the Naval Hospital, Pensacola, Fla., for treatment, with a diagnosis "Undetermined (conjunctivitis acute, right eye). 8-20-35."

At the hospital the chief complaint was pain and inflammation, right eye. History: Pain and itching were first noted an hour following swimming 4 days previously. (It was thought occasioned by a foreign body, though, as stated, none was found). The eye became injected. An area of erythema was noted about the right side of the nose.

Physical examination.—8-20-35: The scleral conjunctiva and the conjunctiva of the lids present signs of acute inflammation. There is a small amount of mucoid exudate. The conjunctiva of the lids is studded with numerous elevated patches. Inflammation extends toward the inner canthus and superficially involves the tissues upon the right side of the nose. There is no nasal obstruction. Physical examination is otherwise normal. 8-22-35.

8-22-35: Very little response to routine treatment has occurred. Copper sulphate applied to lids. Inflammation subsiding slightly. A routine blood Kahn test was reported 4 plus.

8-23-35: Diagnosis changed to conjunctivitis follicular. Reason for change: Diagnosis established. On the eighth day of hospitalization a repeat blood Kahn test was 4 plus. The patient has no luetic history or penile scar. He recalled that he had been closely associated with a fellow worker who was syphilitic. He has cared for minor wounds and injuries the companion had sustained while working with airplane motors. Many times the skin of the patient's hands were abraded and might have been contaminated with his friend's blood. Cellulitis of nose improved. Slight scleral injection.

8-27-35: Antiluetic treatment instituted with neo-salvarsan and bismuth concurrently.

9-2-35: There is beginning circum corneal injection. Vitreous somewhat cloudy. The right cornea appears steamy at 9 o'clock at outer border. The pupil was dilated with atropine sulphate 1 percent.

9-3-35: Circumcorneal injection persistent. Atropine 1 percent gtt. 1 B. i. d. Corneal opacity increased in size.

9-10-35: Cornea cloudy at 9 o'clock; taking on an appearance of interstitial keratitis. Vitreous also somewhat cloudy.

9-10-35: Diagnosis changed to kerato-iritis (right eye). Reason for change: Concurrent disease (syphilis).

9-17-35: Opacity clearing. Atropine and hot compresses continued.

9-24-35: Circumcorneal injection very slight. Opacity clearing.

10-2-35: Circumcorneal injection persists. Opacity clearing. Thiosinamine ointment 10 percent applied to right eye at night. Atropine, 1 gtt. daily, continued.

10-5-35: Bordering ulcer of nose resorbing centrally, but seems to be spreading peripherally. Thiosinamine and atropine continued. KI. gtts. 10 B. i. d. P. c. Kahn 4 plus. Urological consultation as to advisability of spinal puncture requested. (Spinal puncture was not done.)

10-5-35: Diagnosis changed to syphilis (interstitial keratitis). Origin not misconduct. Reason for change: Error. This change was based upon persistent keratitis and positive Kahn tests. The date and site of initial lesion cannot be definitely determined.

10-10-35: Condition of eye remains about the same.

10-15-35: Patient has received eight injections of neo-salvarsan or a total of 4.6 gm. Hg inunctions begun. KI. continued. Local treatment to eye consists of atropine and dionine 2 percent.

10–21–35: No change.

10–24–35: Dental examination. Negative findings.

10–25–35: Eye slightly improved. X-ray findings of chest: Plate reveals both apices slightly cloudy with Gohn's nodes thruout both lung fields. The chest is of barrel shape and does not show good expansion. Each lung field is characterized by fibrotic changes with excess bronchial markings and peri-hilic infiltration.

10–28–35: Ulcer of nose almost healed. Opacity of cornea persists.

10–30–35: Opacity about same. Vision 20/20 both eyes.

11–7–35: Condition unchanged. Patient has had maximum benefit of hospitalization. Anti-luetic treatment, KI. and atropine to be continued.

11–8–35: To duty this date. The syphilitic abstract contains the following information: Diagnostic evidence.

8–16–35: Primary ulcer right side of nose with cellulitis. Treponema not found. Blood reaction 4 plus Kahn. Secondaries: Adenopathy. (Definite bilateral, inguinal and epitrochlear, slight posterior cervical.) Tertiaries: See below.

Clinical data.—History contact (accidental) with fellow worker whom he states has syphilis and whom he treated for bruises and cuts sustained while working on motors. States he had open abrasions of skin when he attended fellow worker. Probably primary: Ulcer right side nose with cellulitis. Repeated dark fields negative for treponema. Circumcorneal injection, iritis. Complications: Corneal ulcer and opacity, interstitial keratitis. Infection considered not due to patient's own misconduct.

This patient was not seen by the writer until he was discharged from the Naval Hospital, Pensacola, Fla., on November 8, 1935, and referred to me for continuation of his antiluetic treatment. I am indebted to Lt. J. J. V. Cammisa (M. C.), United States Navy, for his kind permission to use the above excerpts from August 20, 1935, to November 8, 1935, written by him.

On November 8, 1935, when the patient returned to the dispensary, there was a slightly depressed, minutely modular, irregular scar about 1 cm in diameter on the right lateral nasal wall, an inch below the right internal canthus. The right pupil was dilated. A wedge-shaped area with the apex at the dilated pupil extended laterally to the corneal limbus and involved approximately one-fifth of the corneal surface. This area was definitely hazy and had a ground-glass appearance. Dark glasses were being worn.

Following the thirty-fifth mercurial inunction antiluetic treatment was continued: 0.6 gm of neo-salvarsan and 0.13 gm bismuth salycilate in oil, one injection each, are being administered weekly. KI., drops 10 daily, are also taken. Atropine sulphate is being instilled in the right eye daily under the direction of Dr. Cammisa. The patient reports to the eye clinic every 3 weeks. The vision was normal on December 2, 1935. Dark glasses are still worn. The steaminess has decreased slightly in density. The left or companion eye has thus far escaped involvement. Intensive and continuous treatment, it is believed, will forestall similar disease of the normal eye.

DISCUSSION

There are a number of uncommon factors in this case. The sex, age, unilateral involvement and development of acquired syphilitic interstitial keratitis are progressively unusual in that order. The pathogenesis and site of initial lesions are almost equally infrequent. Accidental infection with syphilis is of course possible. The coworker

mentioned does have a persistently positive Kahn; he had been treated for syphilis, which diagnosis was established by serological evidence only. Laterally the coworker's treatment has not been intensive because of an idiosyncrasy to neo-arsphenamine. The site of the alleged initial lesion being in close proximity to the eye may have a bearing on the keratitis although the lymphadenopathy in this area is least marked of all. Had a positive dark field been attained enormously more credence would be accorded. However, this lesion had been treated repeatedly with various chemicals, including alcohol, though this does not appear in the record. Lastly and fortunately the visual acuity is normal and the cosmetic disfiguration is being steadily attenuated by treatment.

NEW DEVICES

THE ZIPPER STRETCHER SUIT

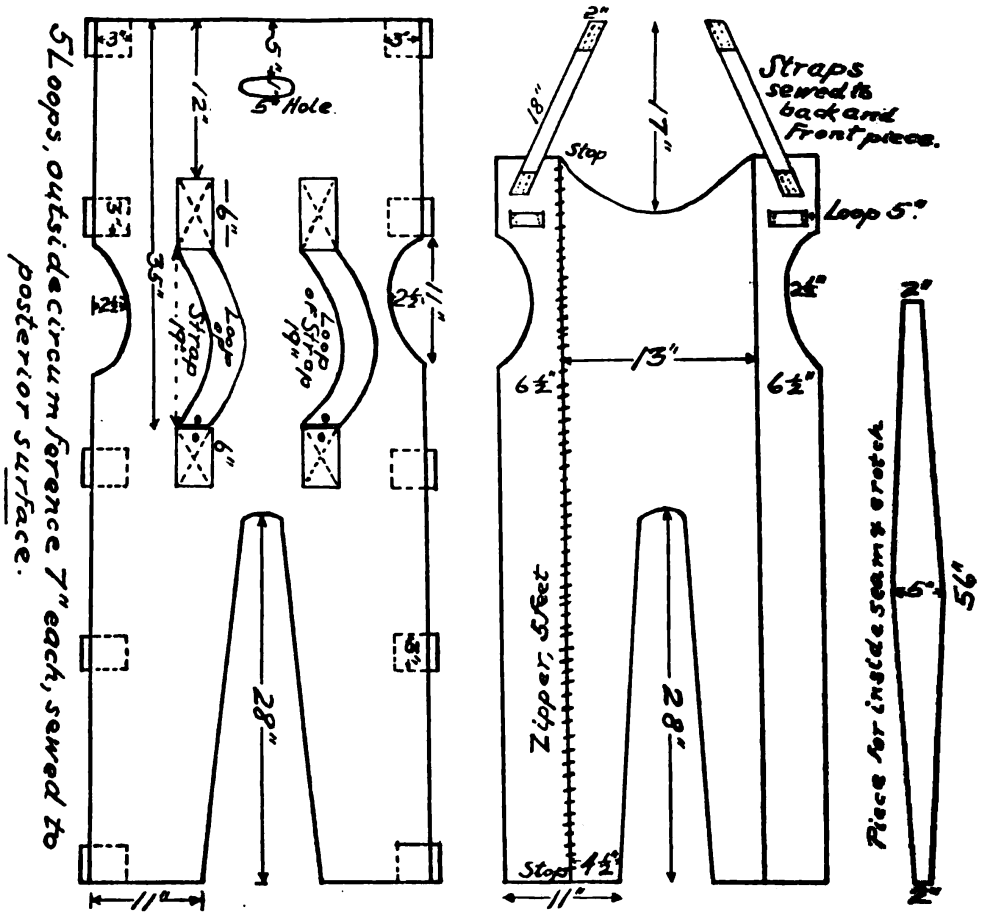
By HENRY C. WEBER, Lieutenant Commander, Medical Corps, United States Navy

The medical officer attached to a ship is often confronted with difficulties of transporting patients from inaccessible places, when a Stokes stretcher does not meet the requirements. One can readily visualize how this problem can become still much greater during and after battle on account of debris and confusion. As examples may be mentioned, removal of patient from a catapult, lookouts, control room, from magazines and handling rooms, and from fire rooms through the airlocks, or from any place where a short turn must be made with the stretcher. With these difficulties and speedy evacuation in mind, the writer designed the "zipper stretcher suit" which is presented herewith. Accompanying this description are drawings, giving the dimensions of an average suit, and 10 photographs which are self-explanatory.

The suit is made of no. 12 canvas. It consists mainly of a back piece, going the full length of the body and extending about 10 inches beyond the patient's head, and three front pieces. The back part is cut from a rectangular piece of canvas, dimensions 72 by 28 inches. The space between the legs is cut out to conform to the legs, having a 4 inch space at the crotch. The bottom of the trousers should be about 24 inches in circumference, which makes 12 inches for the posterior surface. For the arm holes, ovals are cut out from both the front and rear piece, 11 inches long and 2½ inches wide, beginning 18 inches from the top. Three inches from the center of the top is a slot about 1 by 5 inches for insertion of the hand or hook. This slot is reinforced on both sides with leather. To the back are sewed two straps, each 25 inches long over all, to serve as shoulder straps; they are 2½ inches wide and are sewed on for a length of 6 inches, with three rows of stitches 12 inches from the top and 7 inches from the side of the suit. They should be made of two thicknesses of canvas and stitched securely with extra heavy thread. At the loose ends of the straps are attached signal halliard snappers. The lower these straps are attached, the higher the patient's feet will be raised from the deck in the back-to-back carry. Below, the shoulder straps hook on to metal rings fastened to tongues of reinforced canvas 2½ by 6 inches, which are sewed on the posterior surface of the back piece above the hips, 34 inches from the top of the suit and in line with the shoulder straps.

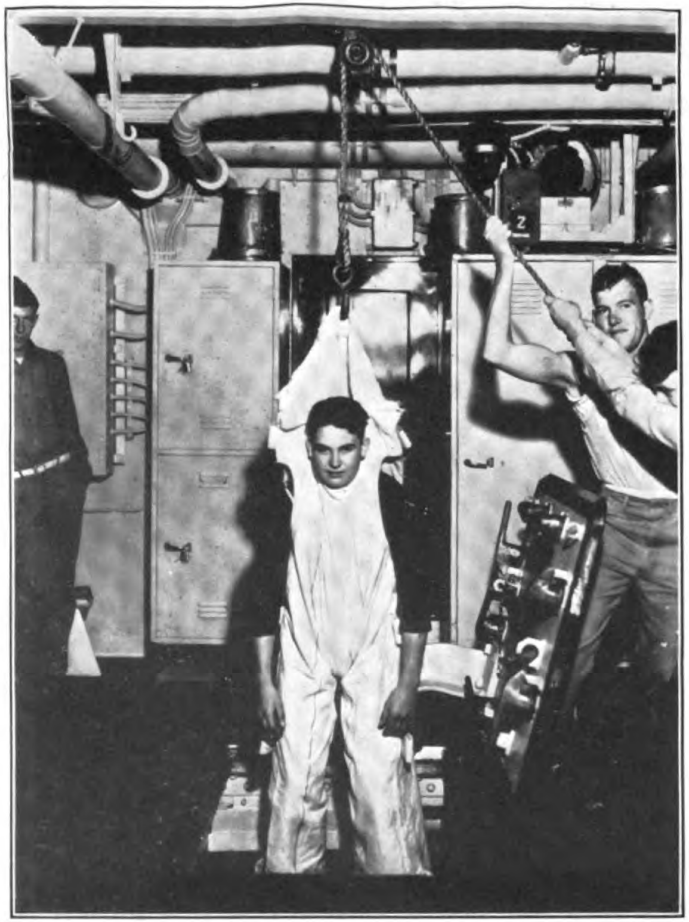
The front of the suit consists of three pieces of canvas, two side, and one center. The side pieces are identical, 60 by 6 or 7 inches. The lower edge is straight. The upper edges form a downward curve with the centerpiece as illustrated to allow room for the neck. The center piece of the front is 13 by 55 inches, a little longer at the outer edges to meet the line from the side pieces. The lower part is

Zipper Stretcher Suit.
H.C. Weber,
Lieutenant Commander (M.C.), U.S.N.



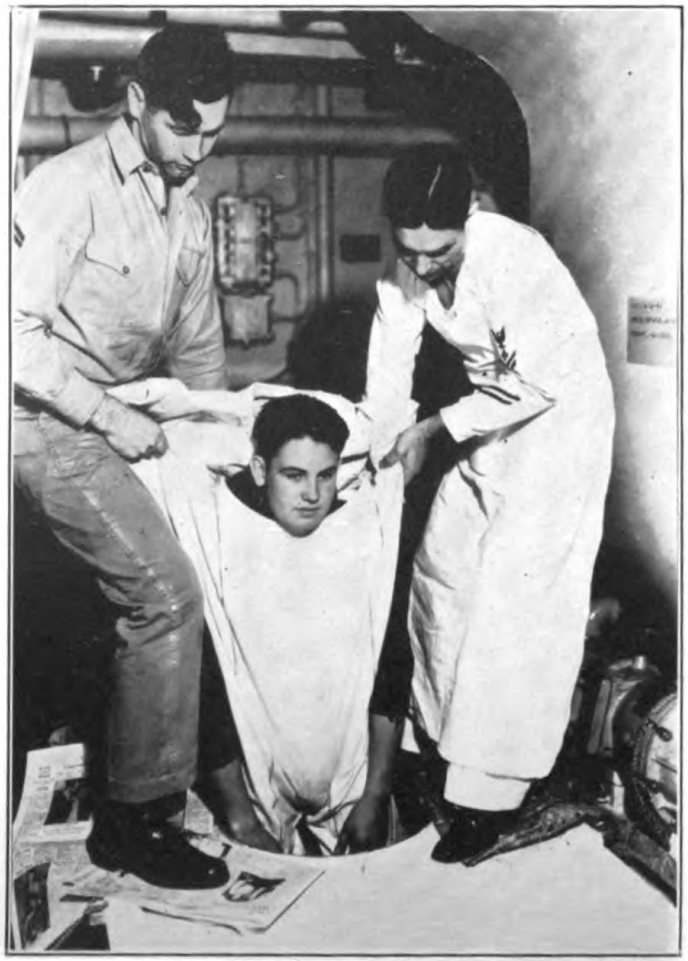
Back Piece 72"x 28" Front Piece 60"x 28"

cut out to conform to the dimensions of the back piece canvas of the legs. To give extra room in the crotch, a piece of canvas, 5 inches at the widest portion—i. e., the crotch—and gradually tapering off to 2 inches, is sewed between the front and back piece of the legs. See drawing. To the inside of the crotch is sewed a pad of hair-felt horizontally, dimensions 10 by 6 by 2 inches.



BRINGING PATIENT UP THROUGH A NARROW HATCH WITH LINE AND PULLEY.

BACK TO BACK CARRY ON LADDER. SHOWS FREEDOM OF CARRIER TO USE HANDS.



COMING UP THROUGH ESCAPE SCUTTLE.

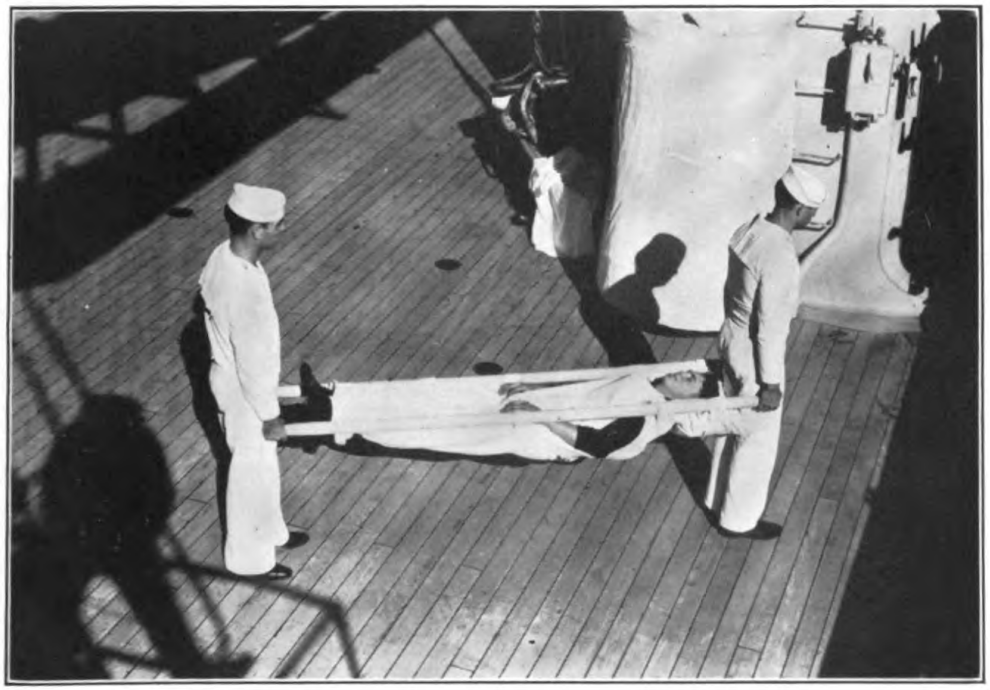
PATIENT IN SUIT, LEFT SIDE OPEN, RIGHT SIDE PARTLY CLOSED. PILLOW IN PLACE.

The zipper parts are sewed to the inner edge of the outer and to the longitudinal edges of the center pieces, so that the suit can be opened quickly all the way from the shoulders to the feet. The lower ends of the trousers are held together by stitching the metal stop in place, so that it will be easier to start closing the zipper mechanism. At the top of the zipper line, is secured a metal clip as a stop to prevent the moving part from being pulled of inadvertently.

From near the tops of the side pieces, straps 1½ by 17½ inches are sewed in a diagonal direction to near the upper edge of the back piece; they help to take the strain off the zippers. When necessary, a piece of 3-inch roller bandage can be tied to them horizontally to prevent the head from sagging when an unconscious patient is carried in the upright position. To the upper ends of the outer front pieces are stitched two loops 1½ by 5 inches for pulling the patient through holes. To the back piece, 3 inches from either seam, are sewed 5 loops to admit 2 by 2-inches carrying poles. They are spaced thus; one each on top and bottom, one above armhole, one 22 inches below the preceding one and one 19 inches above bottom. This will place the loops at top, shoulders, hips, knees, and bottoms. When used as a stretcher in the ordinary way, a strip of 3-inch roller bandage can be tied to both poles to support the feet.

The weight is all carried in the crotch when the patient is in the upright position and by the back piece when he is supine; there is no strain on the zippers, except that needed to keep the patient from doubling up. It appears that the stretcher would be ideal for improvised treatment of cases of broken pelvis on board ship. In such an emergency, the poles could be supported on the frame of the surgical beds, after the mattress were removed. It is believed that the stretcher suit would be useful in aviation medicine. The methods of carrying can best be seen in the photographs. This stretcher was not designed with the idea of replacing the Stokes stretcher, but to take care of conditions where that old reliable stretcher can be used either not at all or only with great difficulty. The advantages are lightness, compactness, adaptability, cheapness, freedom of action of carrier, and ease of conversion from one method of carrying to another.

The writer acknowledges gratefully his obligation to Commander H. V. Bryan, United States Navy, for furnishing material and help from the Construction and Repair Department; to P. Simpson, chief pharmacist's mate, United States Navy, for the photographs; to J. M. Howard, pharmacist's mate, first class, United States Navy, for the typing; F. F. Hallinski, sailmaker's mate, first class, United States Navy; and E. O. Johnston, seaman first class, United States Navy, for making the suit, and other members of the crew for their cooperation.



CARRYING POLES INSERTED IN LOOPS. THREE-INCH ROLLER BANDAGES TIED TO POLES AS SUPPORT FOR LEGS. PILLOW UNDER HEAD.

NAVAL RESERVE

OFFICERS OF THE NAVAL RESERVE RECEIVING CERTIFICATES IN AVIATION MEDICINE

Certificates for the successful completion of a course in aviation medicine were awarded, through the office of the district medical officer, headquarters, Third Naval District, to the following United States Naval Reserve medical officers at a dinner held on February 15, 1936, at the Hotel Cambridge, New York, N. Y.:

Lt. Comdr. William J. Fordrung, M. C.-V. (S), U. S. N. R.
Lt. (jg), Lawrence A. Gerlach, M. C.-V. (G), U. S. N. R.
Lt. Comdr. Manfred J. Gerstley, M. C.-V. (S), U. S. N. R.
Lt. Comdr. Charles M. Gratz, M. C.-V. (S), U. S. N. R.
Lt. Comdr. Abraham Jablons, M. C.-F., U. S. N. R.
Lt. Comdr. Walter R. Loewe, M. C.-V. (S), U. S. N. R.
Lt. Comdr. Oscar Wald, M. C.-V. (S), U. S. N. R.

The course was conducted at the district medical office, Third Naval District, Cornell University Medical School, and the Floyd Bennett Air Field.

UNITED STATES NAVAL RESERVE MEDICAL OFFICERS—PROMOTIONS—FIRST QUARTER, 1936

Lt. Gordon Maxwell James, M. C.-V. (G), U. S. N. R. Promoted from: Lieutenant (junior grade), N. C.-V. (G). January 15, 1936.

Lt. James Peery Williams, M. C.-V. (G), U. S. N. R. Promoted from: Lieutenant (junior grade), M. C.-V. (G), U. S. N. R. January 17, 1936.

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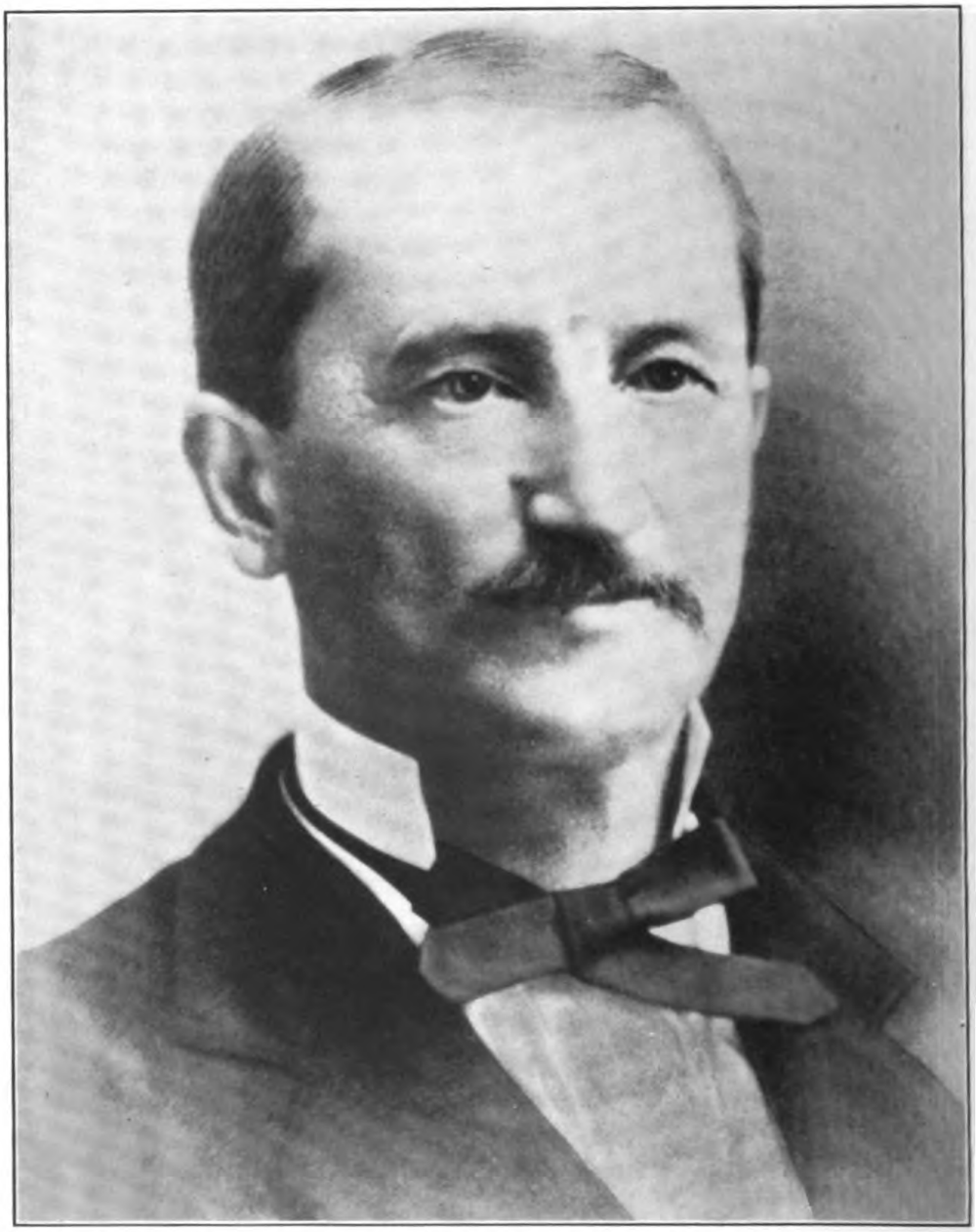

J. WINTHROP TAYLOR.
SURGEON GENERAL, U. S. NAVY, 1878-79.

NOTES AND COMMENTS

J. WINTHROP TAYLOR, SURGEON GENERAL OF THE NAVY, 1878–79

The tenth Chief of the Bureau of Medicine and Surgery and the sixth to hold office as Surgeon General of the Navy was born in New York on August 19, 1817. He was appointed as Assistant Surgeon in the Navy from New Jersey on March 7, 1838. He saw considerable sea service during the first years of his naval career, principally in the West Indies and home squadrons. He was commissioned a Surgeon on May 1, 1852. During the Civil War he was on the steam sloop *Pensacola*, of the west Gulf blockading squadron, from 1861 to 1863, and saw much of the arduous service connected with the capture of New Orleans under Farragut and the regaining of naval control of the Mississippi River by the Union forces. The latter part of the Civil War he spent at the naval rendezvous, as it was called, at Boston. Subsequent duty included that of fleet surgeon of the North Pacific Squadron and at the naval hospital, Chelsea. He was appointed Surgeon General of the Navy on October 28, 1878, by President Rutherford B. Hayes, retiring from that office on August 19, 1879.

His death occurred on January 19, 1880.

During the administration of this Surgeon General, for the first time, exact, uniform, and rigid physical standards were instituted for entrance to the Naval Academy. Medical Inspector Albert L. Gihon, then on duty at Annapolis, made careful studies of the height and weight of midshipmen and their rate of growth and published extensive tables embodying the results of his observation.

The naval hospital fund in 1878 showed a balance of $47,746.25. The chief of Bureau stated in his report that approximately $100,000 a year was required for the maintenance of the naval hospitals. A bill "to authorize the appointment of apothecaries as warrant officers in the United States Navy", the foundation of the present group of pharmacists, passed the House of Representatives, a revised book of instruction for medical officers was issued, and a study of the Navy ration was instituted during Surgeon General Taylor's regime.

Surgeon J. B. Parker was assistant chief of Bureau and had also held this office during the greater part of the time that Dr. William Grier was Surgeon General.

NEED FOR FLIGHT SURGEONS

There is a definite need for more medical officers in the field of aviation medicine, and the Bureau would welcome requests from medical officers, particularly in the grades of lieutenant and lieutenant junior grade to enter this specialty. Candidates whose requests are approved will receive a 4 months' course at the Army School of Aviation Medicine, Randolph Field, Tex. The next class will begin August 15, 1936. The subjects covered by the course include physiology, psychiatry, administration, cardiology, psychology, ophthalmology, and otology.

THE LIMITATIONS OF DIATHERMY

Medical diathermy is defined as "the production of heat in the body tissues for therapeutic purposes by high-frequency currents." The current is applied by—

(*a*) Contact metal electrodes (½ to 2 million cycles per second).

(*b*) High-frequency alternating electric field (10 million to 100 million cycles per second).

(*c*) High-frequency electromagnetic field.

In any of these methods burns may occur.

A vast number of claims have been made regarding the therapeutic effects of diathermy. The statement, however, of Martin and Osborne places these claims in the proper place, at least in the light of our present knowledge. They say:

> There is no conclusive evidence from the literature, nor were we able to substantiate the claim of specific biologic action of high-frequency currents (short-wave diathermy). In our opinion the burden of proof still lies on those who claim any biologic action of these currents other than heat production.

It may be said, therefore, that there is no definite evidence of effect from diathermy not explained on the basis of the generation of heat in the tissues. Selective bactericidal action or biologic effects cannot be satisfactorily demonstrated.

There are certain well-defined contra-indications in the use of high-frequency currents which for convenience for reference are briefly tabulated here:

(1) In acute inflammatory processes such as acute cellulitis without drainage, acute infectious arthritis, and acute pelvic infections.

(2) Where there is a tendency to haemorrhage (gastric ulcer).

(c) Over areas where heat sensation is lost (after certain nerve injuries).

(4) Over pelvic and lower abdomen in pregnancy.

(5) Over areas of suspected malignancy.

(6) Where simpler methods of applying heat give satisfactory results.

A NOTE ON THE HISTORY OF THE TREATMENT OF HEAT EXHAUSTION WITH SODIUM CHLORIDE

The loss of salt as an important etiological factor in heat exhaustion, and the use of sodium chloride both in the prophylaxis and treatment of the condition, are factors now pretty well established and well known to the medical profession. What is not so well known is that a naval medical officer, as long ago as 1908, strongly recommended it in the treatment of this condition, although not aware of the significance of salt loss in the etiology of heat exhaustion.

The history of the Medical Corps of the Navy shows that its members made many contributions to medical progress and have been among the pioneers in a number of important professional fields, though many of their efforts have gone unregarded. The present case is one in point.

In 1908, Surgeon (now Rear Admiral, Medical Corps) Middleton S. Elliott, United States Navy, contributed to the March number of the Military Surgeon an article entitled "Heat Exhaustion on Men-of-War", incidentally a fine seagoing title for a medical paper. In addition to a careful survey of the subject of "prostratio-thermica", as the condition was called in the official nomenclature of that day, Surgeon Elliott described three very severe cases, one of which was fatal. One was treated with both hypodermoclysis as well as by the injection into the rectum of hot saline. This case recovered. So did the third case, who also received hot saline by rectum. So impressed was he by this that Surgeon Elliott in discussing the treatment of heat exhaustion makes the following significant statement: "The main reliance should be placed on high rectal administration of hot saline solution."

He mentions further that he believes that the first patient's life could have been saved if this treatment had been used. He says:

> In cases 2 and 3 this treatment was quickly efficacious and the patients in a complete state of collapse were in half an hour out of danger.

This strong lead, however, was not followed up, and it was not until 25 years later that other workers, notably Talbot and Michelson at the Boulder Dam project, established clearly the etiologic relationship between chloride exhaustion and heat exhaustion and clearly pointed the way both for prevention and treatment.

It is of historic interest, nevertheless, that a medical officer of the Navy was perhaps the first to notice the beneficial effects of the administration of salt and to strongly recommend it in the treatment of heat exhaustion.

SPECIALIST NEEDS OF THE NAVY AT PRESENT

The maintenance of the proper number of medical officers in each of the specialties is a matter of great importance and one to which the Post graduate Board gives the most careful attention. The requirements of the Navy are peculiar in that the necessity for an officer to perform sea duty as well as administrative duty are added to the losses from normal attrition, such as death, retirement, or similar causes. Changes, too, occur in the needs of the service, due to changing service conditions. All of these factors, therefore, must be considered.

The specialist requirements, arranged in the order of the greatest need for 1934, 1935, and 1936, are here given, not only to show the requirements at present but to afford a comparison with the two previous years.

1934

Clinical laboratory and pathology.
X-ray.
Neuropsychiatry.
Submarine duties.
Surgery.
Obstetric and gynecology.
Internal medicine.
Eye, ear, nose, and throat.

1935

Aviation medicine
Dermatology.
Clinical laboratory and pathology.
X-ray.
Submarine duties.
Field service.
Obstetric and gynecology.

1935—Continued

Neuropsychiatry.
Surgery.
Preventive medicine.
Tropical medicine.

1936

Aviation medicine.
Submarine duties.
Field service.
X-ray.
Surgery.
Obstetrics and gynecology.
Neuropsychiatry.
Clinical laboratory and pathology.
Eye, ear, nose, and throat.
Tropical medicine.

PATHS TO MEDICAL SPECIALISM

It has long been a subject of discussion among medical men as to the proper preparation for entrance into a specialty. If a man is to be internist, surgeon, obstetrician, or eye, ear, nose, and throat specialist, how many years should be spent in general practice or some other accessory or related specialty as preparation for his chosen field? Here they all agree that from 3 to 5 years of general practice are a prerequisite, but there agreement ends and opinions vary as to the other roads to be followed and what other related specialties offer the best road to travel toward the ultimate goal.

Three of the most eminent American obstetricians entered their specialty by different routes. One was a general surgeon, one was an internist, and one was a pathologist. Each one of these men attained to eminence in the field of obstetrics and has written a

textbook well known to English speaking medical students and physicians. Each made a particular contribution to obstetrics because of his previous training. It is of interest to remember that one of the greatest of American pediatricians began his professional career as a psysiologist.

It has long been thought by many medical men that the best entrance to internal medicine is through the laboratory, and there has been a tendency to make that the necessary prerequisite. It must be pointed out, however, that this lessens the number available for selection and consequently much lessens the availability of talent. It would seem that for the specialty of internal medicine, general practice itself is perhaps the best preliminary training. X-ray, because of its close connection to both the diagnostic and therapuetic aspects of medicine and surgery, is an excellent entrance to both of these specialties. Bacteriology and dermatology, as well as genito-urinary surgery are excellent preparation for general surgery, and general surgery excellent preparation for specialization in genito-urinary work. Psychiatry, itself a branch of internal medicine, offers a fine preliminary training for the internist.

The most important factor in the selection of specialists is really the individual, his ability and inclinations. He will usually himself tend to follow the road that will best lead him to the professional field which he desires to occupy. An acute mind, tact, discretion, a sympathetic and engaging personality, and a prepossessing appearance; these are required to make a successful physician. They are the requisities for a specialist and their possessor is certain to utilize the opportunities of the preliminary years before specialization no matter what path to the specialty has been followed. In other words, the path is less important than the man who walks in it.

THE EARLY DIAGNOSIS AND TREATMENT OF MALIGNANT DISEASE IN THE NAVY

The Naval Hospital, Brooklyn, N. Y., and the Naval Hospital, San Diego, Calif., are the regular treatment hospitals for cases of malignant disease in the Navy, the former serving the Atlantic coast, the latter the Pacific coast. The Naval Hospital at Great Lakes, Ill., sends cases to the Naval Hospital at Brooklyn. The designation of the naval hospitals at Brooklyn and San Diego as treatment centers for neoplasms has gone hand in hand with the provision at them of special facilities and contacts making available the latest and best advice and equipment needed in the treatment of malignant disease. At both of these hospitals special consultants assist in the diagnosis and treatment. Treatment by X-ray and radium is available. Patients with malignancy or in which it is strongly suspected,

should be transferred as early as possible to one of these treatment centers. Such a transfer can be expedited by a dispatch requesting approval prior to the routine approval of the medical survey. In sending such a patient it is advisable if a biopsy has been done that a slide and a portion of frozen tissue be forwarded as well. If no biopsy has been done a statement to that effect should accompany the patient. The importance of time in dealing with possible malignant disease should be uppermost in the mind of every medical man and the earliest possible diagnosis is more important in neoplasm than in almost any other pathological condition which runs a course of equal length.

NOTICE IN REGARD TO MICROTOME KNIVES

The Bureau's circular letter of September 11, 1933, in regard to the sharpening of microtome knives, has been canceled, and the Naval Medical School desires the following information on this subject be published here.

(*a*) Microtome knives which require sharpening should be forwarded to the Naval Medical School, Washington, D. C., in individual cases and suitably packed, with name and address of sender securely attached.

(*b*) The apparatus used, a Franz automatic microtome sharpener, requires that each blade be accompanied by its honing back.

(*c*) If, as may be found the case, the back is too worn down to permit adjustment of the sharpener to the bevel of the blade, the work cannot be done at the school. Nor is the sharpener capable of reconditioning knives with deep nicks, greatly curved edges, or extremely narrow blades.

(*d*) Resharpened knives usually can be returned 1 week after receipt at the Medical School.

BOOK NOTICES

Publishers submitting books for review are requested to address them as follows:

The Editor, United States Naval Medical Bulletin,
Bureau of Medicine and Surgery, Navy Department,
Washington, D. C.

(For review.)

Index of Differential Diagnosis, *by various writers. Edited by Herbert French, M. D., F. R. C. P.* 1,145 pages, 742 illustrations of which 196 are colored. Fifth edition. William Wood and Co., Baltimore. 1936. $16.00.

The Index of Diagnosis, Index of Prognosis, and Index of Treatment first published many years ago and revised from time to time, form almost a medical library in themselves, so complete that if a medical man's library for a Crusoe island were to be limited to three books, these might be the best choice. For there is this noticeable difference in this diagnosis and most of those published, that this is not confined to medical diagnoses alone but includes the surgical and obstetrical fields as well. Laboratory tests are given in sufficient detail for it to be used as a laboratory manual of technique. It is a real encyclopedia of diagnosis and arrangement is alphabetical by symptoms. As an example of completeness, a subject such as erythema includes a table of causes which at a glance give more information than could very well be obtained after searching through texts on dermatology. Two features of the book which should not pass unmentioned are the remarkably fine illustrations and the index. The latter takes up 218 pages and is probably the most complete index found in any medical book.

The Discovery of the Elements, *by Mary Elvira Weeks, Assistant Professor of Chemistry at the University of Kansas.* 371 pages ; illustrated. Third edition. The Journal of Chemical Education, Mack Printing Co., Easton, Pa., 1935. $3.00.

The first edition of this book was made up of collected articles published in the Journal of Chemical Education. The fact that in a short space of time this book has gone through three editions is an evidence of its excellence. Dr. Weeks has written chemical history so entertainingly that she has had no trouble in finding readers. The potential readers in this country are many, for there are millions of

high-school and college students of chemistry and their teachers, while others who have forgotten nearly all their chemistry are likely to read this book. Miss Weeks has followed the biographical method so well employed by Garrison in his History of Medicine, and the thumbnail sketches and glimpses of the personalities of the pioneers of chemical science supply what a newspaper reporter refers to as "human interest." A physician is much impressed by the great number of medical men and pharmacists who have contributed to the development of chemistry. The paramount position of these two professions in the development of the biological sciences is equalled in the rise of chemistry. Of the discovery of the circulation of the blood by Harvey, old Sir Thomas Browne said "I prefer it to that of Columbus." Miss Weeks has shown again that the thrills of scientific discovery are equal to those of geographic exploration. The hundreds of illustrations collected by Dr. F. B. Davis, professor of chemistry at the University of Kansas, constitute almost a pictorial history of chemistry.

A TEXTBOOK OF SURGERY, *by American Authors. Edited by Frederick Christopher, M. D., F. A. C. S., Associate Professor of Surgery at Northwestern University Medical School.* 1,608 pages, 1,349 illustrations. W. B. Saunders Co., Philadelphia. 1936. $10.00.

This is a fine manual of surgery and appears to have as few defects as any book of composite authorship can have. One of the most striking sections is that entitled "Bacteriology of Surgery", written by Dr. Meleney, of Columbia University. The section devoted to fractures appears to be relatively less than in most surgical manuals, but lack of quantity is more than compensated for by quality. The illustrations to this section are also particularly fine. A section on minor surgical procedures will appeal to many general practitioners. A practical subject dealt with very carefully is one entitled "Management of Surgical Diabetic Patients", by Dr. Wilder. Short discussions on water balance and acidosis and alkalosis, outlining the principles to follow, are also valuable features. There is an excellent index and the book is very handsomely bound.

One of the great merits of Dr. Christopher's Surgery is that by utilizing a number of authors a more authoritative character is given to it than would be the case with a book by any single author. The field of surgery is so large that it is virtually impossible for any one surgeon to be so familiar with every phase of the subject. The employment of a number of authors, each intimately acquainted with the work on which he writes, gives a weight to opinions expressed not otherwise attainable. It is thus a general surgery and a regional as well. Altogether it is a text in which authority, brevity, and clarity are preeminent. Naturally such a book, too, contains the latest and most up-to-date procedures in surgery today. It is really a 1936 surgery.

LABORATORY METHODS OF THE UNITED STATES ARMY. *Edited by James Stevens Simmons, B. S., M. D., Ph. D., Major, Medical Corps, United States Army; Director of Laboratories, Army Medical Center; Associate Editor, Cleon J. Gentzkow, M. D., Ph. D., Major, Medical Corps, United States Army;* 1091 pages, 133 tables, 69 figures. Lea and Febiger, Philadelphia. 1935. Fourth Edition. $6.50.

A new edition of an excellent manual which was first compiled during the World War by members of the Surgeon General's staff. It has been revised and enlarged with each new edition and includes valuable material other than strictly technical procedures. Some 20 experts in their specialties have contributed their services in rewriting the 50 chapters which contain all the recent and important advances in the different fields.

For purpose of description the manual is divided into 11 major parts: Clinical pathology, chemistry, mycology, bacteriology, rickettseae and filterable viruses, protozology, helminthology, entomology, pathology, special veterinary laboratory methods, and statistical methods.

The figures illustrating amoebae, flagellates, ciliates, malarial plasmodia, trypanosomes, guinea pigs infected with endemic typhus, etc., are well done. The numerous tables make available at a glance a large amount of data used daily by the medical officer, laboratory worker, bacteriologist, general practitioner, internist, etc., and presents it in a form which can be easily memorized by the student.

A TEXTBOOK OF PHARMACOGNOSY, *by George Edward Trease, B. Phar., Ph. C., Lecturer on Pharmacognosy in the University College of Nottingham.* 653 pages, illustrated. William Wood and Co., Balto.

This is a very handsomely printed and illustrated textbook. In comparing it with such a well-known American text as that of Youngken, one is struck by the relatively greater amount of space in this English book given to such subjects as the cultivation of plants, the collection, drying and storage of drugs, and the commercial features of preparing for shipment, the transportation, prices and buying and selling of crude drugs. The sections dealing with chemical tests, fluorescence analysis, and microscopic identification are also particularly fine. Another feature of this book is the pictures, which are not only fine but really interesting. It would make an excellent text in teaching pharmacognosy, or splendid collateral reading with another text.

CLINICAL DIAGNOSIS OF DISEASES OF THE MOUTH, *by Louis V. Hayes, D. D. S., Lieutenant Commander United States Naval Reserve, Associate Professor of Oral Surgery, College of Dentistry, New York University.* 461 pages, 12 colored plates and 353 other illustrations. Dental Items of Interest Publishing Co., Inc., Brooklyn, N. Y. 1935. Price $7.50.

A feature of this book is its illustrations. It is really a hand atlas of the subject and as the author says "a guide for students and

practitioners of dentistry and medicine." This warm praise of the illustrations does not mean that the text is deficient. Far from it. The descriptions are excellent and include a survey of etiology, symptoms, diagnosis, and treatment in each case. It is a really valuable book, complete and very practical in its character.

LOBAR PNEUMONIA AND SERUM THERAPY. WITH SPECIAL REFERENCE TO THE MASSACHUSETTS PNEUMONIA STUDY, *by Frederick T. Lord, M. D., and Roderick Heffron, M. D.* 91 pages. The Commonwealth Fund. Humphrey Milford: Oxford University Press. 1936. $1.00.

This is the first report of a study of pneumonia initiated by Dr. George H. Bigelow and carried out in Massachusetts. Other publications are planned. This little volume deals with certain aspects of this important disease which causes somewhat over half of the 100,000 deaths from pneumonia in the United States each year, with particular reference to the value of serum therapy. The writers' conclusions are that properly applied serum therapy, at least in the cases of types 1 and 11, reduce the death rate by one-half. They feel that this serum therapy should be more widely used by the general practitioner.

PRACTICAL ENDOCRINOLOGY, *by Max A. Goldzieher, M. D.* Illustrated. 326 pages. D. Appleton-Century Co., inc., New York and London. 1935.

This is a book written primarily for the use of the general practitioner rather than the specialist, and contains a brief summary of the author's experience of 25 years' study of endocrinology.

DIAGNOSIS AND TREATMENT OF PULMONARY TUBERCULOSIS, *by John B. Hawes 2nd., M. D., and Moses J. Stone, M. D.* 215 pages, 43 engravings. Lea and Febiger, Philadelphia. 1936. $2.75.

A brief, compact, little book, packed with useful information for either physician or layman.

THE PARATHYROIDS IN HEALTH AND IN DISEASE, *by David H. Shelling, M. D., Johns Hopkins University and Hospital.* 335 pages, illustrated. The C. V. Mosby Co., St. Louis, 1935. $5.

A splendid and scientific monograph on the subject. The importance of the parathyroids is being more and more appreciated by both the medical and dental professions. The relation of these glands to calcium and phosphorus, metabolism, and the growth of the bones and teeth is better understood by a study of this new and authoritative work.

IMMUNOLOGY, *by Noble Pierce Sherwood, Ph. D., M. D., Professor of Bacteriology, University of Kansas.* 608 pages, illustrated. The C. V. Mosby Co., St. Louis. 1935. $6.

This is a carefully prepared textbook intended primarily for teaching purposes to give medical students a knowledge of the underlying principles involved in infection and resistance. There is an excellent

chapter on colloids and on hypersensitiveness and the significance of allergy. The important laboratory tests having their source in bacteriology and serology are described in detail. An excellent book. There is a good bibliography and index.

THE MEDICAL TREATMENT OF GALL BLADDER DISEASE, *by Martin E. Rehfuss, M. D., Clinical Professor of Medicine, and Guy M. Nelson, M. D., Instructor of Medicine, Jefferson Medical College.* 465 pages; illustrated. W. B. Saunders Co., Philadelphia. 1935. Price $5.50.

This volume presents the subject of gall-bladder disease from the medical point of view. It contains much of the clinical wisdom and thoughtful judgment of the authors from their study of several thousand gall-bladder patients. In the first eight chapters, after an introduction to the problem, the history, the physical and X-ray examination, duodenal intubation and the differential diagnosis receive careful discussion. Each of these subjects has been given a strong clinical emphasis.

In the following 16 chapters the plan of medical treatment of the gall-bladder patient is outlined in detail. The three fundamental factors of metabolism, infection, and stasis have been thoroughly covered. Clear and explicit directions for the different situations encountered have been given. The optimism of the authors is encouraging. They hold that by attention to details "three out of four patients who present themselves to the physician in his routine practice can be helped or even completely relieved."

A MANUAL OF THE COMMON CONTAGIOUS DISEASES, *by Philip Moen Stimson, M. D., Assistant Professor of Clinical Pediatrics, Cornell University Medical College.* 439 pages, 53 engravings, and 3 plates. Limp binding. Lea and Febiger, Philadelphia. $4.00.

Dr. Stimson's mimeographed lecture notes given to his students proved so popular and practical a guide that this book was written. The way the first edition was received by the medical profession led very quickly to the production of this second enlarged and revised edition. As it stands it is as useful a little manual of the contagious diseases as one can desire, packed with practical clinical features of particular value to the general practitioner.

ABORTION, SPONTANEOUS AND INDUCED. MEDICAL AND SOCIAL ASPECTS, *by F. J. Taussig, M. D., F. A. C. S., Professor of Clinical Obstetrics and Gynecology, Washington University School of Medicine, St. Louis.* 536 pages, 437 illustrations. The C. V. Mosby Co., St. Louis. 1936. $7.50.

Prospective medical authors would do well to examine Dr. Taussig's book carefully for the general design, what one might call the architecture of the book, is of sufficient excellence to be considered as a model. He divides his subject into four parts—history and background, spontaneous abortion, induced abortion, and social and legal aspects. There is a fine index, bibliography, and glossary. Another

feature is an appendix containing the statutes of the various States, the Federal laws and the common law regarding abortion. An index of illustrations is another useful addition.

DENTAL ROENTGENOLOGY, *by Le Roy M. Ennis, D. D. S., Lieutenant Commander, Dental Corps, U. S. Naval Reserve, Assistant Professor of Roentgenology, School of Dentistry, University of Pennsylvania.* Second edition, 351 pages, 693 engravings. Lea and Febiger, Philadelphia. $6.50.

A revised and enlarged edition of this excellent manual. Special features are the careful descriptions of technique, the emphasis placed upon prevention of injury to both operator and patient, and the still greater emphasis placed upon conservative interpretation of pictures to prevent the needless extraction of teeth.

MEDICAL PAPERS. DEDICATED TO HENRY ASBURY CHRISTIAN, *from His Present and Past Associates and House Officer at the Peter Bent Brigham Hospital, Boston, Massachusetts.* 1,000 pages, illustrated. Waverly Press, Inc., Baltimore. 1936.

This splendid collection of medical articles and case reports was issued in honor of Dr. Christian's sixtieth birthday on February 17, 1936. Not only the internist but the surgeon and general practitioner will enjoy and profit by browsing in this book. But there is one particular paper the reviewer wishes to draw to the attention of physicians and laymen. This is "The Case of Mrs. B." The title is exciting and sounds rather like one of Poe's tales or Conan Doyle's detective stories. Really it is a case report so full of dramatic interest, so well told that it keeps the reader's attention fixed to the end. Most of all it carries a wonderful message and inspiration to the physiciain, the nurse, and the patient.

A GUIDE TO PSYCHIATRIC NURSING, *by F. A. Carmichael, M. D., and John Chapman, M. D.* 175 pages, illustrated. Second edition. Lea and Febiger, Phila. 1936. $2.25.

New edition of an excellent guide which forms one of the nurses textbook series issued by these publishers.

HOW THE PRESIDENT, THOMAS JEFFERSON, AND DR. BENJAMIN WATERHOUSE ESTABLISHED VACCINATION AS A PUBLIC HEALTH PROCEDURE, *by Robert H. Halsey, M. D.* History of Medicine Series of the Library of the New York Academy of Medicine. Published by the author. New York. 1936.

Dr. Halsey in this little pamphlet gives us an extremely interesting account of an important piece of American medical history, the introduction of smallpox vaccination into the United States. The part played by Dr. Waterhouse and President Jefferson is well set forth and the letters and documents quoted are of the greatest interest.

DENTAL INFECTION AND SYSTEMIC DISEASE, *by R. L. Haden, M. D., Chief of the Medical Division, Cleveland Clinic, Cleveland, Ohio.* Second edition. 163 pages, 63 illustrations. Lea and Febiger, Phila. 1936. $2.50.

A study of this subject with a number of brief case histories.

SYNOPSIS OF CLINICAL LABORATORY METHODS, *by W. E. Bray, M. D., Professor of Clinical Pathology, University of Virginia.* 324 pages, 32 text illustrations, 11 color plates. C. V. Mosby Co., St. Louis. 1936. $3.75.

A very useful little laboratory manual handy in size and packed with an immense amount of information on clincial laboratory methods.

ACHIEVEMENT SCALES IN PHYSICAL EDUCATION ACTIVITIES FOR COLLEGE MEN, *by F. W. Cozens, Ph. D., Professor of Physical Education, University of California.* 118 pages. Lea and Febiger, Philadelphia, 1936. $2.50.

This little book is filled with tables which show the physical abilities of college men for certain athletic events and tests. They are grouped in various practical ways for determining the possible physical standard for any physical type and use in correction of defects.

UROLOGICAL NURSING, *by David M. Davis, M. D., Professor of genito-urinary surgery, Jefferson Medical College.* 195 pages, 67 illustrations. Second edition. W. B. Saunders Co., Philadelphia. 1936. $2.25.

An excellent little book on urological nursing.

THE DIVISION OF PREVENTIVE MEDICINE

S. S. Cook, Commander, Medical Corps, United States Navy, in charge

DEATHS FOLLOWING THE ADMINISTRATION OF ARSENICALS IN THE UNITED STATES NAVY, 1919–35

By S. S. Cook, Commander, Medical Corps, United States Navy

It is the purpose of this article to review the records of 63 deaths which have occurred in the United States Navy following the administration of arsenicals in the 17-year period 1919–35. Data will be presented with respect to age, duration of infection or syphilitic age, the amount of arsenical treatment, the type of arsenical, the principal clinical manifestations, and the postmortem findings in the 44 cases in which an autopsy was performed.

Age.—Of the 63 individuals the youngest was 18 years of age and the oldest 47 years of age. There were 42 deaths in individuals less than 30 years of age, 15 between 30 and 40, and 6 over 40.

Syphilitic age.—The duration of infection or syphilitic age ranged from 15 days to 20 years. There were 35 cases of less than 6 months' duration, 1 of 6 to 12 months, 3 of 1 to 2 years, 16 who contracted syphilis more than 2 years previously, and in 8 instances the duration of infection was not stated.

Treatment.—None of the deaths occurred after the first injection, one man had received more than one injection, the total number not being given, and 62 men received the following number of injections.

Men;	*Injections*
16	2
9	3
4	4
2	5
16	6–9
4	10–19
11	Over 20

The type of arsenical given at the final injection is known in 61 of the 63 instances. It was salvarsan in 5 cases, arsphenamine in 3, arsenobenzol in 2, neoarsphenamine in 50, and novarsenobenzol in 1 case. The preponderance of deaths from neoarsphenamine does not necessarily indicate that this arsenical is more toxic than the others. It is the only arsenical given aboard ship and at stations other than hospitals and is the principal one used at hospitals. For example,

in the 11-year period 1925–35, 994,176 injections of neoarsphenamine were given and 37,101 injections of arsphenamine.

The 63 cases have been classified, largely on the basis of postmortem findings, as:

Hemorrhagic encephalitis	24
Arsenical dermatitis	11
Blood dyscrasias	10
Vascular damage	8
Liver and kidney damage	4
Acute pancreatitis	1
Broncho-pneumonia	1
Multiple emboli	1
Unneutralized arsenobenzol	1
Cause unknown	2

Hemorrhagic encephalitis.—The designation of encephalitis is not entirely satisfactory because it implies inflammation, which is seldom present, and fails to emphasize the fact that death is due to edema and hemorrhage. However, the term is generally accepted and well understood and a change would be confusing.

The usual history is that the condition arises early in the course of treatment and without previous warning. There is nausea, vomiting, headache, and perhaps a chill with rise of temperature to 101° to 102° F. One or more of these symptoms appear in a few hours after the injection. The patient frequently recovers in 6 to 12 hours and may feel entirely well the next day. After 24 to 48 hours signs of cerebral irritation such as severe headache, nervousness, apprehension, and excitability appear to be followed by signs of rapid increase in intracranial pressure with vomiting, convulsions, paralyses, hyperpyrexia, coma, and death. In the 63 deaths from 1919 to 1935 there were 24 cases of encephalitis.

In table 1 are shown the age, the syphilitic age, the interval between treatment and onset, the symptoms, the interval between onset and death, the treatment the individual had received, and the autopsy findings.

The syphilitic age is unknown in 7 of the 24 cases. In 12 of the remaining 17 cases it was less than 3 months, in 2 others it was 4 months, and in the remaining 3 it was more than 5 years.

The interval between treatment and onset varied from 3 minutes to 4 days.

All the men died within 4 days and one man died in 45 minutes. They usually died in 24 to 48 hours.

None of the fatalities followed the first injection of an arsenical, 13 followed the second injection; 3 the third injection; none the fourth; 3 the fifth; 1 the sixth; and the remaining 4 had received several courses of treatment prior to the fatal injection.

Postmortem examinations were performed in 18 instances, in 4 of which the brain was not examined. The usual findings were either edema or hemorrhage of the brain or both. It is worthy of note that with two exceptions pathological changes similar to those found in the brain were found in other organs.

Arsenical dermatitis.—There were 11 deaths in which dermatitis was a factor. In one instance the man had acute infectious jaundice which was apparently thought to be due to syphilis as he received two injections of neoarsphenamine. Following treatment he developed exfoliative dermatitis. In the other 10 cases syphilitic infection had been recently acquired—not more than 3 months in any instance.

The interval between the last injection and the onset of dermatitis varied from 1 to 15 days, and was usually less than a week. The interval between onset and death was in five instances 1 month and in the others 15 to 20 days.

Complications were in two instances pneumonia; in another multiple abscesses of the lungs; in another congestion of the lungs and enlarged liver; in two instances secondary infection; and in the remaining five there is no record of complications. All of the men were on their first course of treatment and they all received neoarsphenamine. One man received three injections; one, four injections; three, six injections; one, seven injections; and four had completed nine injections.

Autopsies were performed in three instances with the finding of pulmonary abscesses and enlarged kidneys in one; broncho-pneumonia, enlarged liver, granular degeneration of kidneys, and punctiform hemorrhages of the stomach in another; and edema of lungs, brain, liver, and kidneys in the third.

It was noted that the man who received four injections experienced no reaction after the first three injections and a man who received nine injections experienced none after the first eight injections. One man who received six injections had slight reactions after the first, second, and fifth injections.

Blood dyscrasias.—There are 10 cases which have been classified as blood dyscrasias and are shown in table 2. Of the 10 cases, 2 were recently infected with syphilis while the remaining 8 were old cases, having acquired their infections 7 to 20 years previously.

The interval between treatment and onset could not be determined in five of the cases as symptoms developed gradually and were well advanced before they attracted attention. In the other five the interval varied from a few hours to 29 days.

Clinically, the cases that show evidence of blood destruction can usually be divided into three groups, namely, purpura, agranulocytosis, and aplastic anemia. In the purpuric group they have bleeding gums, petechiae of the skin, nose bleed, and in the severe cases bleeding from the gastrointestinal tract. The agranulocytic group usually

has extensive ulcerations of the mouth and throat and may be mistaken for septic sore throat or Vincent's infection. Those with aplastic anemia give a history of progressive weakness and pallor and often have swelling of the feet and legs.

In the less severe cases that recover the three groups may present fairly clear-cut and distinct clinical pictures. However, in the fatal cases there is overlapping with evidence of involvement of the entire hematopoietic system. For this reason it is felt that the inclusive term blood dyscrasia should be used rather than confuse the issue by the employment of terms that are probably phases or stages of one condition.

It is noted that all of the cases except one had received nine or more injections of one of the arsphenamines.

Vascular damage.—There are eight cases in this series that may be classed as examples of vascular damage. In each case there were indications of extensive damage to the vascular system of more than one organ in the body. They are shown in table 3.

It is noted that five of them had been infected more than a year and three of them for 5 months or less.

The onset of symptoms was sudden and in each case occurred less than 5 hours after treatment. Death ensued in from 45 minutes to 14 days. One man had received two injections of neoarsphenamine, another three, another four, and the remaining five, eight or more injections each. The outstanding pathologic findings were congestion, edema, and hemorrhage of various organs.

Liver and kidney damage.—There were four cases in which lesions of the liver and kidneys were the principal clinical and pathological findings. The important facts of each case are:

L. L. H., 26 years of age, with a syphilitic infection of 1 month's duration, experienced a severe reaction immediately on completion of his third injection of salvarsan. The next day he had severe abdominal pain accompanied by diarrhea and anuria. The following day he became irrational, had two convulsions, and died on June 6, 1919. At autopsy both kidneys were found to be swollen and hemorrhagic. The liver and spleen were enlarged and engorged with blood.

He had received a total of three injections of salvarsan of 0.6 gm each on May 20, 27, and June 3, 1919.

M. S. M., age 20 years, acquired syphilis 2 months previously. His reaction occurred 3 days after his sixth injection and consisted of coma and convulsions with death on February 5, 1921, 24 hours after onset of symptoms.

The postmortem findings were numerous areas of beginning necrosis in liver, spleen, and kidneys. The left adrenal gland was large and congested. There was no pathology in brain and spinal cord.

His arsenical treatment had consisted of six injections of arsphenamine between December 22, 1920, and February 1, 1921. The first injection was of 0.3 gm and the remainder of 0.6 gm each.

E. W. H., 24 years of age, with a syphilitic infection of 4 months' duration, had his treatment discontinued in August 1927 because of albumin in his urine.

He had no further symptoms until November 8 when he was treated for jaundice for 6 days. Shortly thereafter (November 21) his liver became enlarged with marked increase in the jaundice. On November 27 he vomited blood and had some speech difficulty. At this time there was an icterus index of 120 and an immediate direct Van den Bergh.

The blood chemistry findings were sugar 98 mgms per 100 cubic centimeters of blood, nonprotein nitrogen 30, urea nitrogen 13.6, and creatinine 1.21. The blood picture was —red blood count 3,930,000 hemoglobin 80 percent, white blood count 16,450, polymorphonuclears 72, lymphocytes 27, transitionals 1.

Urine—albumin 3 + with many granular casts.

Death—November 28, 1927. The postmortem findings were:

1. Intense jaundice.
2. Petechial hemorrhages on arms and trunk, along mesentery, and in kidneys.
3. Engorgement of surface vessels of the brain, no pathology found on sections of brain substance.

His treatment had consisted of four injections of neoarsphenamine and 14 injections of mercury between July 11, 1927 and August 25, 1927.

L. A. C., 21 years of age, who acquired syphilis about 1 year previously, developed jaundice shortly after his ninth injection of neoarsphenamine. He gave a history of 30 pounds loss in weight during the previous month. The liver was enlarged, there were casts in the urine, an icterus index of 210 and blood pressure 110/50.

He died on September 25, 1929, 44 days after his last injection.

Autopsy report:

1. Liver—small, firm, nodular, and grayish green in color.
2. Kidneys—slightly enlarged and greenish in color.
3. Stomach and intestines—mucosa of stomach deeply congested, a few ecchymotic patches beneath serosa of small intestines and along mesentery.
4. Histopathology—marked destruction of liver cells and swelling of tubular epithelium of kidneys.

His treatment had consisted of five injections of neoarsphenamine of 0.6 gm each between May 6, 1928 and June 12, 1928 and four injections of neoarsphenamine between July 1, 1929, and August 12, 1929.

Acute pancreatitis.—This patient, who was infected with syphilis in 1927, received 27 injections of neoarsphenamine in 1927 and 1928. He also received treatment in 1929 but the amount is not stated. The record states he had petechiae on his arms and legs in March 1929. On August 8, 1930, he received 0.3 gm of neoarsphenamine; on August 14, 0.6 gm neoarsphenamine; and on August 20, 0.4 gm neoarsphenamine. During the last injection he became nauseated and severely shocked. The next day he was jaundiced, had acetone in his urine, and his blood sugar was 323 milligrams per 100 cubic centimeters. There was progressive increase in symptoms with evidence of acute pancreatitis, and death on September 10, 1930, 21 days after the last injection.

The autopsy findings were: acute pancreatitis with abscess formation, widespread fat necrosis in the abdomen, congestion and cloudy swelling of the liver and kidneys, congestion of the meningeal vessels and atherosclerosis of the aorta.

Broncho-pneumonia.—N. M. C., who was found to have syphilis in February 1932 received injections of 0.3 gm of neoarsphenamine on February 16 and 19 and 0.6 gm on February 23 and March 1. About 5 hours after the last injection he complained of a dull headache, general aching, and had a temperature of 99° F. The next day he was acutely ill, with temperature 104.4° F. and was delirious. White blood count 13,500, polymorphonuclears 78 percent, small lymphocytes 10, large lymphocytes 8, transitionals 2, mononuclears 2. He became progressively worse and died of broncho-pneumonia 7 days after his last injection. There was no autopsy.

Multiple emboli.—P. H., a syphilitic of 2 years' duration, received 0.6 gm of arsenobenzol on September 8, 1919, without reaction. On September 15 he received 0.6 gm in 200 cubic centimeters of salt solution. Twenty-four hours later he had a temperature of 103° F. The next morning his temperature was subnormal, he was nervous and apprehensive, had pain in the epigastrium, and nose bleed. He died suddenly 48 hours after the injection, following several convulsions. Autopsy: Embolus in cephalic vein above needle puncture and several white emboli in right auricle and ventricle and in the pulmonary artery.

Unneutralized arsenobenzol.—O. B. was found to be infected with syphilis early in February 1921 and on February 8 received 0.6 gm neoarsphenamine. On March 2, 1921, he received 0.9 gm of unneutralized arsenobenzol. Immediately he became cyanotic and dyspneic and died in 1½ hours.

Autopsy findings were: Lower left lung adherent posteriorly and laterally. Atheromatous patches in aorta. Thymus gland 5 x 1 x 1½ centimeters.

Cause unknown.—L. K., a syphilitic of 1 month's duration, received 0.3 gm salvarsan on June 24, 1920, and 0.6 gm on July 1. On July 11 he received 0.75 gm novarsenobenzol. Within 2 hours he became restless, had a rapid, feeble pulse, and temperature of 101° F. He died in 12 hours. There was no autopsy. The medical officer noted that 217 doses of the same lot had been given without reaction and on the same day two other men received injections of the same dosage from the same box without reaction.

J. P., who was infected with syphilis in August 1933 received six injections of neoarsphenamine between August 15 and October 24. He received his sixth and final injection which consisted of 0.3 gm neoarsphenamine, on October 24. In 5 minutes he collapsed and died in severe shock within 10 minutes. There was no autopsy. The medical officer noted that 29 other injections of the same drug were given that morning without reaction.

Data are available for a portion of the period regarding the ratio of injections to deaths and are shown in the table below. In the 11-year period 1925–35, 1,096,220 injections of arsenicals were given and 42 deaths occurred, a ratio of 1 death to 26,100 injections. The ratio varied between 1 to 10,990 in 1927 and 1 to 64,726 in 1935, and there was one year (1931) in which no death occurred.

Ratio of deaths to injections of arsenicals of all types, 1925–35

Year	Number of injections	Deaths	Ratio, 1 to—	Year	Number of injections	Deaths	Ratio, 1 to—
1925	48,875	2	24,436	1932	138,716	4	34,679
1926	64,381	5	12,876	1933	146,629	7	20,947
1927	76,932	7	10,990	1934	126,388	3	42,129
1928	82,560	7	11,794	1935	129,453	2	64,726
1929	85,495	3	28,498				
1930	92,878	2	46,439	Total	1,096,220	42	26,100
1931	103,913	0					

In the 11-year period 1925–35 there were 14 deaths from hemorrhagic encephalitis, giving a ratio of 1 death to 78,158 injections. In the same period there were 8 deaths from arsenical dermatitis, a ratio of 1 death to 137,022 injections. There were 9 deaths from blood dyscrasias, a ratio of 1 to 121,802 injections. Vascular damage caused 6 deaths, a ratio of 1 to 182,704 injections.

OBSERVATIONS

1. In the 17-year period 1919–35, 63 persons died at naval activities following the administration of all types of organic arsenicals. All of them except two were in the Navy, one being the wife of a member of the naval service and the other a veteran. It cannot be stated that these were all the deaths that resulted from the administration of arsenicals, but this is believed to be the case, as the original records of all deaths recorded as due to arsenic poisoning, nephritis, hepatitis, and jaundice, skin diseases, all types of anemia, and syphilis have been reviewed.

2. The ages of the individuals were from 18 to 47 with the largest number, 42 or 67 percent, under 30 years of age.

3. There were four Filipinos, one negro, one woman, and the remainder white males.

4. All of them had syphilis with the possible exception of one man who was recorded as having acute infectious jaundice. He was evidently thought to have syphilis, as he had a 4 plus Kahn and was treated with arsenicals.

The duration of the syphilitic infection was quite variable, the shortest time being 20 days and the longest 20 years. Early syphilis predominated, 56 percent with a duration of less than 6 months.

5. The coexistence of other diseases appears to have been the exception rather than the rule. One man had several conditions, including involvement of the cardiovascular system; one had an early syphilitic aortitis; one man recovered from a severe blood dyscrasia induced by arsenic, had a cerebral hemorrhage, and again had a blood dyscrasia when arsenical treatment was resumed. Other than these three the records do not indicate the presence of diseases that would likely influence the outcome.

6. The symptomatology and clinical picture of arsphenamine poisoning may be more clearly understood if one has in mind the effect of arsenic on the tissues of the body.

It has been known for a long time that the principal action of arsenic is exerted on the capillaries. This may vary widely in degree from a transitory dilation or vasoparesis to extensive damage to the capillary endothelium. A phenomenon frequently observed after arsphenamine administration is the so-called nitritoid reaction, in which temporary vasoparesis gives rise to a clinical picture closely resembling shock. At the other extreme is the condition which I have characterized as vascular damage with evidence of extensive destruction of the capillary network of several organs. An example of this is case 23 who collapsed immediately upon completion of his eighth injection and died in 45 minutes. At autopsy, hemorrhages were found in all the tissues of the body.

It is evident that edema and hemorrhage are important sequelae of increased permeability of the capillaries. Failure to recognize this fact may prove serious because efforts to combat arsenic poisoning through the use of large quantities of fluids will certainly increase edema.

SUMMARY

1. In the 17-year period 1919–35 there were 63 deaths in the United States Navy attributable to arsenic poisoning. Of these, 44 were examined post mortem and the results have been presented.

2. All those who died were less than 48 years of age and 42 of them were less than 30 years of age.

3. The duration of syphilitic infection was in 35 cases less than 6 months, in 4 between 6 months and 2 years, in 16 over 2 years, and in 8 it was unknown.

4. There were no deaths after the first injection, 31 had received less than 6 injections, and 31 more than 6 injections.

5. The deaths have been classified on a clinicopathologic basis and the findings discussed.

6. In the 11-year period 1925–35 there was a ratio of 1 death to 26,100 injections of all types of arsenicals. In the same period the ratio for hemorrhagic encephalitis was 1 death to 78,158 injections, for dermatitis 1 to 137,022, for blood dyscrasias 1 to 121,802, and vascular damage 1 to 182,703.

7. The usual post-mortem findings were edema and hemorrhage in various organs.

TABLE 1.—*Hemorrhagic encephalitis, U. S. Navy, 1919–35*

Case	Age (years)	Syphilitic age	Interval between treatment and onset	Symptoms	Interval between onset and death	Date of death (year)	Autopsy findings	Previous treatment
1	20	1 month	24 hours	Dizziness, convulsions. Temperature, 101° F. Edema of eyelids. Urine, albumin, and hyaline and granular casts.	24 hours	1919	Left kidney, enlarged and soft. Heart, fibrosis with thickening of aortic valve.	Feb. 17, 1919, 0.6 g salvarsan; Feb. 27, 1919, 0.5 g salvarsan.
2	20	(?)	2 hours	Admitted with constitutional psychopathic state. Nausea and vomiting for 4 hours. Next day, convulsions.	72 hours	1920	Brain, marked edema and minute hemorrhages. Congestion of lungs, heart, spleen, and kidneys.	Aug. 25, 1920, 0.6 g salvarsan; Sept. 1, 1920, 0.6 g salvarsan.
3	19	20 days	12 hours	Temperature, 105.6° F. White blood count, 17,400. Urine, much albumin and many casts.	48 hours	1921	Liver and spleen, mottled in appearance. Marked enlargement of abdominal lymphatic glands. Superficial congestion of brain with whitish exudate along the veins between the convolutions.	July 13, 1921, 0.5 g neoarsphenamine; July 20, 1921, 0.8 g neoarsphenamine.
4	31	1 month	Immediate	Temperature, 103° F. Next day, normal. Next day, delirium and convulsions. White blood count, 15,000. Spinal fluid clear and under considerable pressure.	48 hours	1922	No autopsy	November 1921, 3 injections neoarsphenamine; Dec. 6 to Mar. 8, 5 injections neoarsphenamine.
5	28	Unknown	do	Marked prostration with recovery in 48 hours; then marked nervous symptoms, vomiting, and stupor.	96 hours	1922	Hemorrhages in spleen and liver; edema of the lungs; pus in kidney pelves; brain not examined.	Feb. 23, 1922, 0.9 g neosalvarsan; Mar. 2, 1922, 0.9 g neosalvarsan.
6	21	2 months	2 days	Nervousness, then frequent convulsions. White blood count, 16,600 with 85 percent polys. Urine, marked trace of albumin with white blood corpuscles and red blood corpuscles.	24 hours	1922	No autopsy	Aug. 21 1922, 0.45 g neoarsphenamine; Aug. 28, 1922, 0.6 g neoarsphenamine.
7	36	1 month	48 hours	Headache, temperature, 104° F., unconsciousness.	24 hours	1923	Slight general glandular enlargement and slight injection of kidneys; brain not examined.	Aug. 9, 1923, 0.45 g neosalvarsan; Aug. 16, 1923, 0.9 g neosalvarsan.
8	24	19 days	3 days	Nervous and excitable; temperature, 97° F.; pulse, 100. Next day, quiet; temperature, 99° F. Next day, unconscious, with nystagmus; temperature. 104° F. Babinski positive.	48 hours	1924	Injection and edema of brain substance, especially in region of lateral ventricles; hypostatic injection of lungs; blood cultures positive for staphylococcus aureus.	Nov. 12, 1924, 0.4 g salvarsan; Nov. 19, 1924, 0.6 g neosalvarsan.
9	23	1 month	Unknown	Temperature 104° F.; diarrhea, vomiting, partial left facial paralysis with speech interference. Left knee jerk diminished. White blood count, 14,200; urine, albumin with many white blood corpuscles and red blood corpuscles.	Unknown	1924	Mild degree of acute congestion of brain and acute congested nephritis. Spleen, twice normal size with acute congestion.	May 28 to June 5, 1924, 3 injections neoarsphenamine.

TABLE 1.—*Hemorrhagic encephalitis, U. S. Navy, 1919–35*—Continued

Case	Age (years)	Syphilitic age	Interval between treatment and onset	Symptoms	Interval between onset and death	Date of death (year)	Autopsy findings	Previous treatment
10	20	1 month	18 hours	Malaise and vomiting, temperature 102° F., pulse 100. That night, moaning, unconscious, pupils sluggish to light. Next morning, convulsions.	24 hours	1925	No autopsy	Apr. 21, 1925, 0.9 g neoarsphenamine; Apr. 28, 1925, 0.9 g neoarsphenamine.
11	32	Unknown	Immediate	Chill, temperature 100.6° F. Recovery in 2 hours with malaise, and weakness 3 days later; then convulsions and stupor.	2 days	1925	do	Unknown amount several years before. Oct. 11, 1925, 0.9 g neoarsphenamine; Oct. 24, 1925, 0.9 g neoarsphenamine.
12	29	do	24 hours	Convulsions. Spinal fluid, clear with no increase in pressure. Luetic curve. Kahn 2 plus. Urine, positive for albumin with occasional white blood corpuscles and red blood corpuscles. Blood chemistry, n. p. n. 50, sugar 40, urea 23. Blood pressure 240/120.	24 hours	1926	Brain, weight 1,425 g. Injection of the superficial vessels of the cortex of the cerebrum. Heart, hypertrophied, auricles distended with blood, weight 390 g. Lungs, red hepatization lower right lobe. Liver, passive congestion left lobe. Kidneys, blue in color with mottling of cut surfaces.	Apr. 1 to May 3, 1926, 5 injections neoarsphenamine, the last of 0.75 g.
13	27	1 month	48 hours	Abdominal cramps and vomiting, white blood count, 8,600. Next day, repeated convulsions. Spinal fluid, clear, no increase in pressure.	48 hours	1926	Hemorrhages in brain, liver, spleen, and kidneys. Weight: Brain 1,500 g, liver 1,700 g, spleen 300 g, and kidneys 200 g each.	October 1926, 3 injections neoarsphenamine—0.45, 0.6, and 0.9 g.
14	21	18 days	6 hours	Temperature 100° F. Much better next morning. In 36 hours backache, nervousness, temperature 101° F. Convulsions later in day. Next day, generalized petechial eruptions. Spinal fluid, increased pressure and globulin.	4 days	1926	Edema of brain with marked injection of cerebral capillaries.	Apr. 20, 1926, 0.45 g neosalvarsan; Apr. 27, 1926, 0.6 g neosalvarsan.
15	22	15 days	12 hours	Coma and convulsions. Spinal fluid, slight increase in pressure. Blood pressure 160/90. Urine, much albumin with many granular casts and red blood corpuscles.	72 hours	1926	Edema and congestion of brain, lungs, and kidneys. Healed pulmonary tuberculosis.	Feb. 5, 1926, 0.45 g neosalvarsan; Feb. 9, 1926, 0.65 g slavarsan; Feb. 16, 1926, 0.9 g neosalvarsan.
16	40	Unknown	do	Admitted with psychosis, Kahn 4 plus. After treatment, flushed face, temperature 103° F. Next day, patient feels well. At 6:30 p. m. vomiting, weakness, and shock.	36 hours	1927	Intense congestion of cerebral meninges with a serofibrinous exuate.	Mar. 23, 1927, 0.3 g salvarsan; Mar. 30, 1927, 0.6 g salvarsan.

17	26	4 months	48 hours	Dizziness and backache. Temperature 101° F., pulse 104. Spinal fluid, cloudy and under great pressure. Convulsions.	12 hours	1927	Wet brain with hemorrhages in both internal capsules. Marked edema of lungs, liver, and kidneys. Petechiae in mucosa of small intestines.	Nov. 29, 1927, 0.45 g neosalvarsan; Dec. 8, 1927, 0.6 g neosalvarsan.
18	28	3 months	2 hours	Temperature 107° F., pulse 120, twitching of muscles. Urine, albumin and casts.	36 hours	1927	No autopsy	July 26, 1927, 1 injection (type ?); Aug. 4, 1927, 0.9 g neoarsphenamine.
19	28	5 years	4 days	Headache, dizziness, and backache. Urine, trace of albumin and few white blood corpuscles and red blood corpuscles. White blood count 16,000. Later, convulsions, cyanosis, and unconsciousness. Spinal fluid, low pressure.	48 hours	1928	Hemorrhages in epicardium and endocardium. Marked congestion of kidneys. Multiple hemorrhages in brain with focal necrosis.	From 1923 to 1926, 24 injections salvarsan and neoarsphenamine; June 12 to Aug. 2, 1928, 8 injections neoarsphenamine.
20	38	Unknown	3 minutes	Dyspnea and cyanosis	45 minutes	1928	Marked edema of lungs and brain and flabby heart muscles. Kidneys, normal.	Sept. 29 to Nov. 10, 1928, 6 injections neoarsphenamine. First, 0.25 g; others, 0.9 g.
21	31	11 years	1½ hours	Unconsciousness and shock	7 hours	1932	Hemorrhagic encephalitis. Edema of lungs. Ac. degeneration of liver and kidneys. Hypoplastic bone marrow.	From 1921 to 1932, 65 injections arsenicals. Last injection, 0.2 g.
22	30	10 years	6 hours	Chill, fever, temperature 100° F. Increased intracranial pressure. Blood sugar, 214.	4 days	1932	Edema of brain with small hemorrhagic areas over the hemispheres. Hyperemia of liver, spleen, and kidneys.	From 1922 to 1930, 60 injections arsenicals. Mar. 14 to Nov. 11, 1932, 19 injections of neoarsphenamine.
24	24	Unknown	3 minutes	Nausea, dizziness, convulsions. White blood count 28,000. Juveniles 2, band forms 12, segs. 64, lymphs 14, eos. 8.	6 hours	1934	Congestion and edema of brain and lungs. Congestion of spleen and kidneys. Submucous petechiae of small intestines. Chr. fibrous pleuritis.	Apr. 14 to May 12, 1934, 5 injections neoarsphenamine, the last of 0.6 g.
25	35	3 years	Immediate	Slight reaction. Next day, headache. Next day, worried, nervous, and confused. Temperature 99.5° F. Next day, stuporous, dilated pupils, white blood count 3,300, blood pressure 118/75. Next day, Cheyne Stokes resp. Urine, albumin with hyaline and granular casts. Blood Wassermann, 4 plus; spinal fluid Wassermann, negative. Convulsions.	4 days	1920	Enlarged kidneys with small punctate hemorrhages. Spleen greatly enlarged. Liver dark and soft. Brain not examined.	History of considerable treatment in 1917. On Oct. 26, 1920, 0.6 g neoarsphenamine with slight reaction. On Oct. 31, 0.6 g neoarsphenamine.

NOTE.—g=grams.

TABLE 2.—*Blood dyscrasias, U. S. Navy, 1919–35*

Case	Age (years)	Syphilitic age	Interval between treatment and onset	Symptoms	Interval between onset and death	Date of death (year)	Autopsy findings	Previous treatment
39	33	11 years	Unknown	Salivation, bleeding gums. Red blood count 3,960,000; hemoglobin 85 percent, polys 58, lymphs 35. Blood pressure 104/56. Continuous bleeding from gums and finally extensive gastrointestinal hemorrhages.	About 1 month	1922	Autopsy refused by relatives	1911, 1 injection salvarsan; Feb. 10, to Apr. 10, 1922, 8 injections neosalvarsan.
40	36	do	Few hours	Vomiting and headache. Red blood count 4,500,000; white blood count 19,800. Hemoglobin 85 percent, polys 40, lymphs 29, monos 1. 5 days later, marked jaundice with generalized macular pruritic rash. 11 days after onset, nasal hemorrhages, red blood count 2,500,000, white blood count 6,000, hemoglobin 60 percent, polys 11, lymphs 26, trans 1, mast cells 2.	13 days	1926	Liver, markedly enlarged and friable with nutmeg appearance. Intense congestion of myocardium and pericardium.	July 15, 1926, 1 injection neoarsphenamine (amount not stated), severe reaction; July 22, 1926, 0.45 neoarsphenamine.
41	42	20 years	Unknown	Weakness, pains in joints, jaundice, anemia, ulcerated and bleeding gums. Positive Babinski. Red blood count 1,080,000, white blood count 1700, polys 25; lymphs 75. No platelets or reticulocytes seen.	(?)	1927	Subcutaneous emphysema due to gas bacillus infection.	Feb. 24 to Aug. 4, 1927, 16 injections neoarsphenamine.
44	27	7 years	do	Marked weakness with generalized petechial rash. Red blood count 800,000, hemoglobin 20 percent; white blood count 25,000, polys 74, coagulation time 3 minutes, bleeding time 18 minutes.	Unknown	1928	No autopsy	In 1924 and 1925, 10 injections, neoarsphenamine; November 1926, 3 injections neoarsphenamine; Apr. 2 to May 21, 1927, 8 injections neoarsphenamine; record of recent treatment (amounts not given).
45	40	19 years	15 days	Malaise, nausea, and vomiting. Slight jaundice. Red blood count 730,000, white blood count 4,900. No reticulocytes.	About 1 month	1928	Abdominal incision only. Slight enlargement of spleen and bright red color of muscles.	May 8 to Sept. 15, 1928, 10 injections (amount and type not given).

46	24	10 months	Unknown	Bleeding gums, enlarged liver, red blood count 1,540,000, hemoglobin 45 percent, white blood count 2,000, polys 13, lymphs 87, platelets none. Slight response to transfusion, then multiple petechial hemorrhages. Red blood count 1,500,000, hemoglobin 30 percent.	About 3 weeks.	1929	Aplastic anemia. Hemorrhagic broncho-pneumonia.	1928, 12 injections neoarsphenamine, 0.45 g each; Feb. 6 to July, 22, 1929, 14 injections neoarsphenamine, the last of 0.45 g.
47	41	17 years	do	Remarkable case. Macular rash in 1925, purpura in 1927. In February 1928 petechial hemorrhages, enlarged spleen and liver, red blood count 1,520,000, hemoglobin 35 percent, white blood count 3,300, polys 15, lymphs 82, platelets 36,480. 9 transfusions up to July 25, 1928, with apparent recovery. January 1929, cerebral hemorrhage with paralysis of left side. Purpura followed treatment in April and May with death of terminal pneumonia on June 1.	Unknown	1929	Cerebral softening from previous hemorrhage, profound secondary anemia, hepatic cirrhosis, and nephrosis.	December 1912, 1 injection salvarsan; 1915, 8 injections neosalvarsan; 1924, 13 injections neosalvarsan; 1927, 4 injections salvarsan; Apr. 13 to May 11, 1929, 5 injections neosalvarsan.
49	22	3 months	48 hours	Itching, purpuric spots, absence of platelets and granular cells, hematuria, epistaxis, high fever, and terminal pneumonia.	14 days	1930	Hemorrhages in heart, kidneys, bladder, and intestines. Hypoplasia of bone marrow and edema of lungs.	Nov. 7 to Dec. 27, 1929, 8 injections neoarsphenamine, total 5.4 g; Feb. 6 to Feb. 27, 1930, 4 injections neoarsphenamine; total 2.1 g.
50	34	10 years	29 days	Progressive weakness, edema of legs, hemorrhages into skin and retina. Progressive anemia.	Unknown	1934	Numerous petechiae in skin. 4 liters of bloody fluid in left pleural cavity. Hemorrhages into heart muscles and right kidney. Bone marrow of ribs and sternum, pale, static, and acellular. Parenchymatous degeneration of liver, kidneys, and adrenals.	1924, 6 injections salvarsan; 1925, 4 injections neosalvarsan; 1926, 2 injections neosalvarsan; 1933, 8 injections neosalvarsan; Feb. 3 to Apr. 7, 1934, 8 injections neoarsphenamine, total 3.55 g.
51	28	3 months	4 days	Acutely ill and depressed, temperature 102° F. White blood count 3,200, bands 4, segs 46, lymphs 42, monos 8. Nose bleed, painful glands in neck. Extensive ulcerations of throat, jaundice. Urine, 6 plus albumin.	9 days	1935	Pneumonia left lower lobe, hypertrophy and fatty infiltration of liver, passive congestion of spleen, parenchymatous nephritis, jaundice, and ulcerations of throat and mouth.	Oct. 19 to Nov. 13, 1934, 2.1 g neoarsphenamine; Dec. 5, 1934, to Jan. 29, 1935, 8 injections neoarsphenamine; total 4.5 g.

TABLE 3.—*Vascular damage, U. S. Navy, 1919–35*

Case	Age (years)	Syphilitic age	Interval between treatment and onset	Symptoms	Interval between onset and death	Date of death (year)	Autopsy findings	Previous treatment
29	32	11 years	1 hour	Violent pains in lumbar region, severe shock, blood pressure 70/50. Good response to 500 cc blood transfusion. Next day, 60 cc dark red urine. Petechial hemorrhages in skin.	9 days	1933	Hemorrhages in myocardium. Right side of heart greatly dilated, slight edema of lungs, congestion of liver and kidneys.	1921, 8 injections neoarsphenamine; 1922, 8 injections neoarsphenamine; 1925, 6 injections neoarsphenamine; July 11, 1933, 0.3 g neoarsphenamine; July 18, 1933, 0.6 g neoarsphenamine.
30	29	6 years	do	Severe pain in back and legs, vomiting. Temperature 102° F., bloody urine. Next day, slight jaundice with vomiting and nose bleed. After 4 days persistent nausea and vomiting, blood chemistry—urea N, 100, creatinine 7.5, icterus index 20. Death in 12 days after stormy course with diminished urine, severe vomiting, and backache.	12 days	1933	Liver, small with nutmeg mottling. Cut surface of lungs striped with dark red blood. Small hemorrhagic area in the lower pole of the kidneys; both kidneys enlarged and pale.	1927 to 1932, 41 injections neoarshpenamine; Nov. 25, to Dec. 9, 1933, 4 injections neoarsphenamine, the last of 0.6 g.
31	26	1½ years	Immediate	Vomiting, cramps, shock, and severe pain over both kidneys. Temperature 100° F., pulse 118, blood pressure 106/68. In afternoon voided 200 cc of bloody urine. Next morning, 90 cc. of reddish black urine. Hemorrhages about teeth. Blood pressure 92/48. Late in afternoon sudden tingling of lower legs and feet; collapse; and terminal paralysis of both legs.	36 hours	1935	No autopsy	May to September 1933, 5 injections neoarsphenamine and 10 injections sulpharsphenamine; Apr. 9 to Sept. 10, 1935, 18 injections silver salvarsan, 3.5 g; Dec. 17, 1935, 0.3 g neoarsphenamine.
33	43	14 years	1 hour	Nausea, vomiting, pain in abdomen and back. Temperature 102.5° F. Bloody urine that night. Next day, intense icterus, blood pressure 90/70, fluid intake 1,500 cc, output 55 cc. Next day, hiccoughs, 35 cc of bloody urine. Next day, nose bleed, severe pain in upper abdomen and chest. Next day, blood in stools, urine, and from nose.	5 days	1933	Acute yellow atrophy of the liver, acute parenchymatous nephritis, marked edema of both lungs with much fluid in thoracic cavity. Parenchymatous degeneration of heart, spleen, and adrenals.	Feb. 14, 1933, 0.3 g neoarsphenamine; Feb. 21, 1933, 0.6 g neoarsphenamine; Feb. 28, 1933, 0.6 g neoarsphenamine; Mar. 7, 1933, 0.6 g neoarsphenamine.

26	29	3½ years	5 hours	Nausea, vomiting, dilated pupils and thready pulse.	6 hours	1924	Edema and congestion of brain; congestion of kidney, spleen, liver, and lungs; and infarct of spleen.	May 13 to Nov. 30, 1921. 7 injections arsenicals; Oct. 7, 1924, 0.45 g neoarsphenamine; Oct. 14, 1924, 0.9 g neoarsphenamine; Oct. 21, 1924, 0.9 g neoarsphenamine.
38	22	5 months	Few hours	Shock. Next day, purpuric spots on arms and legs with marked shock. 2 days later, marked jaundice, urine 1,500 to 2,500 cc. In 8 days hemorrhages from nose, bowels, and kidneys.	14 days	1921	Marked jaundice, 150 cc. of bloody fluid in pleural cavity, hemorrhages in perirenal fat of both kidneys and in liver, spleen, pancreas, and intestinal mucosa.	May 17, 1921, 0.3 g. arsphenamine; May 24, 1921, 0.4 g arsphenamine.
42	29	1 month	2 hours	Flushed skin, temperature 103° F., and sore throat. Vomited blood during the night and had blood in the stool. Next day, lividity of dependent portions of body.	31 hours	1927	Brain, negative; lungs, congested; many petechial hemorrhages in stomach and small intestines; kidneys and liver, pale.	Nov. 7, 1927, 0.45 g salvarsan; Nov. 10, 1927, 0.3 g salvarsan; Nov. 14, 1927, 0.45 g salvarsan.
23	35	4 months	5 minutes	Collapse with severe shock and death in 45 minutes.	45 minutes	1933	Marked congestion and hemorrhagic infiltration of all tissues, especially lungs, endocardium, kidney, pancreas, and meninges.	Nov. 23, 1932, to Jan. 10, 1933, 6 injections neoarsphenamine; Mar. 4, 1933, 0.3 g neoarsphenamine; Mar. 11, 1933, 0.45 g neoarsphenamine.

NOTE.—g=grams.

HEALTH OF THE NAVY

Based on returns for diseases and injuries occurring in October, November, and December 1935, the rate for all causes, entire Navy, was 470 per 1,000 per annum. The rate for the corresponding quarter of 1934 was 622 per 1,000 and the median rate for the preceding 5 years, 557.

The admission rate for diseases was 409 per 1,000 per annum, which is 9.65 percent higher than the rate for the preceding quarter but 18 53 per cent lower than the expected rate for the fourth quarter, 502.

Except when influenced by unusual accidents the rate for accidental injuries varies little from quarter to quarter, approximating 60 per 1,000 per annum as a general rule.

The admission rate for communicable diseases transmissible by oral and nasal discharges, entire Navy, was 134.09 per 1,000 per annum, as compared with 126.85 the expected rate. This increase was evident both ashore and afloat.

A decrease of 43 percent was noted in the rate for venereal diseases for the quarter when compared with 126.85, the median for the 5 preceding years.

A total of 1,732 cases of acute respiratory diseases was reported from shore stations in the United States during the quarter, 1,375 of which were admissions for catarrhal fever. The stations recording the greatest number of admissions are shown in the following tabulation:

Station	October	November	December
Naval Academy, Annapolis, Md. (midshipmen)	25	37	32
Marine barracks, Quantico, Va	50	29	44
Naval training station, Newport, R. I	46	52	72
Naval training station, Norfolk, Va	221	140	276
Naval training station, Great Lakes, Ill	13	23	15
Marine Corps base, San Diego, Calif	28	21	17
Naval training station, San Diego, Calif	61	57	100
Fleet air base, Pearl Harbor, T. H	15	19	8

The medical officer of the naval air station, San Diego, Calif., states in his December sanitary report that "The incidence of communicable diseases among service personnel continues to be quite low. The morbidity report of the city and county of San Diego shows a moderate number of cases of scarlet fever and varicella in the surrounding communities."

One case of chickenpox was reported in November by the receiving station, Norfolk, Va.

Two cases of cerebrospinal fever were admitted at shore stations in December. An apprentice seaman, 18 years of age, with 3 months' service, was admitted to the sick list at the naval training station, San Diego, Calif., on December 16 and transferred to hospital the same day. The case remained on the sick list at the end of the

year. The other case was reported by the naval torpedo station, Newport, R. I. The patient, a seaman, first class, 36 years of age, with 7½ years' service, became ill very suddenly on December 3 while on duty on the torpedo testing barge *No. 4*, moored off Gould Island. He was admitted to the sick list and died the same day. The sanitary report from the naval torpedo station for the month of December contained the following statement: "There are about 50 officers and men attached to barge *No. 4*. Close personal contacts were isolated for close observation and all members of the crew were inspected daily; contacts with the torpedo station were limited to military necessity. No second case has developed during a period of 4 weeks."

Meningitis, cerebrospinal, was recorded as the secondary cause of death in two cases of fracture, compound, skull. A seaman, first class, 24 years of age, with 5$^{8}/_{12}$ years' service, attached to the naval air station, Anacostia, D. C., was admitted to the sick list at the Naval Hospital, Washington, D. C. on October 26, 1935, following an automobile accident. On November 11, 1935, the patient developed definite clinical signs of a severe meningitis and died the following day. A private, United States Marine Corps, 25 years of age, with 5$^{1}/_{12}$ years' service, attached to the Navy mine depot, Yorktown, Va., was admitted to a civilian hospital on November 4, 1935, following an automobile accident. He developed a pneumococcic meningitis on November 9 and died at the Norfolk Naval Hospital, Portsmouth Va., on November 10.

In the sanitary report from the Naval Academy, Annapolis, Md., for the month of December the senior medical officer reports "The general health of the regiment has been good. Fifty-five cases of gastroenteritis occurred in the regiment for the period December 2 to 9, inclusive. After careful investigation and study of all factors, the senior medical officer concluded that these cases were probably a type of intestinal influenza or due to a streptococcic sore throat, as a mild epidemic of acute pharyngitis was prevalent in the community at the time."

Shore stations outside the continental limits of the United States reported 106 cases of acute respiratory diseases for the quarter as follows: Marine detachment, American Embassy, Peiping, China, 49; Fourth Marines, Shanghai, China, 45; naval station, Guantanamo Cuba, 8; and naval station, Guam, and naval station, Tutuila, Samoa, 2 each. The annual rate per 1,000 per annum for these diseases was 173.56 for the fourth quarter of 1935 as compared with 171.04, the rate for the corresponding quarter of 1934.

In addition to the acute respiratory diseases, one case of chickenpox was reported by the naval station, Guam, in October and two cases

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of scarlet fever by the Fourth Marines, Shanghai, China, one in October and one in November.

The admission rate, all causes, for forces afloat was 457 per 1,000 per annum, a 22 percent decrease when compared with the fourth quarter of 1934. The median rate for the fourth quarter of the preceding 5 years is 524.

The admission rate for diseases was slightly greater than the rate for the previous quarter but showed reductions of 24 percent and 16 percent respectively when compared with the rate for the corresponding quarter of 1934 and the median rate for the preceding 5 years.

The fleet medical officer in his annual sanitary report for the calendar year 1935 has this to say regarding motor vehicle casualties: "The mounting number of fleet personnel killed and maimed in motor vehicle accidents parallels with relentless accuracy the rate in civil life. One out of every 20 admissions to the hospital ship in 1935 was for injuries incurred in traffic accidents. Of the 94 patients, 4 died. The situation constitutes a problem for which no solution has been found."

There were 1,883 cases of acute respiratory diseases reported by all ships of the Navy during October, November, and December, 1935, indicating a 59 percent increase from the number of cases notified for the preceding quarter. The admission rate for the quarter, 118 per 1,000 per annum, was considerably lower than the rate for the corresponding quarter of 1934 but slightly greater than the expected rate.

The ships notifying 40 or more cases of acute respiratory infections distributed over the quarter are as follows:

Ship	October	November	December	Total
U. S. S. California	33	14	9	56
U. S. S. Henderson	6	15	20	41
U. S. S. Idaho	14	15	15	44
U. S. S. Nevada	24	28	15	67
U. S. S. New Mexico	17	18	11	46
U. S. S. Pennsylvania	26	16	12	54
U. S. S. Saratoga	22	44	53	119
U. S. S. Whitney	8	20	27	55
Total	150	170	162	482

Catarrhal fever was responsible for all but 15 of the 482 admissions recorded above.

The admission rate for venereal diseases, forces afloat, for the fourth quarter was 93 per 1,000 per annum, indicating a 33 percent decrease when compared with the corresponding quarter of 1934. The Medical Bulletin of the Battle Force for the month of January 1936 states that "the trend of the Battle Force for the past 4 years has been toward a decline of annual venereal rates—most marked in 1935. The rates in general, however, still remain excessively high."

The U. S. S. *Mississippi* reported 33 cases of mumps for the quarter. A few sporadic cases occurred during the June to September overhaul period and reached moderate epidemic proportions during the following months.

Eight cases of chickenpox were reported for the quarter as follows: In October, one from the U. S. S. *Saratoga* (Fleet Air Detachment, San Diego, Calif.); in November, one each from the U. S. S. *Melville* and the U. S. S. *Wright;* and in December, one each from the U. S. S. *Dobbin*, U. S. S. *Farragut*, U. S. S. *Trenton*, U. S. S. *Vestal*, and the U. S. S. *Mississippi.*

Four cases of cerebrospinal fever and one case of meningitis, cerebrospinal were reported from forces afloat as follows:

Rate	Age	Place of original admission	Date of admission	Length of service (years)	Disposition
Seaman, second-class	24	U. S. S. Bobolink	Nov. 15, 1935	4 1/12	Duty Jan. 14, 1936.
Do	19	U. S. S. Tennessee	Nov. 29, 1935	1 5/12	Do.
Coxswain	23	U. S. S. Pennsylvania	Dec. 5, 1935	3 2/12	Died Dec. 19, 1935.
Do	24	U. S. S. Ellis	Dec. 17, 1935	6 9/12	Continued.
Boatswain's mate, second class.	34	U. S. S. Kanawha [1]	Dec. 30, 1935	16 9/12	Died Dec. 20, 1935.

[1] Septicemia was recorded as the primary cause of death and meningitis, cerebrospinal, as the secondary cause.

One case of scarlet fever was reported in November by the U. S. S. *Hovey.*

A mild case of typhoid fever was readmitted to the U. S. S. *Relief* from the U. S. S. *Indianapolis* on December 24, 1935. The probable place of infection was reported as "San Pedro-Long Beach area." A complete course of typhoid prophylaxis had been administered in June 1934. Two moderately severe cases of paratyphoid B occurred during the quarter, one in October on board the U. S. S. *John D. Ford* and one in November on board the U. S. S. *Canopus.* The patient admitted to the sick list on the U. S. S. *John D. Ford* (a chief pharmacist's mate with 16 years' service) stated that he had received no typhoid inoculation since 1924 when triple vaccine was administered. Cholecystitis, chronic, was reported as the complication in this case and Hong Kong, China, as the probable place of infection. A complete course of straight typhoid vaccine had been administered in July 1934 to the patient admitted to the sick list on the U. S. S. *Canopus.* No complication was reported in this case and Shanghai, China, was recorded as the probable place of infection. The U. S. S. *Sacramento* reported one case of paratyphoid A in October. No questionnaire has been received in the Bureau for this case, consequently information regarding prophylaxis, etc., is not available.

TABLE No. 1.—*Summary of morbidity in the United States Navy for the quarter ended Dec. 31, 1935*

	Forces afloat, 72,686[1]		Forces ashore, 43,269[1]		Entire Navy, 115,955[1]	
	Admissions	Rate per 1,000	Admissions	Rate per 1,000	Admissions	Rate per 1,000
All causes	8,312	457.42	5,308	490.70	13,620	469.84
Disease only	7,227	397.71	4,616	426.73	11,843	408.54
Injuries	1,080	59.43	684	63.23	1,764	60.85
Poisonings	5	.28	8	.74	13	.45
Communicable diseases transmissible by oral and nasal discharges (class VIII):						
(A)	151	8.31	95	8.78	246	8.49
(B)	2,010	110.61	1,631	150.78	3,641	125.60
Venereal diseases	1,693	93.17	426	39.38	2,119	73.10

[1] Average strength.

Deaths reported, entire Navy, during the quarter ended Dec. 31, 1935

Cause		Navy			Marine Corps		Nurse Corps	Total
Primary	Secondary or contributory	Officers	Midshipmen	Men	Officers	Men		
Average strength		9,665	2,019	86,692	1,238	15,993	348	115,955
DISEASE								
Angina pectoris	None					1		1
Appendicitis, acute	Obstruction, intestinal, from external causes.			1				1
Carcinoma, intestines	Dysentery, amoebic			1				1
Cellulitis, arm	Septicemia			1				1
Cerebrospinal fever	None			2				2
Endocarditis, acute	do			1				1
Leukemia	Septicemia			1				1
Myocarditis, chronic	Embolism, cerebral			2				2
Nephritis, chronic	None			1				1
Do	Arterial hypertension			2				2
Obstruction, intestinal, from external causes.	None			1				1
Pneumonia, broncho	do			2				2
Pneumonia, lobar	Abscess, lung			2				2
Septicemia	Meningitis, cerebrospinal.			1				1
Syphilis	Poisoning, neoarsphenamine.			1		1		2
Tonsillitis, acute	Abscess, lungs			1				1
Do	Septicemia			1				1
Thrombosis, coronary	None			1		1		2
Do	Myocarditis, chronic	1						1
Tuberculosis, pulmonary, chronic.	None					1		1
Tuberculosis, pulmonary, acute general miliary.	do			1				1
Tumor, malignant, mixed, adeno-carcinoma.	do	1		1				2
Teratoma, testicle	do			1				1
Total for disease		2		25		4		31
INJURIES AND POISONINGS								
Asphyxiation, smoke inhaled.	Alcoholism, acute			1				1
Burn, multiple	None			1	1	1		3
Drowning	do	1		4		1		6
Do	Thrombosis, coronary			1				1
Fracture, compound, skull	None	1						1
Do	Injury, multiple, extreme.			1				1
Do	Meningitis, cerebrospinal, acute.			1		1		2
Fracture, simple, skull	None	1						1
Do	Intracranial injury			3				3

Deaths reported, entire Navy, during the quarter ended Dec. 31, 1935—Continued

Cause		Navy			Marine Corps		Nurse Corps	Total
Primary	Secondary or contributory	Officers	Midshipmen	Men	Officers	Men		
INJURIES AND POISONINGS—continued								
Injuries, multiple, extreme.	None	1		7	2	1		11
Intracranial injury	do			3		1		4
Do	Pneumonia, broncho			1				1
Rupture, traumatic, lungs	None			1				1
Strangulation, neck	do			1				1
Wound, gunshot:								
Abdomen	do			1				1
Abdomen, chest, and forearm.	do			1				1
Head	do			1				1
Wound, punctured: Axillary vein.	do			1				1
Poisoning, acute:								
Cyanide	do			1				1
Strychnine	do					1		1
Total for injuries and poisonings.		4		30	3	6		43
Grand total		6		55	3	10		74
Annual death rate per 1,000:								
All causes		2.48		2.54	9.69	2.50		2.55
Disease only		83		1.16		1.00		1.07
Drowning		.41		.23		.25		.24
Poisoning				.05		.25		.07
Other injuries		1.24		.11	9.69	1.00		1.17

ADMISSIONS FOR INJURIES AND POISONINGS, FOURTH QUARTER, 1935

The following table, indicating the frequency of occurrence of accidental injuries and poisonings in the Navy during the fourth quarter, 1935, is based upon all form F cards covering admission in those months which have reached the Bureau:

	Admissions, October, November, and December 1935	Admission rate per 100,000, per annum	Admission rate per 100,000, year 1934
Injuries:			
Connected with work or drill	721	2,487	2,397
Occurring within command but not associated with work	588	2,028	2,064
Incurred on leave or liberty or while absent without leave	455	1,570	1,699
All injuries	1,764	6,085	6,160
Poisonings:			
Industrial poisoning	6	21	15
Occurring within command but not connected with work	3	10	244
Associated with leave, liberty, or absence without leave	4	14	15
Poisonings, all forms	13	45	273
Total injuries and poisonings	1,777	6,130	6,433

Percentage relationships

	Occurring within command				Occurring outside command	
	Connected with the performance of work, drill, etc.		Not connected with work or prescribed duty		Leave, liberty, or A. W. O. L.	
	October, November, and December, 1935	Year 1934	October, November, and December, 1935	Year 1934	October, November, and December, 1935	Year 1934
Percent of all injuries	40.9	38.9	33.3	33.5	25.8	27.6
Percent of all poisonings	46.2	5.3	23.1	89.3	30.7	5.3
Percent of total admissions, injury, and poisoning titles	40.9	37.5	33.3	35.9	25.8	26.6

Poisoning by a narcotic drug or by ethyl alcohol is recorded under the title "drug addiction" or "alcoholism", as the case may be. Such cases are not included in the above figures.

There were no cases during the third quarter of 1935 worthy of notice from the standpoint of accident prevention.

STATISTICS RELATIVE TO MENTAL AND PHYSICAL QUALIFICATIONS OF RECRUITS

The following statistics were taken from monthly sanitary reports submitted by naval training stations:

October, November, and December, 1935	United States Naval Training Station			
	Norfolk, Va.	Newport, R. I.[1]	Great Lakes, Ill.	San Diego, Calif.
Recruits received during the period	1,604	210	750	1,528
Recruits appearing before Board of Medical Survey	3	0	1	0
Recruits recommended for discharge from the service	3	0	1	0
Recruits discharged by reason of Medical Survey	3	0	3	0
Recruits held over pending further observation	0	0	0	0
Recruits transferred to the hospital for treatment, operation, or further observation for conditions existing prior to enlistment	36	12	10	29

[1] For December. Data for October and November not included in sanitary reports.

The following table was prepared from reports of medical surveys in which disabilities or disease causing the surveys were noted as existing prior to enlistment. With certain diseases, survey followed enlistment so rapidly that it would seem that many might have been eliminated in the recruiting office.

Cause of survey	Number of surveys	Cause of survey	Number of surveys
Abscess, acquired, teeth	1	Enuresis	8
Adhesions, abdominal	1	Epilepsy	2
Amblyopia	1	Fistula thyroglossal	1
Anomaly of form, lumbo-sacral spine	1	Flat foot	7
Arterial hypertension	8	Hydrocele	1
Astigmatism	2	Malformation, congenital, both feet	1
Bronchitis, chronic	1	Meningitis, chronic	1
Caries, teeth	3	Neuritis, sciatic nerve, right	1
Cicatrix, lower third left shin	1	Perforated nasal septum	1
Constitutional psychopathic inferiority, without psychosis	2	Pneumoconiosis	1
Constitutional psychopathic state, emotional instability	1	Prolapse, rectum	1
Constitutional psychopathic state, inadequate personality	3	Psychoneurosis, hysteria	3
Cyst, thyroglossal	1	Psychoneurosis, neurasthenia	5
Cyst, sacro-coccygeal	1	Psychoneurosis, unclassified	1
Deformity, acquired (shortening, right leg)	1	Sinusitis, maxillary, chronic	1
Dementia praecox	2	Sprain, right sacro-iliac joint	1
Dislocation, articular cartilage, right knee	1	Stricture, ureter	1
Effort syndrome	1	Syphilis	4
		Ulcer, duodenum	2
		Valvular heart disease, aortic insufficiency	1
		Valvular heart disease, mitral insufficiency	2

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PUBLISHED *for the* INFORMATION OF MEDICAL DEPARTMENT *of the* NAVY

THE MISSION OF THE MEDICAL CORPS OF THE NAVY

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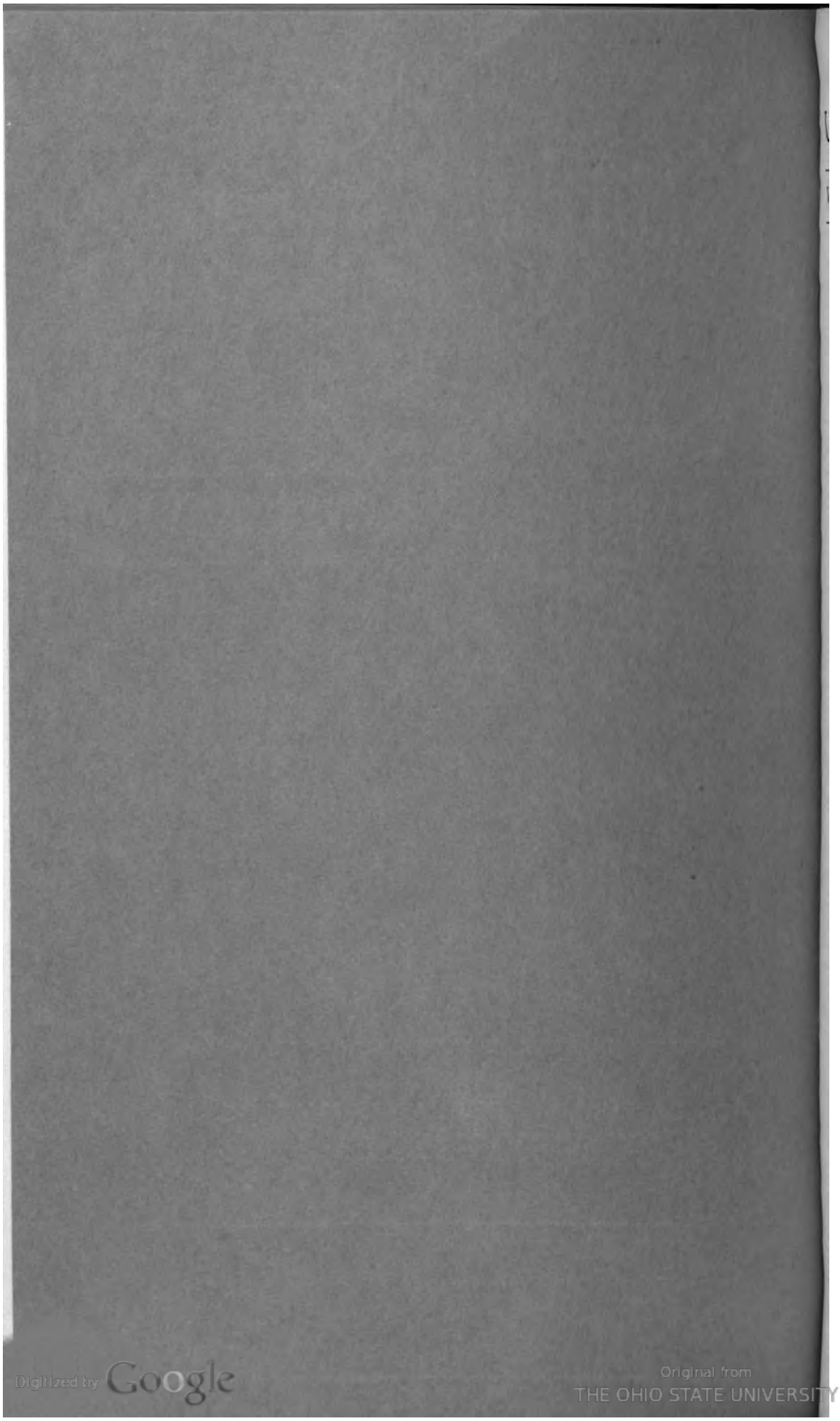
TO KEEP AS MANY MEN AT AS MANY GUNS AS MANY DAYS AS POSSIBLE

Issued Quarterly by the Bureau of Medicine and Surgery
Washington, D. C.



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NAVY DEPARTMENT,
Washington, March 20, 1907.

This UNITED STATES NAVAL MEDICAL BULLETIN is published by direction of the Department for the timely information of the Medical and Hospital Corps of the Navy.

TRUMAN H. NEWBERRY,
Acting Secretary.

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PREFACE

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It is the aim of the Bureau of Medicine and Surgery to furnish in each issue special articles relating to naval medicine, descriptions of suggested devices, clinical notes on interesting cases, editorial comment on current medical literature of special professional interest to the naval medical officer, and reports from various sources, notes, and comments on topics of medical interest.

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P. S. ROSSITER,
Surgeon General, United States Navy.

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SPECIAL ARTICLES

THE POST-TRAUMATIC ABDOMEN. DIAPHRAGMATIC HERNIA AS A SEQUEL OF WAR INJURIES[1]

By LUCIUS W. JOHNSON, Captain, Medical Corps, United States Navy

Great wars stimulate us to study and investigation along many lines which receive little attention during times of peace. Thus, during the World War, we saw a rapid increase in our knowledge of fractures, infected wounds, empyema, and gas gangrene, for examples, and great improvements were made in our methods of treating them. Following the war, elaborate studies were published giving the results of treatment and the lessons learned through observation of large numbers of cases. These form valuable repositories of learning to which we must refer frequently lest the knowledge gained through war experiences be forgotten.

Traumatic diaphragmatic hernia is another of those topics in which our interest increases during and after great wars but subsides during long periods of peace. One who reviews the literature in this field will be impressed by the fact that the notable studies were made and the best papers published during and shortly after important wars. In peaceful times of the past it has received scant attention because those types of injury which are likely to produce diaphragmatic hernia are common to military activities but rare in civil enterprises. This trend may well be changed in the future by the constantly increasing number of severe injuries in automobile accidents. Already a number of cases have been reported in which a crushing injury by automobile has ruptured the diaphragm and produced herniation through it.

As a post-mortem phenomenon, diaphragmatic hernia has been recognized since early times. Hippocrates wrote of large openings in the diaphragm which never healed. Ambroise Paré (1510–90), the greatest of all military surgeons, was the first on record to describe diaphragmatic hernia as a sequel to war injuries. In 1579 he described a case, identical in every way with those observed in the

[1] Part of a symposium on The Post-Traumatic Abdomen, presented before the Eighth International Congress of Military Medicine and Pharmacy at Brussels, Belgium, June–July 1935.

World War. His patient had a penetrating abdomino thoracic wound, from which he apparently recovered but later developed vague symptoms of gastric disturbance. He finally died of intestinal obstruction produced by herniation of the colon through a small hole in the diaphragm.

While excellent studies on the subject were published from time to time, they consisted, until recent years, largely of post-mortem observations. Only since the advent of roentgenological visualization of the gastro-intestinal tract has the diagnosis of diaphragmatic hernia during life and before operation been practical.

A very short and simple definition is that given by Watson, "A protrusion of abdominal viscera through a normal or abnormal opening in the diaphragm into the thorax." More elaborate definitions which involve the fine distinction between hernia and eventration are beyond the scope of this paper.

Diaphragmatic hernias are classified as congenital, acquired, and traumatic. The first two groups are further divided into the true, which have a distinct sac, and the false, which are without a sac. Those of the traumatic type rarely have a sac. Hedblom collected 1,003 cases, reported since 1900, of which 291 (29 percent) were congenital; 361 (36 percent) were acquired, and 351 (35 percent) were traumatic.

More than 78 percent of the nontraumatic and 95 percent of the traumatic hernias were on the left side. The predominance on the left side is usually ascribed to the protection afforded to the right side by the liver.

Hernias resulting from penetrating wounds of the diaphragm have been most commonly produced in recent wars by bullets, fragments of shrapnel, and the bayonet. Hedblom gives the following causes for 127 cases due to war injuries:

Bullet wounds	44
Shell wounds	32
Bayonet wounds	2
Not stated	49

The United States Army provides the following figures of admissions for injuries of the diaphragm:

Rifle ball	11
Shell and shrapnel	19
Other means	37

Falls and crushing injuries are the commonest nonpenetrating causes of diaphragmatic hernia. These indirect injuries usually produce a rupture of the diaphragm in the dome and posterior portion, on the left side.

The stage of respiration and the position of the diaphragm at the moment of injury are important factors in determining the location

and extent of the wound in that organ. It also makes a difference, in stab wounds especially, whether the opening extends parallel to or across the muscle fibers, for small wounds which merely separate the fibers have a tendency to heal while those which cut across them gape widely open and do not heal.

Injuries of the types which are likely to produce diaphragmatic hernia were much more common in the World War than in previous wars because of the prevalence of machine-gun bullets and high-explosive shell fragments. Also, many men were wounded in trench warfare while in the prone position, the longitudinal track of the missile passing through the diaphragm into the abdomen.

While such injuries to the diaphragm are frequent in modern wars, diaphragmatic hernias resulting from them are not common because the associated injuries to other organs are very serious and produce a very high death rate, so only a few live long enough to develop the condition. Also, many patients with diaphragmatic hernia are undoubtedly going about undiagnosed. To illustrate this point, Schloessmann states that, of his 31 cases, 15 were returned to duty with persistent symptoms which were later recognized as being due to this cause.

The list of organs which may be found in the hernia includes all of the abdominal viscera except those in the pelvis. The stomach is most frequently involved, then come the omentum, transverse colon, small intestine, and spleen. Probably many small hernias involving only the omentum or a small part of the stomach go unrecognized for long periods.

The difference between the negative pressure of the thorax during inspiration and the positive pressure of the abdomen plays an important part in producing the heria and increasing its size once the opening in the diaphragm is provided. The constant motion of the organ does not favor healing of the aperture and the oscillations of pressure repeatedly force tissues from below into it, acting as a wedge and stretching it wider and wider. C. J. Smith, while operating to repair a stab wound of the diaphragm, saw the wound enlarge rapidly from 1 inch to 3 inches in size as a portion of the stomach herniated into it and became quickly distended with gas. Increased abdominal pressure while firing in the prone position was probably an important influence in producing sudden herniation in a case reported by J. W. Ellis.

Valuable knowledge of the mechanism which produces diaphragmatic hernia was gained by Truesdale's experimental work. He made an incision in the diaphragm of a dog and closed it with a running suture. After the wound was repaired, the dog was placed under the fluoroscope and the suture of the diaphragm was pulled out. The heart moved immediately toward the opposite side; the

right leaf of the diaphragm contracted downward, increasing the abdominal pressure and forcing the stomach upward. The stomach ascended with each inspiration until it was all within the thorax in an inverted position. The transverse colon and omentum followed it through the opening.

Symptoms of diaphragmatic hernia vary according to its mode of development. A severe crushing injury which produces sudden extensive herniation, as in a case we have recently seen in this hospital [2], may result in death before the thoracic organs can adapt themselves to the sudden change in conditions. At the other extreme we have the cases in which symptoms develop only after a period of months or years of perfect health following the injury. These long-delayed cases may develop suddenly, with acute obstruction, or gradually, with progressively increasing digestive disturbance.

Since our principal interest lies in those hernias which are remote sequels of war injuries, it will be instructive to review the histories of a few cases, each of which illustrates some point that is characteristic of this condition.

1. (Baumgartner and Herscher) Abdimino-thoracic wound by shrapnel ball in March 1915. His condition was very serious, but he was able to return to the front 6 months later. He soon began to suffer from pain after eating, vomiting and tachycardia. In October 1916 he returned to the hospital and was operated on for removal of a shell fragment from the lower lobe of the left lung. No evidence of diaphragmatic hernia was shown by the roentgenologic examination or found at operation. He returned to duty in December 1916. His symptoms persisted and, after the armistice, he returned to the hospital where a diaphragmatic hernia was shown by radiographic study.

2. (Keller) Kicked in the chest by a mule 7 years before and was sick 7 days. Five years later developed a left pleural effusion and digestive disturbances. After 2 years of suffering, the fluoroscopic examination showed a diaphragmatic hernia with the transverse colon in the thorax.

3. (Johnson, personal case, J5–841) Crushed under a falling wall in France 7 years before. For the past 5 years has been under treatment for peptic ulcer, with very little relief afforded. Recently has suffered with angio-neurotic edema. In studying the colonic function, an opaque enema was given. Roentgenologic examination revealed a large loop of the colon in the thorax.

4. (Wiart) Abdomino-thoracic gunshot wound in September 1914. After 3 months in the hospital he returned to duty and was well until May 1917, when he was seized with sudden, violent epigastric pain and vomiting. Roentgenologic examination revealed stenosis of the stomach due to diaphragmatic hernia.

5. (Lecene) Wounded in 1914 by a rifle bullet which entered the right shoulder and lodged in the left flank. He was in the hospital 4 months with peritonitis and hematuria. He returned to duty and remained well until 1924 when he was suddenly seized with symptoms of acute obstruction. Roentgenologic examination showed a diaphragmatic hernia involving the stomach.

6. (Gaudier) Abdominal gunshot wound in 1915. After a few days in the hospital he returned to duty and remained well for 12 years. Then, while con-

[2] U. S. Naval Hospital, San Diego, Calif.

stipated and straining at stool, the scar in the diaphragm ruptured, the organs in the resulting hernia became obstructed and he soon died.

7. (Gaudier) Abdomino-thoracic bayonet wound. After 4 months in the hospital he returned to duty. He soon began to have a gastric discomfort which subsided when he reclined. His symptoms recurred periodically and became progressively worse until, 4½ years later, roentgenologic examination showed a diaphragmatic hernia involving the stomach.

Here are clearly pictured two distinct groups of symptoms, either of which may appear any time from a few minutes to 12 years or more after the injury to the diaphragm. The first is an acute, fulminating affair with violent pain, vomiting, and other symptoms which quickly lead to the diagnosis of partial or complete obstruction. The other begins gradually with occasional pain in the upper left abdomen or lower chest, coming on after eating and subsiding when the stomach is empty. This pain comes periodically, with free intervals at first, but grows progressively more severe and more frequent. Vomiting soon appears and there is loss of weight and strength because the patient fears the discomforts that follow each meal. At first the pain subsides on reclining, especially on the left side, but eventually even this surcease is lost and he is comfortable only when the stomach is empty. In the final stage the diagnosis is made when increasing obstruction demands operative relief or else roentgenological study shows the true cause of the trouble.

Pleural effusion, tachycardia, and other thoracic conditions may be caused by interference with adjacent organs. Extensive adhesions usually occur and lead to further impairment of their functions. Obstruction to some hollow organ, most frequently the stomach or transverse colon, may follow any injury or unusual strain.

Gaudier and Labbé emphasize the fact that in all their four cases, the patients could retain food only when in the reclining position. When they ate in the upright posture they suffered greatly until the stomach was emptied by vomiting. Many writers corroborate this point and some regard it as pathognomonic.

Physical signs of diaphragmatic hernia are inconstant and seldom give any important aid in making the diagnosis. The heart may be moved to the side opposite to the hernia, more often the right. Tympany, distant breath sounds, decreased expansion, and loss of vocal fremitus may be found in the lower chest on the side of the hernia. Peristaltic sounds characteristic of the large bowel may occasionally be heard over the chest.

Diagnosis is difficult and is rarely made except by combined radiographic and fluoroscopic study, when it frequently comes as a complete surprise, not having been considered as a possibility. If obstruction occurs, the true cause is not often found until the abdomen is opened and the operator is distressed to find himself with a long

incision in the wrong place. Errors are common and Harrington found that, in 60 of his cases, the diagnosis of cholecystitis had been made in 29, stomach trouble in 23, secondary anemia in 7, heart disease in 7, intestinal obstruction in 4, and esophageal obstruction in 10.

If the examining physician has diaphragmatic hernia in mind and can elicit the typical history, a clinical diagnosis can sometimes be made. But the latent period is often so long that the injury to the diaphragm has been forgotten or its relation to the urgent condition seems very remote.

In the roentgenologic examination, both fluoroscopy and the plate method should be used. Keller emphasizes the importance of examining the patient in the prone position as well as in the upright. In one of his cases the hernia appeared only when the patient was in the prone position. Cases in which there is a small opening in the diaphragm are the most difficult to detect and it is in these small hernias that there is the greatest danger of acute obstruction.

Treatment is by surgical operation, no matter whether the symptoms are acute or chronic. On this there is general agreement by all writers but there is wide variance of opinion in the choice of the route of access. Some prefer a thoracic incision, others like the abdominal approach, while many advocate a combined abdomino-thoracic route. There seems to be a certain racial predilection in the choice of operation. Among writers who express a preference I find that, out of 11 Americans and English, 8 choose the thoracic route while, of 22 French and German surgeons, 10 prefer the abdomino-thoracic route and 9 the thoracic.

Each type of operation has distinct advantages and disadvantages. The abdominal route allows more complete examination of the abdominal organs and it is necessary to use this incision if there is acute intestinal obstruction or if an enterostomy is required. It is difficult to free dense adhesions to the pleura and pericardium from below and the negative pressure of an intact pleura offers great resistance when efforts are made to return the viscera to the abdomen. The incision may be high midline, left rectus, or along the costal border.

The thoracic route gives better access and wider exposure of the hernial ring and makes it easier to free the adhesions to the pleura and pericardium. It is easier to close the hernial ring from above. The heart is already displaced and the lung collapsed, so the dangers of the resulting surgical pneumothorax are greatly reduced. I believe this to be the best route if the diagnosis is made before operation and there is no acute obstruction. Many incisions are advocated, from resection of a long piece of a single rib to elaborate U-shaped flaps containing several ribs.

The abdomino-thoracic route gives wide access to both cavities and thus facilitates freeing the adhesions and closing the hernial ring. But it is accompanied by a greater risk and higher mortality. It is the preferred operation for difficult and complicated cases. Either an abdominal or a thoracic incision may be continued into the other cavity if the obstacles are found to justify it. I have had occasion to extend a thoracic incision into the abdomen and found that it gave very free access to the regions above and below the diaphragm, but as happened to many others, my patient died of shock.

The indications may be summarized thus: When there is acute obstruction due to diaphragmatic hernia, use the abdominal incision. When the hernia has been diagnosed in advance and there is no acute obstruction, the thoracic route is preferable and preliminary pneumothorax is recommended as a protective measure. Either incision can be extended into the other cavity when the difficulties of the case require it.

The chief obstacles to a successful operation lie in the adhesions to delicate organs and closure of the hernial aperture. To free the colon from the pericardium, for instance, requires a clear view and good working space, conditions which are hard to obtain. The opening in the diaphragm can be closed, in many instances, by simple mattress sutures. Keller used a large flap of latissimus dorsi muscle to close the hiatus. Baumgartner and Herscher cut the diaphragm from its costal attachments and sewed it to the chest wall higher up, making it flat and horizontal instead of a dome. They also sewed the stomach into the opening in one case, as did Schloessmann and the latter suggests using the spleen for this purpose. I see no reason why the method of repairing hernias in other regions by the use of fascia lata, as recommended by Gallie and Le Mesurier, should not be equally valuable for diaphragmatic hernias. Certainly strips or patches of fascia lata would seem to be a more suitable material than the stomach or spleen.

Phrenicotomy, either by cutting or crushing the nerve, has been found a valuable aid in many cases. It produces immobility and relaxation of the diaphragm, thus making the closure much easier and maintaining rest during the period of healing. A return of function may be expected after 3 to 6 months.

Enterostomy has proved to be a lifesaving procedure, especially in very adherent hernias with acute obstruction. It carries the patient past the crisis and allows a secondary operation for the repair of the hernia under more favorable circumstances.

Schwartz produced artificial pneumothorax as a preliminary to operation by the thoracic route, to lessen the evils of surgical pneumothorax by accustoming the patient to the changed conditions in

advance. A similar procedure was employed by Rienhoff and Broyles in two cases of total pneumectomy. They began 2 weeks prior to operation; the lung was compressed by a gradually induced and finally complete artificial pneumothorax. The patient thereby became inured to breathing only with the noncollapsed lung and so established, before operation, a respiratory and circulatory equilibrium under altered intrathoracic pressure. When the pleura was opened, the pulse rate and blood pressure remained unaltered. Removal of the entire lung was accomplished with practically no shock. This method would seem to offer great promise in cases of diaphragmatic hernia when the necessary delay is permissible. Shock, which causes more than half of the deaths, would be greatly reduced or entirely eliminated.

Choice of the anesthetic plays an important role in the successful operation. Positive pressure anesthesia given through the intratracheal tubes gives the anethetist excellent control over the patient's breathing and avoids respiratory embarrassment from the surgical pneumothorax, when the thoracic route is used. It also makes possible full inflation of the lungs when closing the incision, which is much more satisfactory than aspirating the air from the pleura after closure.

The mortality rate is high in operations for diaphragmatic hernia, even under most favorable conditions and when obstruction supervenes it is greatly increased. Hedblom states that the mortality rate in the presence of obstruction is 2½ times that of nonobstructed cases. In a series operated in the presence of obstruction, the rate following laparotomy was just four times that following thoracotomy while in the nonobstructed cases the mortality was approximately the same. Shock is the most important cause of death, accounting for nearly 50 percent of all fatal cases. Harrington had 5 deaths in his series of 60 operations. When the presence of the hernia is not known before operation and one operates for intestinal obstruction, a low midline or rectus incision is usually employed, just the worst possible location for repair of the diaphragm. This makes the operation longer and more difficult, thus increasing the death rate.

There are two lessons that our experiences with diaphragmatic herina should teach us. First, penetrating wounds in the region of the diaphragm should be thoroughly explored and any injuries of that organ carefully repaired. Second, when a patient with a recent or old abdomino-thoracic wound develops evidences of obstruction or chronic digestive troubles, diaphragmatic herina should be suspected.

ABSTRACT

Wounds of the abdomen or thorax may involve the diaphragm, producing openings in that muscle through which herniation may occur. The hernia may develop immediately or after a latent period of variable length.

Symptoms due to a diaphragmatic hernia may be acute, due to obstruction, or gradual, vague digestive disturbances which grow progressively worse.

Diagnosis is usually made by roentgenological studies, rarely by clinical evidence.

Treatment is by operation through abdominal, thoracic, or abdomino-thoracic incision.

REFERENCES

Watson, L. F. Hernia. St. Louis, C. V. Mosby Co., 1924.

Hedblom, C. A. In Dean Lewis' Practice of Surgery, Prior 1930, Vol. 5, Chap. 7.

Medical Department of the United States Army in the World War. Vol. 11, part 1, 1927.

Schloessmann. Beit. zur Clin. Chir., 113: 669, 1918.

Smith, C. J. Brit. Med Journ., 1: 926, 1915.

Ellis, J. W. U. S. Naval Med. Bull., 26: 924, 1928.

Truesdale, P. E. Trans. Amer. Surg. Assoc., 1929, 174.

Baumgartner, A. and Herscher, D. H. Bull. et Mêm. de la Soc. de Chir. de Paris, 45: 1, 185, 1919.

Keller, W. L. Amer. Journ. Surg., 8: 598, 1930.

Wiart, P. Bull. et Mêm. de la Soc. de Chir. de Paris, 43: 25, 1917.

Lecène, M. P. Bull. et Mêm. de la Soc. Nat. de Chir., 54: 933, 1928.

Gaudier, M. H. Bull. et Mêm. de la Soc. de Chir. de Paris, 45: 1, 949, 1919.

Gaudier, H., and Labbé, M. Bull et Mêm. de la Soc. de Chir. de Paris, 44: 383, 1918.

Harrington, S. W. Journ. Amer. Med. Assoc., 101: 987, 1933.

Gallie, W. E., and Le Mesurier, A. B. Canad. Med. Assoc. Journ., 11: 504, 1921.

Schwartz, A. Bull. et Mêm de la Soc. de Chir. de Paris, 45: 1, 958, 1919.

Auvray. Bull. et Mêm. de la Soc. de Chir. de Paris, 45: 1, 698, 1919.

Rienhoff, Jr., W. F., and Broyles, E. N. Journ. Amer. Med. Assoc., 103: 1121, 1934.

Aimé, P., and Solomon, J. Amer. Journ. Roentgenol., 6: 376, 1919.

Christie, G. W. Brit. Med. Journ., 1: 599, 1925.

Haudek, M. Wien, Klin. Wochenschrift, 25: 1705, 1912.

Bowditch, H. I. Buffalo Med. Journ., 9: 1, 1853. Also, Il. 9: 65, 1853.

Cooper, A. Anatomy and Surgical Treatment of Abdominal Hernia, Lea and Blanchard, Philadelphia, 1844, p. 339.

VENTRAL HERNIA

AS A SEQUEL OF THE TRAUMATIC ABDOMEN[1]

By FREDERICK R. HOOK, Commander, Medical Corps, United States Navy

In approaching the subject of ventral hernia one must necessarily be impressed by the great role that the surgeon's knife plays as a causative agent. In civil life, even in this age of numerous high-speed motor vehicle and industrial accidents, it is unusual to see a ventral hernia, other than one secondary to a surgical operation. I should, however, like to exclude those small protrusions through the linea alba known as epigastric hernias, which are not as a rule traumatic in origin, and comprise about 1 percent of all hernias.

We are told that in ancient times no distinction was made between ventral and umbilical hernias, and that it was not until the fourteenth century that Guy de Chauliac attempted to distinguish between those protrusions outside the umbilicus and those through the umbilical area.

In reviewing the text books on surgery published during the latter part of the last century and the early years of this century, one is impressed by the lack of information contained in them on the subject of ventral hernia. Some authors do not mention it at all while others dismiss it with only a few lines. It is apparent that surgery was considered only in the presence of strangulation and was accompanied then by an operative mortality rate of 50 percent.

Certainly the large ventral hernias we see today were seldom seen then, for which, if we stop to consider, there is sufficient reason. Although Lord Lister published his first report on the use of antisepsis in surgery in 1865, there was much criticism of his work, and the general use of antisepsis by the profession as a whole did not arrive until many years later. Previous to the Listerian period the operation of laparotomy was of such serious import to the patient that it was usually attempted only as a last resort, and few of the patients lived long enough to develop ventral hernias. As antiseptic surgery grew and blossomed into aseptic surgery, it was only natural that abdominal section should become more commonly employed, that recovery from such operations should be the rule rather than the exception, and that this type of hernia should come into prevalence.

In Sir Astley Cooper's (1) classical work on abdominal hernia, published first in 1804, we find a most interesting statement regarding ventral hernia:

> This disease is not frequent, for though my situation at the hospitals give me the opportunity of seeing a great number of herniae, yet neither my notes,

[1] Presented at the Eighth International Congress of Military Medicine and Pharmacy, Brussels, Belgium. June-July 1935.

nor my memory furnish me with more than 20 examples of this disease in as many years.

Ventral hernia therefore might truly be called the homely stepchild of abdominal surgery.

From a practical standpoint there is little difference between those hernias following gunshot wounds of the abdomen and those secondary to laparotomy. During the World War it was not uncommon to see men in whom a large portion of the abdominal wall had been carried away by a shell fragment. Neither is it uncommon now to see patients in whom postoperative wound infection has occurred with marked sloughing and atrophy of the parietes, so that as a whole the differentiation of the two is only of academic interest. Hernias may follow wounds that involve only the abdominal wall, but they are not nearly as frequent as those in which there has been visceral injury that has required laparotomy.

Statistics compiled by the Medical Department, United States Army (2) show that in the American Expeditionary Forces during the World War, 3.3 percent of all patients admitted to the hospitals had wounds of the abdomen and pelvis. As many of those receiving abdominal wounds died on the field of battle or en route to the rear before reaching a hospital, and therefore, were not included in these statistics, we get an erroneous idea of the incidence of this type of wound. Of those reaching the hospitals with abdominal wounds, 43.32 percent terminated in death.

Unfortunately no statistics are available as to the percentage of ventral hernias that have developed in those recovering from abdominal war wounds. Various writers, however, have collected data on incisional hernias, and there is reason to believe that the two will not differ materially. Matthews (3) estimated that incisional hernias occurred in 1 to 6 percent of all clean laparotomies, and in 15 to 16 percent of all drainage cases. The German clinics (4) have reported 8.9 percent hernias after longitudinal incisions after healing per primam, and 31 percent after healing per secundam. Greenwood (5) in 500 cases reported 24 postoperative hernias, 21 of which were due to sepsis. Abel (6) in 1924 examined 586 patients after laparotomy and found postoperative hernias in from 9 to 20 percent of the scars, depending upon whether the wounds had been closed by layers or through and through (en masse). Masson (7) reporting on the work done at the Mayo Clinic between the years 1915 and 1919, stated that of the 28,970 abdominal operations performed, 596 or 2.05 percent were for postoperative ventral hernias. During this same period this type of hernia constituted 14.66 percent of all hernias operated upon at that clinic. The writer (8) in a study of 603 hernia patients admitted to the Brooklyn Naval Hospital over a period of 2½ years,

found 11.6 percent to be of the ventral type. It is interesting to note that Rouffart (9) collected from the literature 1,506 cases in which transverse incisions had been made in the lower abdomen, with postoperative hernias in only 0.33 percent. These statistics, although interesting do not tell the whole story, as there are many patients with weak abdominal scars and few symptoms who prefer wearing an abdominal support to further surgery. Generally speaking, the ones that seek operative relief are those that are having severe symptoms which interfere with their work.

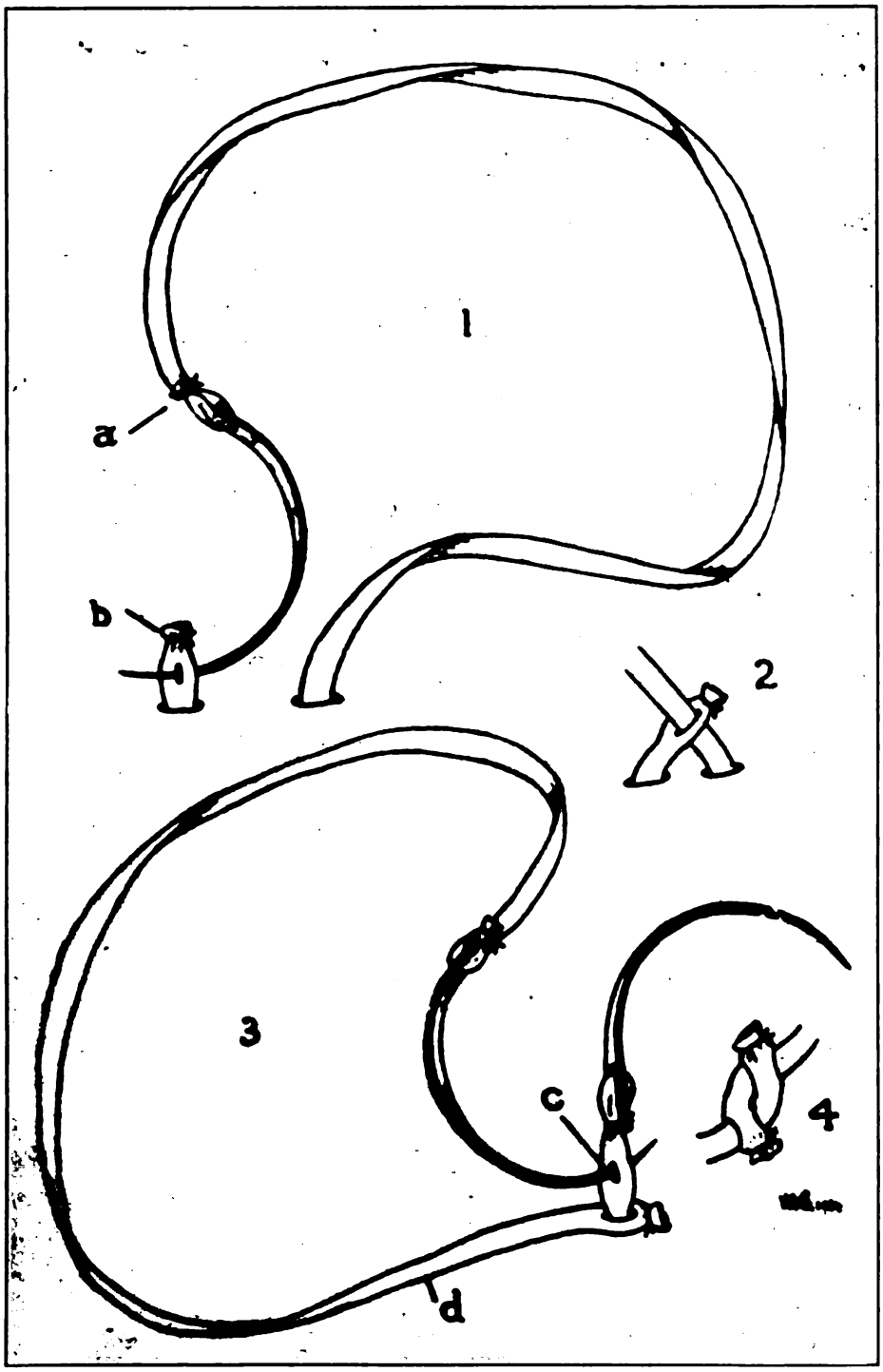
There is no doubt but that wound infection plays the chief role in the production of ventral hernias. Infection may be inherent in the nature of the lesion for which surgery is being undertaken or may be due to some faulty technic. In many cases of severe and extensive abdominal operations, a ventral hernia may be of only secondary importance. Masson (7) in his study of 596 postoperative hernias, reported that drainage had been used in 280 or 54.08 percent. Sloan (4) studied 239 cases, 93 of which had had large drainage tubes in the incisions, and 103 had had small drains. In 99 cases the wounds had been closed without drainage, and had healed per primam. He lays down the following rule:

> The lateral pull upon the suture line following a vertical abdominal incision and the incidence of postoperative hernias are in proportion to the square of the length of the incision. The larger an incision the more apt a hernia is to develop.

Second to wound infection as an etiological factor is atrophy of the abdominal muscles due to injury to the motor nerves. Assmy (10) in 1899 found from his experimental work that severance of the motor nerve supply to a muscle invariably led to atrophy of that muscle. There are undoubtedly many other minor factors that play a part in the production of postoperative hernias, but these two factors by far overshadow all of the rest.

The symptoms vary a great deal, depending upon the area in which the hernia is located, the presence of peritoneal adhesions, the size of the hernial ring, and the contents of the sac. Those with large rings frequently have no smyptoms, while those with small openings may complain of gastro-intestinal symptoms, constipation, and attacks of partial obstruction. A few patients will have such constant trouble that they will be unable to work and will live the lives of invalids. Strangulation is not common but should always be kept in mind. It is most frequently found where the neck of the sac protrudes through the aponeurosis of the external oblique muscle.

Seldom is any difficulty encountered in diagnosing a ventral hernia. By placing the patient on his back on the examination table and asking him to attempt to rise without using the arms as a support,



Courtesy Gallie and LeMesurier.

FIGURE 1.—DIAGRAMS ILLUSTRATING POINTS IN THE TECHNIQUE OF USING LIVING SUTURES OF FASCIA.

1, The strip of fascia lata has been tied into a large-eyed needle with fine silk *a*, and a similar ligature has been tied around the tail of the suture at *b*. The needle has been passed through some strong aponeurotic tissue and then through the tail of the suture to form a slip-knot. *2*, The slip-knot drawn taut. *3*, Method of joining one suture to another. The suture *c* has been used up. The needle of suture *c* is passed through the tail of suture *d*, and the needle of suture *d* is then passed through suture *c*. The needle of suture *c* is cut off, and suture *d* is drawn taut. *4*, The jointing of the two sutures completed.

one can outline the hernial ring with considerable accuracy, and also determine whether or not the sac contents can be returned to the abdominal cavity.

The importance of prevention of ventral hernia is strongly brought to mind when one reviews the literature or attempts a postoperative follow-up on the hernias he has repaired in the past. Masson (7) reported 14.1 percent recurrences; Coley (11), 16.4 percent; Matthews (2), 25 to 40 percent; and Da Costa (12) estimated it at 40 percent.

There are many factors that play important roles in the prevention of ventral hernias following abdominal operations. The most important of these are: The preoperative preparation of the patient, the choice of the incision, the type of drainage employed, the method of closure of the incision, the kind of suture material used, and the nature of the postoperative treatment.

As many abdominal operations are of an urgent nature, any extended preoperative treatment is out of the question. On the other hand, where surgery is one of election, sufficient time should be spent in getting the patient in the best possible condition. Some will require being built up, while in other obese patients a reduction in weight is desired. It has long been known that operative wounds in the cachectic and jaundiced patients healed poorly, and that these conditions accounted for many of the wound disruptions reported in the literature. Sokolov (13) has given us the results of a most exhaustive study of this interesting condition. In replies to questionnaires sent out to 1,000 surgeons throughout Europe, he estimated the normal disruption rate at 2 to 3 percent of all abdominal operations. Most of these cases are followed by hernias.

There has been much written on the advantages and disadvantages of the various incisions. Those who favor the transverse incisions claim a much lower hernia rate than is expected in the vertical incisions. The fact remains, however, that one still finds the vertical incisions employed almost universally in our larger surgical clinics. It is well to remember that the abdominal wall is supplied by the anterior branches of the fifth to twelfth thoracic, with branches from the ilio-hypogastric and ilio-inguinal nerves, and that these nerves run transversely across the abdomen, and further, that any vertical incision through the lateral abdominal or recti muscles will sever the fibers of some of these nerves.

It is believed that no one incision should be employed to the exclusion of all others, and that every effort should be made to protect the nerve and major blood supply to the part. If a vertical rectus incision is to be used it is much better after incising the anterior sheath to retract the body of the muscle laterally than to proceed directly through it.

It has been known for years that the general peritoneal cavity could not be efficiently drained. More recently the advisability of drainage of abdominal wounds where soiling of the peritoneum has occurred, and in localized peritonitis, has been questioned. Buchbinder, Droegemuller, and Heilman (14) produced in dogs an experimental, severe, spreading peritonitis such as follows bowel perforation, and is accompanied by a mortality rate of over 90 percent. They were able to reduce the death rate to 58 percent by removal of the septic focus and closure of the adbominal wall. When soft rubber drainage was used in such experimental lesions, a mortality rate of 100 percent resulted. Their work is very suggestive to say the least.

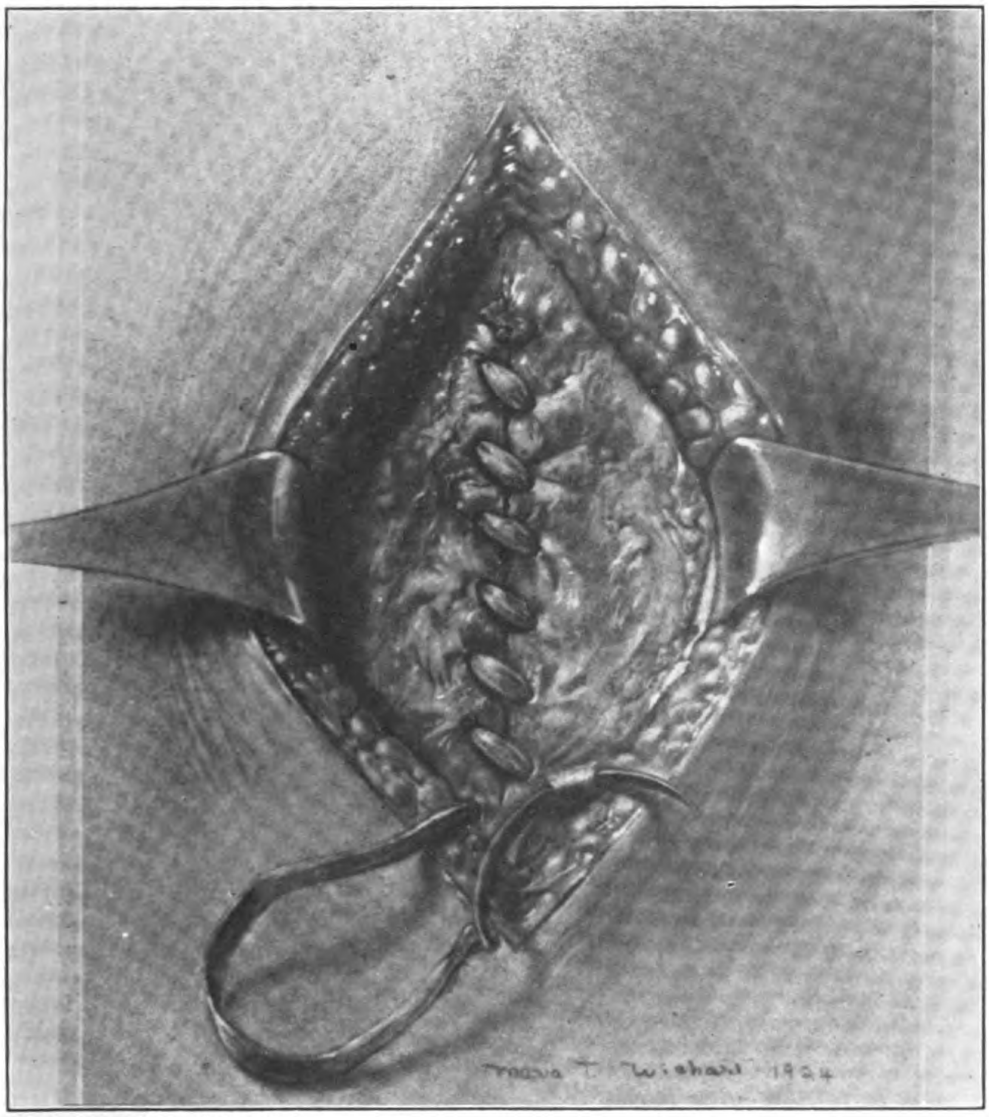
Drains, if considered indispensable by the surgeon, should be as small and as few as possible, preferably brought out through a stab wound, and continued in use for the shortest possible period of time, as they lead to hernias much the same as infection does, in that they prevent edge to edge approximation of the anatomical layers and healing takes place by interposed scar tissue.

In 1915 Lemaitre (15) published his first work on debridgement of wounds. It was really revolutionary as previous to that time nearly all war wounds were expected to suppurate. By excision of the devitalized tissues and primary closure he was able to cure from 90 to 96 percent of all wounds. Undoubtedly, many ventral hernias have been prevented in abdominal wounds by the application of his method of treatment. Felsenreich (16) more recently reporting on the work done in the Vienna accident station, showed that 96 percent of all wounds of the trunk treated by the primary surgical method within 24 hours after injury, healed by first intention.

Many writers have stressed the part that the transversalis fascia and transversus muscle play in maintaining the integrity of the upper abdomen. It should be remembered that these fibers run transversely and that in closing a vertical incision under tension the sutures are apt to tear out. In the lower third of the abdomen imbrication of the fascia is desirable. Absolute hemostasis should be assured before the wound is closed. In the words of Binnie (17):

> The ideal closure is a careful approximation of the parietes in their anatomic layers, and absolute avoidance of strain until the embryonic tissues necessary for the repair of the abdominal wounds have had time to become mature.

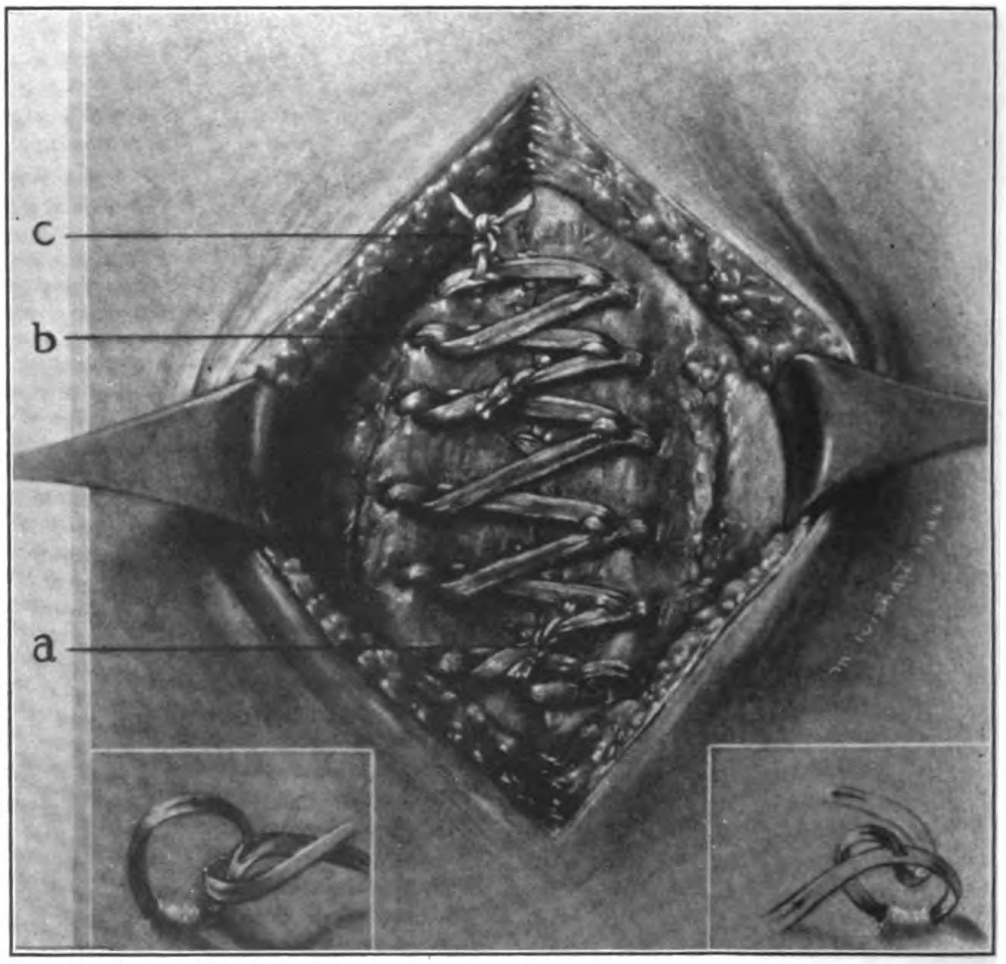
There is much contradictory evidence in the literature in regard to the advantages and disadvantages of absorbable and nonabsorbable suture material, so that one can only arrive at the conclusion that a great deal depends upon the surgeon's personal prejudices. If an absorbable gut is to be used one should be satisfied that what he has chosen will be of sufficient durability to perform the duty intended. In long vertical incisions one should not rely on absorb-



Courtesy of Gallie and LeMesurier.

FIGURE 2.—DRAWING OF THE REPAIR OF VENTRAL HERNIA WITH SUTURES OF FASCIA LATA.

Insertion of the first row of sutures which brings the edges of the defect together or as nearly together as they will come without great tension.



Courtesy of Gallie and LeMesurier.

FIGURE 3.—DRAWING OF THE REPAIR OF VENTRAL HERNIA.

Insertion of the second row of sutures. The needle takes deep bites of the aponeurosis at some distance back from the edges of the opening. *a*, A joint between two sutures; *b*, lock-stitch inserted to prevent slipping; c, knot which terminates the suture. It is transfixed with a ligature of fine silk or catgut. The inserts in the lower corners show methods of making the lock-stitch.

able material alone, but should reinforce the suture line with some form on nonabsorbable retention suture. Generally speaking, nonabsorbable sutures should not be buried in the tissues when wound infection is anticipated. I believe at times a too plentiful use of suture material may lead to a hernia by causing local strangulation and necrosis of the tissues, with serum formation, and a resulting weak suture line.

The early postoperative period of laparotomized patients is fraught with conditions that favor the development of hernias, and there is much the surgeon can do during this period to prevent them. The position in bed should be one of comfort with frequent changes to relieve restlessness. Although many drugs have been recommended, none have proved as satisfactory in the relief of pain and restlessness as the opiates.

The routine use of carbon dioxide and oxygen inhalations at frequent intervals will reduce pulmonary complications to a minimum, and, therefore, will save many patients not only the discomfort, but also the danger from coughing, sneezing, and dyspnea.

Careful observation for the early signs of ileus, with the intelligent use of the stomach tube is of the utmost importance. Retching and vomiting should never be permitted and can easily be prevented by passing a small tube (Levin) into the stomach through the nose. Ileus occurs much less frequently when care is taken to maintain the fluid balance of the body. Cathartics have no place in the treatment of this condition and repeated enemata are not only useless but annoying to the patient. By withholding food and fluids by mouth, and by decompressing the intestinal tract after the method of Wangensteen and Paine (18), and replacement of body fluids by the subcutaneous and intravenous (continuous drip) methods one has little to fear from this condition.

Marked increase in weight should be guarded against in patients that have recently had abdominal operations, as the increase in the intra-abdominal pressure may be sufficient to cause a giving away of the not too firmly healed scar. Opinion is divided as to the advisability of wearing an abdominal support postoperatively. In those patients inclined toward obesity there is certainly something to be said in its favor.

It is entirely beyond the scope of this paper to describe or even to mention many of the various operations that have been devised for the cure of ventral hernia. The large number of operations proposed leads one to the conclusion that all are unsatisfactory, or at least not satisfactory under all conditions. An attempt will be made to group these various operations, and to evaluate each group, rather than each individual operation. Exceptions will be made

where it is evident that some one operation in the group possesses outstanding merit.

The first group considered is that described by surgeons who proceed upon an anatomico-physiological basis under all conditions, in that they attempt to restore the abdominal wall to its normal condition, peritoneum to peritoneum, fascia to fascia, muscle to muscle, aponeurosis to aponeurosis, and skin to skin. This type of operation has not only given a high recurrence rate but also a high death rate, probably due to the sudden and marked increase in the intra-abdominal pressure caused by the diminution of the abdominal circumference by closure under extreme tension. It should be considered only in selected cases where the hernia is small and there has been no serious damage to the abdominal wall.

In the second group the surgeons are satisfied to establish a firm scar between the abdominal pillars, and seek to produce it by imbricating, folding, or doubling of fascia and oponeurosis. This group contains many different operations of merit, the most popular of which is known in the United States as the Mayo operation. This method of imbrication was first described by William J. Mayo (19) in 1898. No attempt is made to reform the anatomy of the abdominal wall or to excise the scar; in fact, the scar is used as a part of the tissues in the imbrication. This group as a whole gives a much higher percentage of cures than the first group, but its shortcomings are somewhat similar, in that the sutures frequently give away and the scar stretches, permitting a return of the hernia.

The experimental work of Gallie and LeMesurier (20) on wounds of fascia and aponeurosis is of the greatest importance in the treatment of hernias of all types. In brief, their findings were: in edge to edge approximation of wounds in fascia and aponeurosis with absorbable sutures, healing took place by means of a delicate scar which developed from the areolar membrane which invests the surfaces and from the base connective tissue stroma which separates the bunches of fibers. The scar is not strong and slowly stretches, leaving a gap between the edges. When nonabsorbable sutures were used the tendency to open up was not so great but there was a tendency to cut through. Simple overlapping of the edges of the wound added little or nothing to the solidity of the healing, but if before overlapping the edges the areolar membrane was thoroughly removed and the areas which were placed in contact scraped, the degree of adhesion was increased, but prolonged and unusual strain, however, resulted in stretching of the scar. Side-to-side suture of undamaged folds of aponeurosis never resulted in permanent adhesions.

The third group comprises those operations in which the surgeons attempt to bridge the defect in the abdominal wall with some type of foreign material. Göbel (21) in 1900 first reported upon his

use of silver wire filigree, and in 1903 Bartlett (22) described an improved filigree. In 1914 Lefèvre (23) reported upon the use of the cuff of a rubber glove fixed in the tissues across the hernial defect. Moure (24) in 1922 described the use of a bronze wire to encircle and pucker the hernial opening. Good temporary results have been reported by the surgeons sponsoring these operations, but unfortunately the foreign material frequently causes severe discomfort and foreign body reaction, and in time the tissues are absorbed, leaving nothing but a firm scar with embedded foreign material.

The fourth group includes those operations that make use of fascia, either as a suture material, or as free or pedicle patch grafts. The use of living fascial sutures was first described by McArthur (25) in 1901. However, it remained for Gallie and LeMesurier (20) 20 years later to popularize their use. Mann (26) in 1914 demonstrated that large patch grafts from the ilio-tibial band of the fascia lata could be transplanted with almost uniform success. Koontz (27) in 1926 recommended the substitution of preserved ox fascia for living fascial sutures. Fascial sutures may be used alone or as a means of reinforcement in one of the other types of operation.

it is necessary to form a fifth group to include many miscellaneous operations that do not readily fall under the headings of any of the other four groups. Many of these operations have been described for the treatment of some peculiar type of hernia, and little time can be given them here. Amongst this large group one might mention; the osteoplastic grafts to the hernial opening as described by König (28) in 1911, the series of releasing incisions in the aponeurosis as described by Hensen (29) in 1914, and Gibson (30) in 1920, the method of inversion of Haynes (31) in 1917, transplantation of the sartorius muscle by Ramlau-Hansen (32) in 1922, transplantation of the rectus muscle by Nuttall (33) in 1926, and the interdigitation method of Babcock (34) in 1925.

There is no question but that the imbrication method of Mayo was a big step forward over the old method of edge-to-edge approximation of the hernial pillars, and that the use of silver wire filigree yielded good results in a few cases previously considered inoperable, but the institution of the Gallie technique of living fascial sutures in the treatment of hernia, opened up an entirely new field. A great many more patients can be operated upon and their hernias cured by this method than would be possible by the older methods.

In the preoperative preparation of large ventral hernias the patients should be put to bed for a period of 3 to 6 weeks on a restricted diet, and elimination encouraged. Weight is reduced, and since the abdominal muscles are used little while at rest, they lose their tone and become more elastic. These two factors permit more stretching

of the tissues and therefore not so marked increase in intra-abdominal tension occurs at operation.

The anesthesia should be one that gives complete relaxation. In this respect spinal anesthesia is ideal. In the smaller hernias local field block is quite satisfactory.

At operation there should be wide excision of the skin scar, keeping in mind the fact that frequently no peritoneal covering to the viscera exists, the hernial contents lying immediately beneath a very thin cicatrix of the skin. It is advisable to develop the incision through fairly normal tissues until the normal aponeurosis is identified. Only then, is it safe to proceed toward the abnormal scarred tissues of the hernial area, working carefully, and constantly on the alert for pockets in which a loop of gut may be adherent.

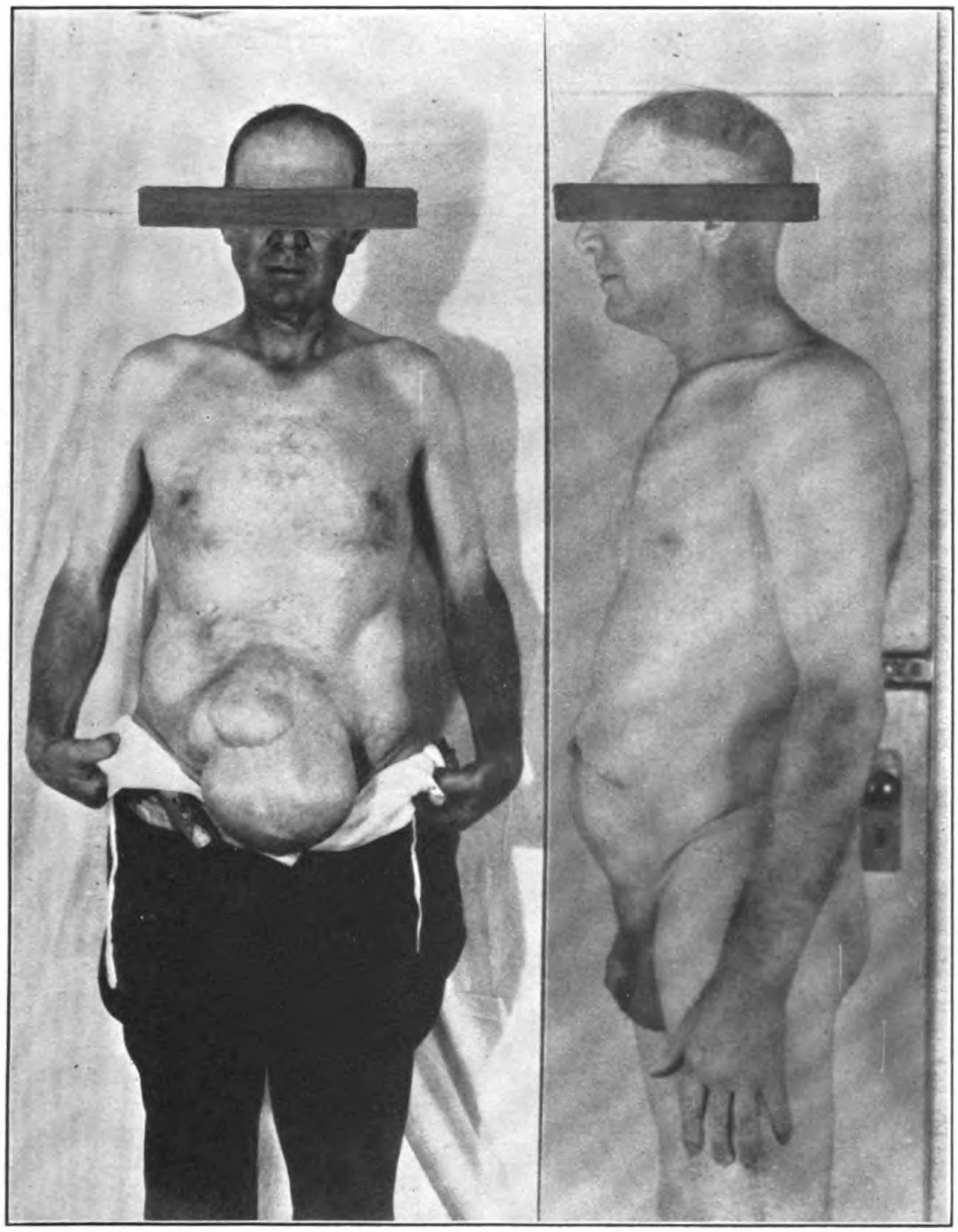
Opinion is divided as to the advisability of opening the sac and breaking up of the peritoneal adhesions. Personally I believe that the adhesions in the immediate vicinity of the hernial ring should be freed and the peritoneum closed as a single layer, whenever possible.

Acute intestinal obstruction is not a common complication but when present resection of the bowel should never be done unless it is hopelessly diseased, as it markedly increases the operative mortality rate.

If the operation of Gallie and LeMesurier, that of living fascial sutures, has been selected as the method of repair, the muscles, fascia, and aponeurosis are not dissected out into their various layers, but are closed as a single layer by running sutures of fascia lata approximately one-quarter inch (0.6 cm) in width (figs. 1 and 2). A second row of sutures placed over the first and going back about 1 inch (2.5 cm) from the edge of the defect may be used when the opening is large and there is apt to be considerable tension on the suture line (fig. 3).

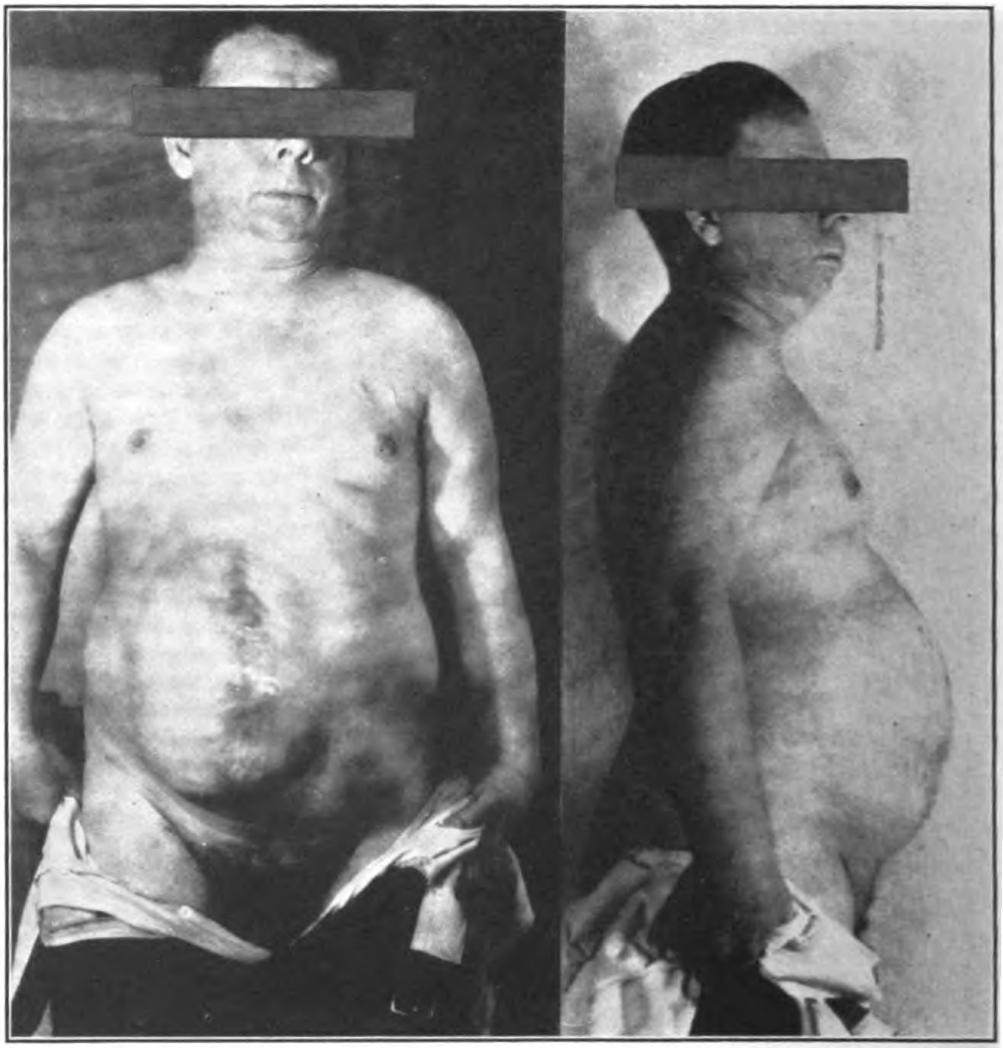
In many of the giant hernias there has been such severe damage to the abdominal wall that it is impossible to completely close the opening, and in others it is possible to do so only with such marked increase in the intra-abdominal pressure as to endanger the life of the patient. It is this type of patient that demands one of the fascial patch operations.

The outstanding operation of this type is that described by Gallie (35) in 1932 (fig. 4). The patch is taken from the fascia lata and greatly resembles the old-fashioned many-tailed abdominal binder. If the hernial defect is more than 3½ inches (8.75 cm) in length, it is necessary to cut two patches from the fascia lata, laying them side by side across the opening. As a rule two patches of fascia 5 by 3½ inches (12.5 by 8.75 cm), can be obtained from the lateral aspect of a single thigh. After the hernial ring has been dissected out, the peritoneum is pushed back from the edge of the opening for



CASE 1.—HISTORY OF HAVING BEEN STRUCK IN THE ABDOMEN FIFTEEN YEARS PREVIOUSLY, PRESENTED AN EPIGASTRIC HERNIA WHICH CONTAINED THE GREATER PART OF BOTH THE LARGE AND SMALL INTESTINE.

Repaired with fascia lata sutures with cure of hernia.



CASE 2.—CHOLECYSTECTOMY IN 1929 FOLLOWED BY WOUND INFECTION AND GIANT INCISIONAL HERNIA. REPAIR OF HERNIA IN 1930 WITH FASCIA LATA SUTURES.

Excellent results.

one-half inch (1.25 cm), or more, so that the needles that have been threaded on the ends of the patch may be passed from within outward through the edge of the ring without entering the peritoneal cavity. At the ends of the hernial opening, special precaution is taken to

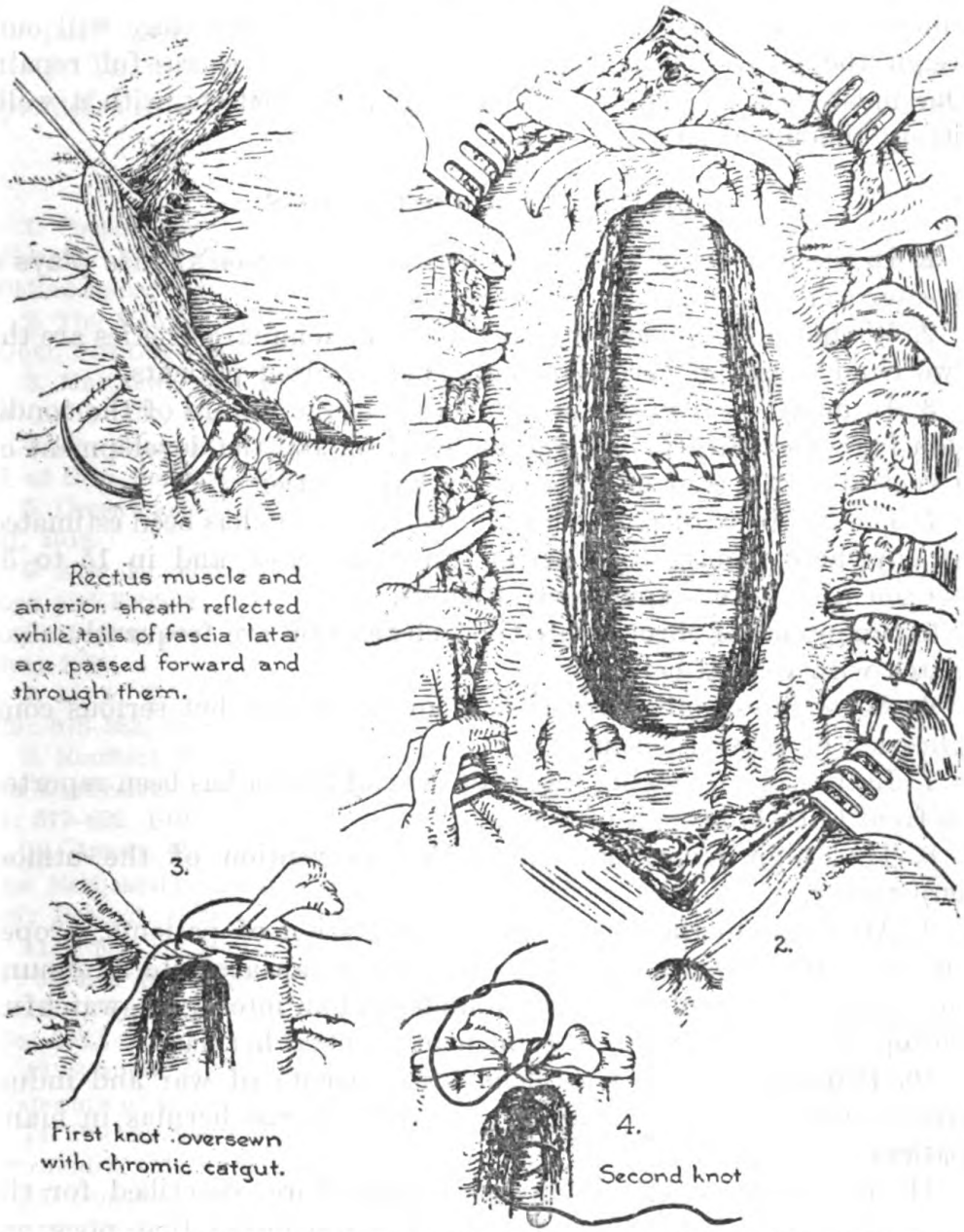


FIGURE 4.—Wide ventral hernial opening closed by two sheets of fascia lata placed edge to edge. The inserts show the method of passing the tails through the edge of the ring, and the method of tieing and oversewing the tails. Courtesy of Gallie.

weave the tails into the edge of the ring so as to prevent a protrusion over the end of the fascial sheet. When all of the tails have been drawn through the abdominal wall they are then tied together, each to its opposite fellow, and the knots oversewn with catgut sutures.

A small drain may be placed beneath the skin to remove any serum that might collect; however, it has been my observation that serum is less frequent in hernia incisions where fascial sutures have been used than with the other types of suture material.

There will be an occasional patient whose general physical condition is such that the dangers from any surgical procedure will outweigh the good that might be derived from a successful repair. One must then be content with providing the patient with a well-fitting abdominal support.

SUMMARY AND CONCLUSIONS

1. In the production of ventral hernia the surgeon's knife plays a greater role than other types of traumatism.
2. Wound infection and atrophy of the abdominal parieties are the two chief causes of hernia in post-laparotomized patients.
3. In many cases infection is inherent in the nature of the condition for which surgery is being undertaken, and the development of a ventral hernia is of only secondary importance.
4. The incidence of postoperative ventral hernia has been estimated at anywhere from 1 to 9 percent in clean cases, and in 15 to 31 percent of infected laparotomy wounds.
5. Symptoms of chronic intestinal obstruction are frequently associated with ventral hernia.
6. Acute intestinal obstruction is an uncommon but serious complication of ventral hernia.
7. Recurrence following repair of ventral hernia has been reported in from 14 to 40 percent of cases.
8. This high recurrence rate makes prevention of the utmost importance.
9. Attention to the preoperative preparation of patients, proper choice of incisions, reduction of drainage to an absolute minimum, the careful closure of operative wounds, and an intelligent, watchful, postoperative regime will prevent many ventral hernias.
10. Primary surgical treatment (debridment) of war and industrial wounds of the abdominal wall will prevent hernias in many patients.
11. The large number of operative procedures described for the cure of ventral hernia leads one to the conclusion that none are entirely satisfactory.
12. An attempt has been made to group the various operations described in the literature.
13. Fundamentally, there is no difference in the treatment of hernias secondary to laparotomy and those due to other forms of trauma, such as gunshot wounds.

14. The type of suture material used plays a very prominent role in the cure of ventral hernia.

15. Catgut. silk, linen, and silver wire sutures have all proven unsatisfactory.

16. The operations that make use of living fascia, either as sutures, or patch grafts, give the highest percentage of cures.

17. The ilio-tibial band of the fascia lata affords an excellent and plentiful supply of living fascia that can be utilized for this purpose.

REFERENCES

1. Cooper, Sir Astley, The Anatomy and Surgical Treatment of Abdominal Hernia. Second London Edition by C. Aston Key, Lea and Blanchard, Philadelphia, 1844.

2. The Medical Dept. of the U. S. Army in the World War, vol. 11: 443–468, Govt. Printing Office, 1924.

3. Matthews, A. A., Repair of Postoperative Incisional Hernia., Surg. Clin. of N. A. 13: 151, February 1933.

4. The German Clinics as quoted by Sloan, E. P., Abdominal Incisions., Amer. J. of Obst. and Gyn., 23: 226–232, February 1932.

5. Greenwood, H. H., Ventral Hernia., Brit. Med. Jour., 2: 312, September 21, 1918.

6. Abel, as quoted by Ochsner's Surgical Diagnosis and Treatment., vol. 1: 68, Lea and Febiger, 1924.

7. Masson, J. C., Postoperative Ventral Hernia., Surg. Gyn. and Obst., 37: 14, July 1923.

8. Hook, F. R., Treatment of Recurrent Hernia., U. S. Naval Med. Bul., 29: 373–383, July 1931.

9. Rouffart, E., De la Valeur des Différentes Incisions dans les Laparotomies en Gynecologie et en Obstetrique., Arch. Mens. d'obst. et de Gynéc., Paris, 8: 577–622, 1919.

10. Assmy, P., Über den Einflus der Durchtrennung Motorischer Nerven auf die Narbenbildung bei extramedianen Bauchschnitten., Beit. z. Klin., Tiibing, 23: 109–125, 1899.

11. Coley, as quoted in Ochsner's Surgical Diagnosis and Treatment., Vol. 1: 68, Lea and Febiger, 1924.

12. DaCosta, J. C., Modern Surgery., Tenth Ed. page 1081, W. B. Saunders Co., 1932.

13. Sokolov, S., rupture of Wounds after Laparotomy with Eventration or Exposure of Intestines, Ergenbn. d. chir. u. Orthop., 25: 306–379, 1932.

14. Buchbinder, J. R., Droegemuller, W. A., and Heilman, F. R., Experimental Peritonitis 111., Surg. Gyn. and Obst., 53: 726–729, 1931.

15. Lemaitre, R., Essai sur la technique Chirurgical dans une ambulance paur grands blesses immobileses a l'avant., Paris Chirurg., 8: 366–384, 1916.

16. Felsenreich, Fritz., Results of Primary Surgical Treatment of Wounds., Wein. Klin. Wochenschrift, 43: 961–964, July 31, 1930.

17. Binnie, J. F., Operative Surgery., Seventh Edition, 1916. P. Blaikiston Son and Co.

18. Wangensteen, O. H. and Paine, J. R., Treatment of Acute Intestinal Obstruction by Suction with the Duodenal Tube., Jour. A. M. A. 101: 1532, November 11, 1933.

19. Mayo, Wm. J., Remarks on Radical Cure of Hernia., Ann., Surg. 29: 51, January 1899.

20. Gallie, W. E. and LeMesurier, A. B., Living Sutures in Operative Surgery. Can. Med. A. Jour., 11: 504, July 1921.

21. Gobel, Central bl. f. Chir., 1900 page 458.

22. Bartlett, W., An Improved filigree for the Repair of Large Defects in the Abdominal Wall. Ann. Surg., 38: 47, 1903.

23. Lefevre, Voluminous Traumatic Hernia with Loss of Substance of abdominal Wall Closed by Plate of Rubbers, Bul. et Mem. de la Soc. de Chir. de Paris, 44: 1855, 1914.

24. Moure, P., Wire Ring Treatment of Hernia, Journ. de Chir., Paris, 4: 19, April 1922.

25. McArthur, L. L., Autoplastic Sutures in Hernia and Other Diastases. Preliminary Report, J. A. M. A., 37: 1162, November 2, 1901.

26. Mann, A. T., The Free Transplantation of Fascia Lata., Ann. Surg., 60: 481, 1914.

27. Koontz, A. R., Experimental Results in the Use of Dead Fascia Grafts for Hernia Repair, Ann. Surg., 83: 523, April 1926.

28. König, F., Die Radikaloperation Grosser Hernien, besonders der Bauchbruche, unter Verlotrung mit frei verpflantzen Periostlappen, Bietr. z Klin. Chir., Tübing 1911, 75: 797–811.

29. Hensen, J. W., A proposed Addition to the Technic in the Radical Operation for Median Ventral Hernia where the Tension of the Sutures Would Be Excessive, Internat. J. Surg. N. Y., 27: 413–414, 1914.

30. Gibson, C. L., Operation For the Cure of Large Ventral Hernia. Ann. Surg., 72: 214 August 1920.

31. Haynes, I. S., Giant Ventral Hernia. N. Y. Med. Jour., 105: 107, January 20, 1917.

32. Ramlau-Hansen, O., Plastic Correction of Postoperative Hernia. J. A. M. A., 78: 1242, April 22, 1922.

33. Nuttall, H. C. W., Rectus Transplantation in the Treatment of Ventral Hernia. Brit. Med. Jour., 1: 138, January 23, 1936.

34. Babcock, W. W., Interdigitation in the Repair of Large Ventral Hernias with Observation on Lipectomy. Surg. Gyn. and Obst., 40: 853, June 1925.

35. Gallie, W. E., Closing Very Large Hernial Openings. Ann. Surg., 96: 551–554, 1932.

REPAIR OF INGUINAL HERNIA

By G. G. Herman, Lieutenant Commander, Medical Corps, U. S. Navy

In order to insure permanence in reconstructive operations for hernia, fascia must be united to fascia. It has been conclusively shown that red muscle will not permanently unite with white fascia and that in the attempt to accomplish this, no good, but actual harm, may result due to the wasting of valuable fascia which can be utilized to great advantage (1). With these facts in mind one may well wonder why the Bassini operation, either in its classical form or in one of the many modifications, attained such wide acceptance. In the calendar year 1925, the United States Navy, out of a total of 410 inguinal hernia operations performed, lists 324 repairs after the

Bassini method with 75 after Ferguson, 9 after Halstead and 1 each after the Andrews method and the Bloodgood—Johns Hopkins method. This popularity of the Bassini operation, in which red muscle is united with white fascia, is not due to any inherent superiority over latter methods, but rather to extraneous factors that were operative immediately prior to its introduction. If we briefly review the history of hernia from the first belief in magic, which consisted of passing a child afflicted with hernia through a cleft in an ash tree, on through the seventh century when Paul of Aegina recommended removal of the testicle because it was thought that the intestines and testicles were enclosed in the same sac, which must be removed in its entirety to bring about proper healing of the peritoneum, we find the contributions somewhat meager until the eighteenth century when Percival Pott, Antonio de Gimbernat, Antonia Scarpa, and Gottlieb Richter contributed numerous important works on hernia. Yet illuminating as we find these early treatises to be, surgery prior to the middle of the nineteenth century was in a mist. Much had to be sacrificed in the actual performance of an operation because pain did not permit of sufficient time to fully develop the described technique. The agony of the sufferer had naturally and rightly compelled the public to demand rapid if not slap dash surgery, and the surgeon to pride himself on it. Cheselden (1688–1752) was reputed to perform lithotomy in 54 seconds. This state of things changed with the introduction of ether as an anesthetic at the Massachusetts General Hospital about 1846. The pain of operation itself no longer counted and the surgeon was enabled not only to be as cautious and sedulous as dextrous, but also to venture upon longer, profound, and intricate operations. Yet unhappily this new enfranchisement seemed to be of no avail because no matter how successful mechanically the surgical undertaking proved to be, the mortality by sepsis was ghastly.

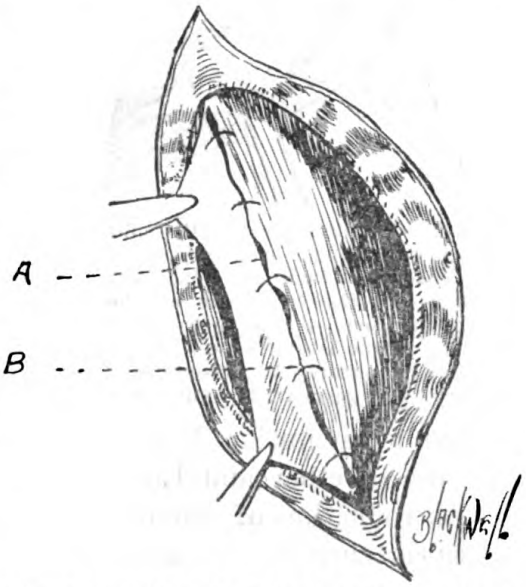


FIGURE 1.—The upper leaf of the previously split aponeurosis of the external oblique secured to the shelving edge of Pouparts ligament—A; with the mattress suture—B. (Modified from Bickham, Operative Surgery, W. B. Saunders Co., Publishers.)

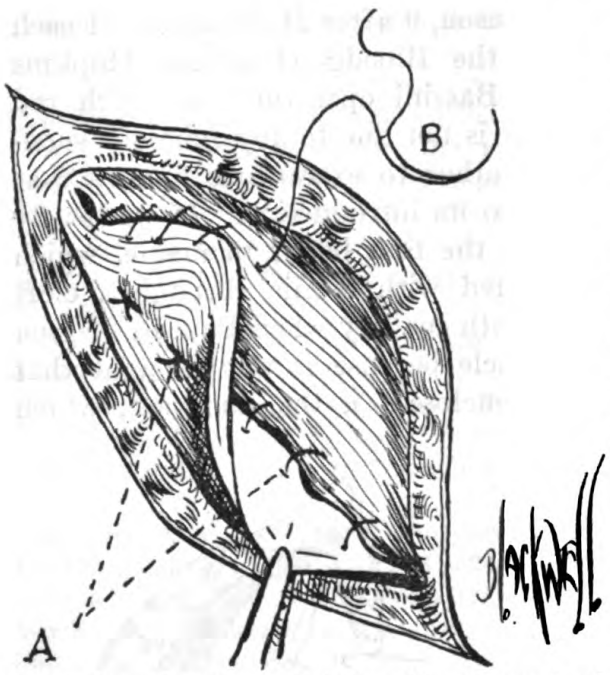


FIGURE 2.—The upper leaf of the split aponeurosis of the external oblique has been sutured to the shelving edge of Pouparts' ligament by mattress sutures—A; the free edge of the lower flap of the aponeurosis of the external oblique is being sutured to the mesial aspect of the lower flaps by continuous suture—B. (Modified from Bickham, Operative Surgery, W. B. Saunders Co., Publishers.)

What a revolution Warren's use of ether and Lister's introduction of sound, antiseptic surgery brought about in the last two decades of the nineteenth century. It was during this period which witnessed the great development from the old surgery into modern scientific surgery that Edoardo Bassini in 1888 introduced the the method of operation for the radical cure of oblique inguinal hernia that bears his name. It was mentioned in the Transactions of the Italian Congress of Surgeons of that year. These are the Healing by first intention, far from being the rule, was a piece of luck too rare to enter into the calculations of the operation.

In 1867 Lister published his first paper on antiseptic surgery. From Pasteur's demonstration that fermentation was a result of minute organisms, Lister came to the conclusion that suppuration was due to the introduction of minute organisms into an open wound. These momentous advances were slow of acceptance and it was not until 1886 that Van Bergman brought forth steam sterilization and general asepsis.

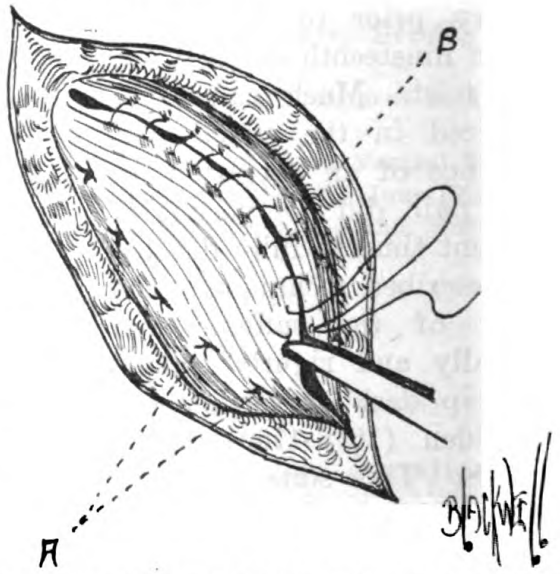


FIGURE 3.—The knots of the mattress sutures securing the upper flap of the aponeurosis of the external oblique to the shelving edge of Pouparts' ligament are shown on the outer surface of the lower flap designated—A; the free edge of the lower flap has been secured to the mesial aspect of the upper flap by continuous suture—B; thus effecting an imbrication.

factors which have largely been operative in bringing about the popularity of an operation that unites red muscle to white fascia and thereby violates the basic tenet that fascia must be united to fascia to insure permanence in reconstrutcion.

In 1924 the writer devised an operation for the radical cure of inguinal hernia that was suggested by the methods described by both Warbasse (2) and Binnie (3), but differing from either method by following the basic precept of suturing fascia to fascia instead of including red muscle and thereby weakening the repair. This method has given such uniformly good results in my hands during the past 12 years that I am using it to the exclusion of all other methods and recommend it be employed and commented upon by other medical officers.

Description.—The usual incision is made parallel to and about one-half inch above Poupart's ligament extending from the internal ring to the center of the external ring. The aponeurosis of the external oblique is cleared of superficial fascia after which the aponeurosis is slit in the direction of the fibers by first making a small incision over the internal ring, then continuing the separation to the external ring, exercising care that the ilio-inguinal nerve be not injured. The inferior leaf of the aponeurosis is dissected free on either side, outward and downward, to the shelving edge of Poupart's ligament. With the exposure of the inguinal canal, the sac is sought for, and isolated from the cord, by blunt dissection. The cord is handled gently and permitted to remain in its bed. The sac is now opened, adherent contents freed, and returned to the abdominal cavity. A finger is inserted into the sac and the contiguous abdominal wall explored from within the peritoneal cavity for evidence of a direct inguinal hernia. In the event a direct hernia is discovered, an attempt is made to join its sac with that of the indirect hernia. This is not a rare occurrence, but is frequently overlooked and is then a prolific source of recurrence of hernia. In either case the peritoneal wound is closed with a continuous suture as in closing any peritoneal wound. The free ends of this suture are passed through the internal oblique muscle from underneath and loosely tied over the muscle. The free edge of the superior flap of the external oblique is now secured to the shelving edge of Poupart's ligament by four or five mattress sutures. All mattress sutures are placed and then tied consecutively, beginning with the lowest suture, the knots being placed on the outside of the inferior flap on a level with the shelving edge. The lowest suture is so placed that when tied the external ring fits snugly about the tip of the little finger. The free edge of the inferior flap is now secured to the upper flap of the external oblique by a continuous suture, thereby affecting an

imbrication of the fascia of the external oblique over the inguinal canal. The skin and superficial fascia are closed by interrupted sutures of silkworm gut that act both as retention and approximation sutures. No drainage is used. The patient remains in bed from 18 to 21 days and returns to duty in another week or 10 days. From the foregoing description we note that the cord is not transplanted, receives a minimum amount of handling, red muscle is not used in the repair, but rather white fascia to white fascia, which satisfies the basic requisite to insure permanence in reconstructive operations for hernia.

REFERENCES

(1) Bickham, Warren S. Operative Surgery. W. B. Saunders Co., 1924.
(2) Warbasse, James P. Surgical Treatment. W. B. Saunders Co., 1919.
(3) Binnie, John F. Operative Surgery. P. Blakiston's Son & Co., 1921.

THE MEDICAL DEPARTMENT OF THE U. S. S. "RANGER"

By George C. Rhoades, Commander, Medical Corps, United States Navy

The first *Ranger* was built and commissioned under Capt. John Paul Jones in 1777 by authority of the Continental Congress. She was the first man-of-war to hoist the new national American flag, and was the first to receive an official salute to it by a foreign nation. The second *Ranger* was a small schooner purchased in Baltimore in 1814. The third *Ranger* was a brig of 14 guns, purchased in 1814 and attached to Commodore Isaac Chauncey's squadron on Lake Ontario. The fourth *Ranger*, launched in 1876, was an iron ship with auxiliary sail power. The fifth *Ranger* was a battle cruiser, authorized by Congress in 1916 and while still on the ways was broken up and scrapped in accordance with the treaty for the limitation of naval armaments, negotiated in Washington during 1922. The present U. S. S. *Ranger*, the sixth so named, is the first vessel of the United States Navy to have been designed and constructed as an aircraft carrier. Previous ships of this class have been converted from other types. Weight has been eliminated by the use of light alloys and other refinements of design and fabrication of structural material. The result is a very large ship for her displacement. She was built by the Newport News Shipbuilding & Dry Dock Co. at Newport News, Va., and was commissioned June 4, 1934.

During the construction period the ship materializes in the fashion set forth on the blueprints.

The specifications and drawings dealing with the proposed medical department features and spaces have presumably been submitted to

the Bureau of Medicine and Surgery for action. Major modifications are only feasible in the early phases.

The advantages of having a particular officer in the Bureau assigned the duty of participating in the formulation of specifications and plans, of supervising the progress of construction and equipage of the medical department, and of acting as sanitary and hygienic consultant until the future ship's medical officer reports for duty are obvious.

Much data as to mistakes to be avoided, omissions to be remedied, and certain new and desirable features to be incorporated in the medical and other departments of proposed or remodernized ships can be gotten by submitting the plans to medical officers of the same type of ships already in commission or in process of building. Constructive criticism and suggestions for improvements if not advanced spontaneously should be solicited.

A great deal of valuable information would thus be immediately available in the Bureau for communication to and indoctrination of the medical officer assigned to the ship. This useful background will be of inestimable value to him in consolidating his position and will insure uniformity of policy and objective not otherwise obtainable. The assignment of the future ship's medical officer should be made as early as possible.

In addition to the main sick-bay spaces, the number, proper location, size, and equipment of battle dressing stations will require much thought. The number of men at and the location of each battle station, available passageways during general quarters, and conditions of material readiness should be carefully weighed in determining the location and the relative importance of each battle dressing station.

Beyond the physical aspects of the medical department are many problems having to do with the sanitary and hygienic features of the ship. The medical officer's opinion will be asked where the benefit of his professional knowledge and training is essential to a proper decision.

All these factors will call for considerable study, some imagination to anticipate new or anomalous situations, the use of the accumulated information of previous experience—personal or communicated by the Bureau—and the closest and most cordial cooperation with other departments to obtain the best results.

Many features of the original design of the medical department of this vessel were unsatisfactory. A number of modifications were made but due to the advanced state of construction at the time of reporting to the ship it was not practical to effect other desirable changes. Two new carriers were being laid down. The naval con-

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structor in charge loaned the blueprints for the medical departments of these ships, asking for constructive criticism. Sketches and specifications were made for an almost complete rearrangement of the lay-out and for the incorporation of certain desirable improvements of equipment and fixtures.

These plans were submitted respectively to the Bureaus of Construction and Repair and Medicine and Surgery. The personnel of the two Bureaus collaborated to evolve a final and greatly improved solution.

As a result, the medical departments of these ships should be splendid. The earlier this process is initiated when necessary, the easier it will be to effect modifications.

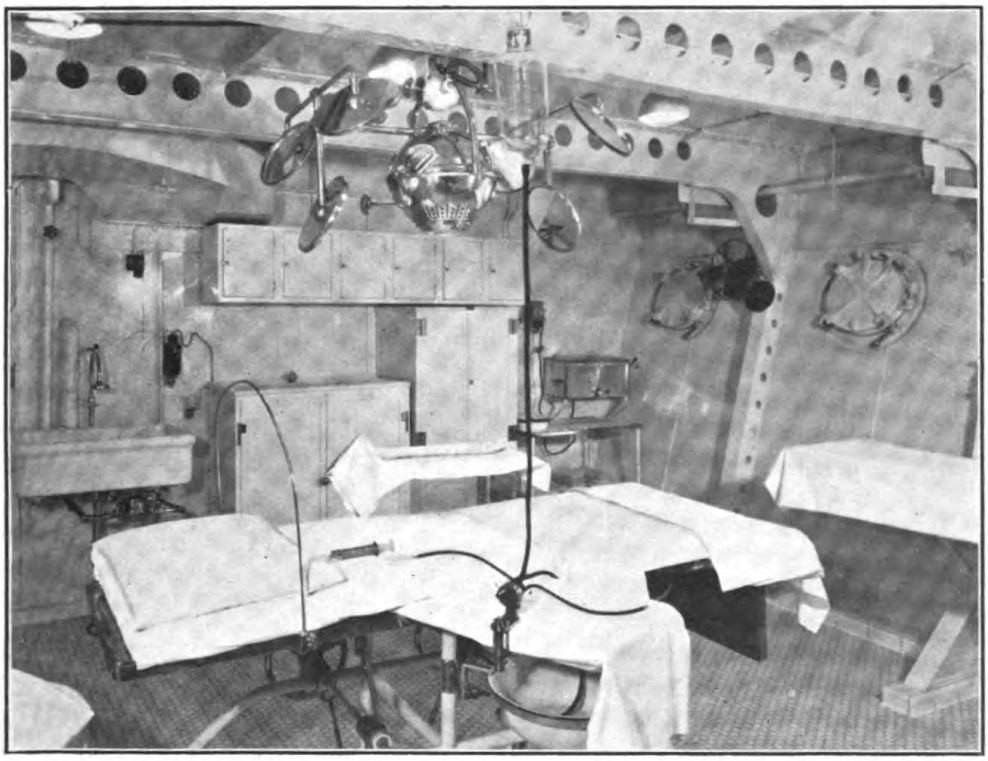
The *Ranger* has 4 medical officers, 1 dental officer, and 16 hospital corpsmen as department complement. When the aircraft squadrons are based ashore one medical officer, a flight surgeon, and three hospital corpsmen accompany them.

The organization of the department is fully set forth in a mimeographed and bound booklet published on board ship. Each member of the department possesses a copy.

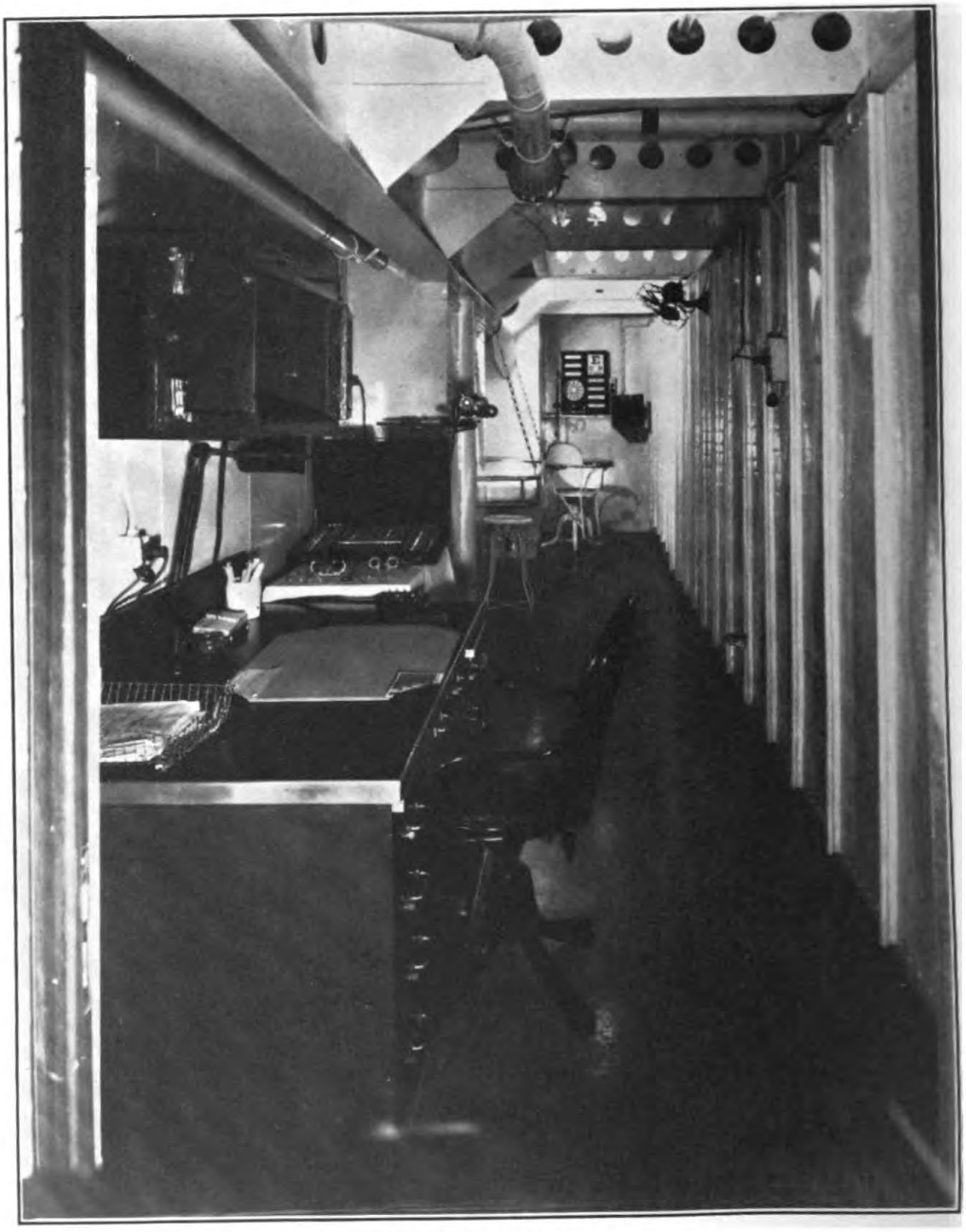
The equipment supplied was generally satisfactory with certain exceptions. The portable X-ray machine is a splendid instrument and excellent films are obtainable. As the available overhead is limited and the various doorways have high sills, the height of the column supporting the shock-proof head or tube assembly and the great weight of the machine practically immobilizes it. This nullifies a designed major characteristic of portability. A machine with a column of an over-all height not exceeding 66 inches and capable of extension to a maximum over-all height of up to 90 inches while retaining its full rigidity, and the installation of no-sill doors in the medical department spaces would restore mobility so that the machine could be taken into the operating room, surgical dressing room, or any part of the ward. Thus the present necessity of carrying seriously injured or sick patients to the machine would be avoided, and the reduction and immobilization of fractures would be greatly facilitated. The Bureau announced recently that a new portable had been adopted. Steps should be taken to insure a sufficiently rigid and stable pedestal for mounting the shock-proof head so as to minimize the vibration and movement incidental to a ship underway.

Other undesirable features of equipment will be noted in detail when the separate activities of the department are discussed.

The main medical department space, occupying a complete athwartship section of the third deck just forward of amidships, is adequate in total area. The accessibility, size, and locations of certain intradepartmental units is not satisfactory.



OPERATING ROOM.



REFRACTION ROOM.

The dispensary, surgical dressing room, dental office, and venereal treatment rooms due to the large number of ambulants treated are, during limited periods, the busiest places in the medical department. On this ship the surgical dressing room and the dental office open at the end of a narrow angulated cul-de-sac remote from the main passageway and very annoying congestion results. It is almost impossible to carry a stretcher into the dressing room. The dispensary, while distant from the main passageway, has sufficient local space to prevent undue congestion. These activities should have been located as to be readily accessible to through passageways and should have had sufficient adjacent free area to prevent congestion during active periods.

Immediately contiguous to the main medical department spaces are the chief petty officer compartments with berthing, pantry, and mess hall space. The mess attendants compartment is just aft. Both have adequate bath and toilet facilities. Thus, a compact integrated hospital unit of very considerable capacity, capable of segregation, can readily be established in an emergency.

No-sill doors throughout the main medical department spaces, especially in the ward, surgical dressing room, operating room, and connecting passageways would be a great convenience in mobilizing the X-ray machine and permitting the use of a wheeled stretcher for intradepartmental movement of seriously ill or injured patients.

The main ward is light and airy and has five airports which can be kept open except in rough weather. The area, cubic feet of space per man, ventilation, and heating are satisfactory. There are 24 standee bunks arranged in 2 tiers, 2 surgical beds, and plenty of locker space.

At the entrance is a large pedestal desk for the corpsman in charge, and a combination drinking fountain and refrigerator, the latter only being large enough to store biologicals and culture media. A much larger refrigerator unit will be necessary to store foodstuffs and supply enough ice for ice caps, etc.

Picture no. 1 shows a view of the ward with several bunks removed in order to display certain features of equipment described in the text.

For the treatment of major burn casualties we have made two canvas tents which can be erected upon the surgical beds within a few moments. They are supported by light wood stretchers connecting the metal T-bars supplied with the beds. The center stretcher carries a battery of electric bulbs for heating purposes. Bottles with enough tannic acid or silver nitrate to make, when filled with water, respectively, 10 percent or 2 percent solutions; the paragin spray pot for use of the above solutions; several hot-water bottles and blankets and the Sorenson air pump, which has been incorporated into a small portable treatment unit, are stored locally.

The entire assembly is shown at the left background of picture no. 1. These beds are fully prepared as a routine procedure during flight quarters, fire drill, and gasoline-fueling periods.

The second surgical bed is shown set up with a fracture frame and necessary accessories which was manufactured on board in order to treat a fractured femur which occurred on our shake-down cruise to South America. Upon the bed is one of the oxygen tents, also manufactured on board, with oxygen and carbon-dioxide flowmeter assemblies supplied by the Bureau of Medicine and Surgery.

The X-ray machine discussed previously is included, incidently, to illustrate the overhead interference to the 84-inch-high column of our machine.

At night the corpsman who has the watch sleeps in the bunk nearest the door. Above his bunk is a lighted sign, "Man on watch", which is clearly visible to persons applying for treatment or venereal prophylaxis late at night. Two other corpsmen also sleep in regularly assigned bunks respectively in the dispensary and the aviation examining room, thus making three men immediately available in an emergency.

The food-tray rack for bunks supplied is quite suitable for its purpose and as a reading and convenience stand for bed patients. Further, when the tray part is removed, a cluster of portable bedside lights can be secured to the rack, the whole covered by a blanket to form a very serviceable heliotherapy unit for local conditions.

A metal box with antidotes for poisons, stomach tube, etc., containing a set of printed instructions for emergency treatment, is kept on hand in the ward.

For the ward bathroom we were able to obtain a sitz bath. This unit has been valuable in treatment of a variety of conditions involving the pelvic and perineal regions. A urinal has been installed which has been a great convenience. Both should be standard equipment of larger ships.

The isolation ward is a four-bed unit with adjacent bathroom. The two hospital beds originally issued to the ship, and later replaced by surgical beds now in the main ward, are installed here. The use of the upper tier standee bunks is not interferred with.

The refraction room and the quiet room are adjacent, and together make a splendid unit for aviation and other physical examinations. The refraction room is a narrow compartment with a depth of 30 feet. On the far bulkhead are a green vision testing cabinet with remote control switch and a depth perception testing apparatus. A large trial lens outfit, trial frames, streak retinoscope, ophthalmoscope, phorometer, Barany chair, stowage locker, and a doublepedestal desk, which is assigned to the flight surgeon handling the squad-

rons, complete the equipment. The quiet room is not suitable for its designed purpose. It is too small, has not toilet or lavatory accommodation, is on a busy passageway, and is now used as an examining room. On the new carriers now building the quiet room will be a two-bed and bathroom unit, off the main passageway, and with soundproof bulkheads.

The dispensary has, of course, the regular equipment supplied capital ships. As a large number of intravenous medications, antiluetic particularly, are given, all major ships need a still to prepare proper water for this and other purposes. The Department approved the furnishing of a Bernstead automatic electric still, capacity of a gallon an hour, which has been highly satisfactory. I consider it one of the most valuable pieces of equipment we have, and believe that a still of this character should be furnished as standard equipment.

A standee desk, originally in the doctors' office, was moved here and placed close to the Dutchsill door. As considerable clerical procedure is connected with the issue of medicines, it is a really desirable and even necessary accommodation for a dispensary in a large ship. To accommodate large stock bottles for solutions in constant demand, we had fabricated a bottle rack providing stowage for four 5-gallon bottles above, and six 1-gallon bottles below. It fits on top of the small safe locker for narcotics.

A large stainless steel drainboard was fitted to the porcelain hopper which has been a help in drying glassware, etc. The glassware rack supplied is fastened to the bulkhead above the tray but is too flimsy, and should be replaced by a sturdier type. The large sink with drainage tray should be standard equipment, the bottle rack and standee desk are highly desirable.

The laboratory.—Space allotted is small but adequate. The equipment is generally satisfactory. A serological water bath working at 56° C., and an enclosed electrical centrifuge were approved by the Bureau. The small electric centrifuge and small copper kettle for heating serums while usable, were a nuisance; especially the centrifuge. In the cramped quarters of the laboratory the unprotected tubes when in motion were only slightly less dangerous than an airplane propeller. A greater amount of more uniform and accurate work is obtainable in shorter time with the new equipment. By using the incubator at 37° C. for precipitin and agglutination tests, and the 56° C. waterbath for softening up the nucleoproteins of the serum for the Kahn test, we have a satisfactory layout.

The operating room is and should be an ample and unincumbered space. In the original plans the furnishings and equipment were so located as to make it impossible to segregate sterile and nonsterile areas during operations. This was remedied by spotting all utensil cabinets, the small instrument sterilizer, and the scrub-up sink on

the after bulkhead, and relocating the head of the operating table further inboard. There is room for the anesthetist, and sufficient passage space between him and the inboard bulkhead, for nonsterile assistants passing to and from the sterilization room doorway, at the forward inboard corner. This arrangement leaves the forward outboard sector capable of being maintained as a sterile area. As the entrance to the operating room is in the after inboard corner, you can be all set up, all personnel of the operating team sterile and clear of the bustle of getting the patient on the table and prepared. The entrance doorway should be a wide no-sill affair to permit the X-ray unit, and a wheel stretcher, to be brought in.

The surgical instruments supplied make little provision for fracture treatment. The characteristics of various individual instruments are unsatisfactory. The Allis forceps are particularly bad, having extremely rigid shanks, and when even the first notch of the locking device is engaged the effect is to puncture or even amputate the part grasped. Other haemostatic forceps suffer from the same fault. A revision of the contents of the various instrument rolls as to selection and quality would be an excellent idea.

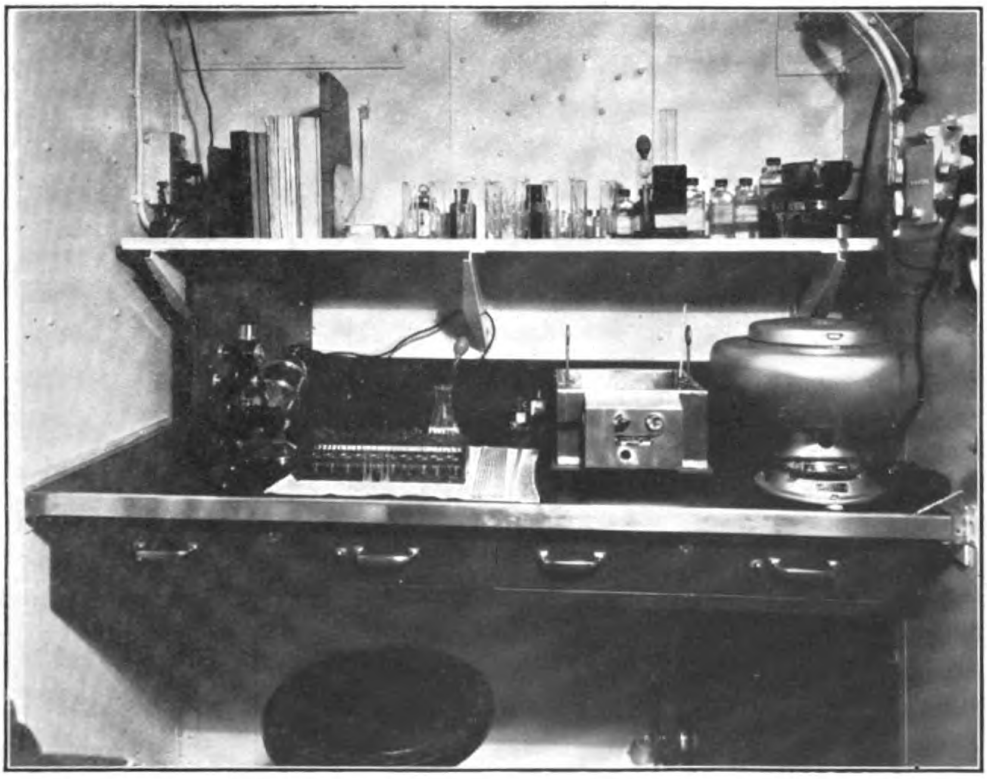
The equipment routinely supplied is unsatisfactory in many respects. The three monel metal-topped tables have not sufficient surface area for a good set-up, and due to the flush top, the gear placed on them slides off in a rough sea, when the ship rolls and pitches. To remedy this we had two large trays, with upturned edges 2 inches high, made and installed on the outboard and forward bulkheads. In each is a circular opening to hold a basin. Each tray is set out from the bulkhead a distance of 6 inches, and there is a clear drop around the back side of 12 inches. This gives you a secure and ample lay-out.

Originally a small hand lavatory was the only means of scrubbing up. The Bureau approved the application for a surgical sink with central levers operating at knee height.

There should be steam radiators for heating purposes. Heating by ventilating air ducts introduces much dust, and creates an undesirable turbulence of the local atmosphere.

The multibeam operating light is not satisfactory due to structural limitations. With a patient on the table the distance from the fixture to patient is 24 inches, much too short for proper focus, and so low that we have broken several of the glass flanges which are placed around the lower lens to catch dust from the lamp interior, by head-on collision, damaging alike to head and glass. A second bad feature is that while we can angulate it as we desire longitudinally on the table, transverse angulation throws the illuminated field outside the operating area. Due to overhead girders and restricted floor area no effective means of overcoming this disability has been found.

SURGICAL DRESSING ROOM LOCKER.



LABORATORY.

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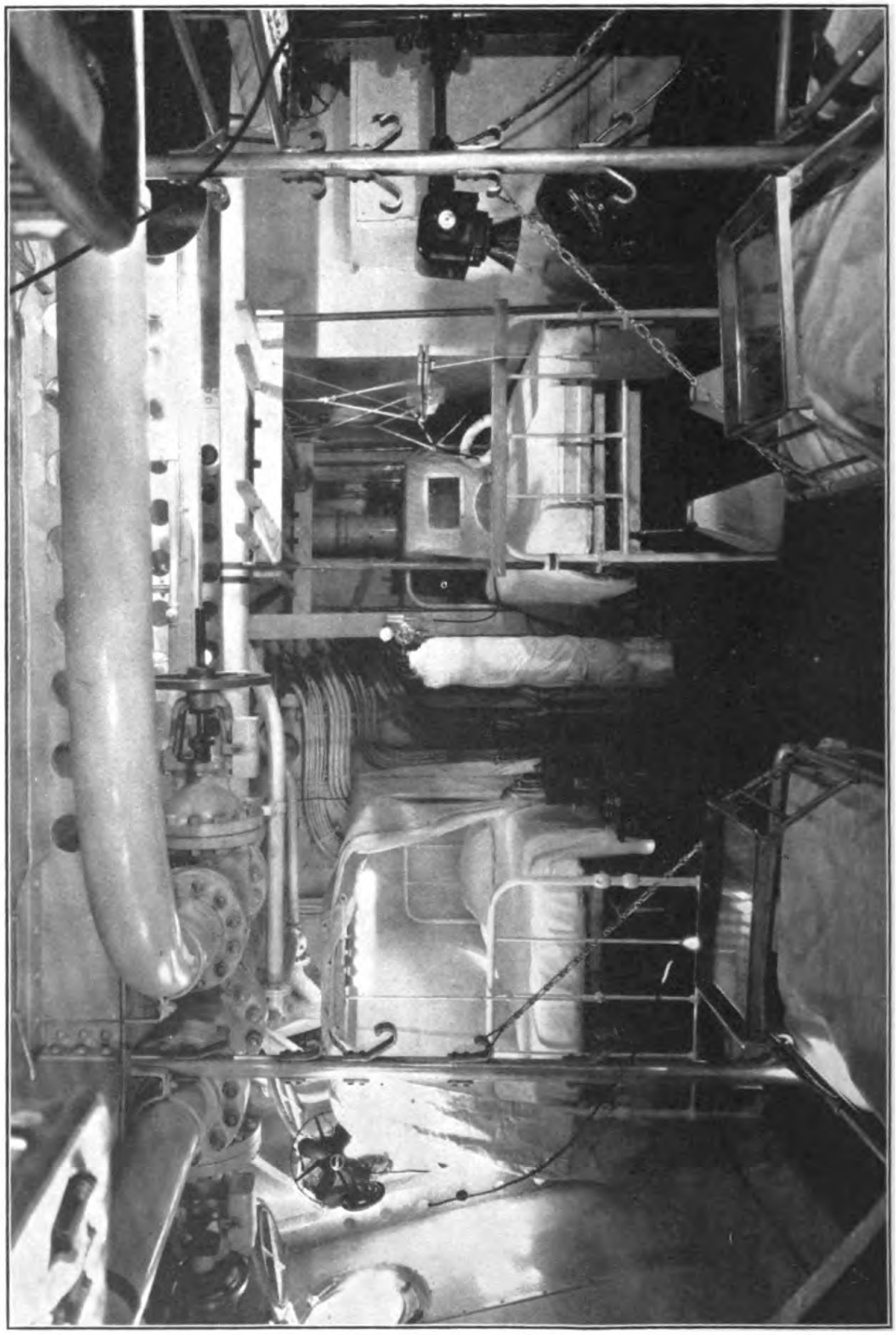

WARD AND SURGICAL BEDS.

A light with shorter focal distance that could be fixed more snugly against the available overhead and still be capable of reasonable mobility should be sought.

A blood transfusion apparatus has been fabricated on board. The machinists made several structural improvements of the model loaned by the originator. It is the simplest, sturdiest, and most foolproof instrument of its type I have seen. A report is published elsewhere in this issue.

The sterilizing room is a small space well furnished with a thoroughly satisfactory equipment.

There is no necessity for the operating room to be outboard. Artificial light will be used, and such natural light as might be present even in bright daylight would be excluded when the battle ports are closed during general quarters. As the surgical dressing room is a busy place with men using it at all hours, and the operating room is a relatively idle place, the former should be outboard for air and sunlight.

The surgical dressing room is the busiest area and paradoxically the least accessible in this ship's medical department. It should be outboard, easily accessible to normal traffic routes, and the outside passage space wide enough to reduce congestion to a minimum. In size it should be as near to 12 by 18 feet as space permits, and its equipment should be sufficient to permit proper handling of at least the initial phases of major surgery of traumatic origin.

If feasible it should connect with the ward and passageway by wide no-sill doorways, to permit the mobile X-ray unit to be brought to the patient in emergency cases, and to facilitate his transfer by a wheel stretcher to a ward bed.

Various therapeutic apparatus such as diatherm, helio-therapy lights, and baking ovens should be located here.

There should be a surgical scrub-up-sink here because the facilities for a quick clean up are at a premium.

No original provision was made for stowage of the large number of bottles and jars of medications and the dressings and other apparatus ordinarily associated with this activity. A locker designed by T. Duer, pharmacist mate, second class, and built in the navy yard has proved a very effective unit and should be made standard equipment for all ships. If a sliding metal tray with raised edges could be incorporated it would be ideal. When the doors are closed it is secured for sea.

The portable X-ray developing room had no provisions for a drying rack nor local storage of supplies. As the battle dressing storeroom was immediately adjacent to the photographic laboratory we installed the tank, fan, and lights here and had a storage locker and a drying rack built. The photographic laboratories Frigidaire fresh and salt

water cooling system was tapped. This eliminated the necessity of constantly iceing the developing tanks and gave a very satisfactory unit. The cabinet is being used as a storage locker in the forward dressing station.

The venereal treatment room has two trough units in a well-equipped and accessible space. One side is used for treatments, and the opposite for prophylaxis. A high stainless steel back board has been added for protection of the bulkhead. To this are attached racks of tubes each holding a patients own syringe or irrigating tip. The medications used are bottled and hung in a rack with a medicine glass in front to hold the solution.

There is a foot operated porcelain hopper basin. The welfare fund supplies paper towels so each man may cleanse his hands before leaving.

A small folding bulkhead desk is desirable for record keeping.

The dental office is a modernly equipped two-chair unit in a spacious compartment containing three air ports. It was originally furnished with a dental X-ray with oil immersed tube. While cruising in the vicinity of the Equator, due to the marked degree in change in temperature, this machine was found to be useless, as it had no expansion bellows in the shockproof head. It was later surveyed and a Victor CDX dental X-ray obtained. The dental office unit is so located as to be very inaccessible and much conjestion results when people using the surgical dressing room and the dental unit are jammed in the small cul-de-sac that serves as common access to both.

Battle dressing stations.—Each airplane carrier is a problem unto itself. The number, locations, and equipage of dressing stations and the tie-up with the main stations will be governed by the following factors. The number should be the least possible to adequately cover the various major regional distributions of men at battle stations. The location of each station must as far as possible be indicated by the immediate availability of living spaces with bunks and toilet facilities; and, by the freest possible accessibility to its coverage area under conditions of general quarters or material readiness. The equipment and personnel assigned must be suitable for the estimated number of casualties the station may have to handle and its accessibility to the main station for evacuation of patients and replenishment of supplies.

Advanced patrols of men trained in first-aid work and furnished with suitable dressing materials should be established if sufficient personnel are available. Prudent distribution of assets during action should be provided for, in order to minimize the result of departmental casualties.

Seventy-two lockable metal boxes, equipped with first-aid materials, are spotted to bulkheads at battle stations remote from dressing stations, and in spaces where accidents, during ordinary work, are most likely to occur.

A mimeographed booklet with first-aid instructions, and suitably illustrated, has been prepared for distribution to division officers. This booklet is to supplement lectures and assist them in the instruction of division personnel.

The medical storeroom space provided, while outside the main sick-bay area, is adequate, convenient, and is accessible at all times.

The doctor's office has three flat-topped desks, a transom, and a large safe locker for alcohol and narcotics. A three-section bookcase was obtained from Bureau of Construction and Repair to replace the shelf originally provided. This was necessary to properly stow and protect the books and periodicals. It should be standard equipment for larger ships.

The clerical office has two typewriter desks, adequate filing cabinets, and a comfortable transom.

Hygienic aspects of ships.—Living spaces for personnel should afford the maximum comfort obtainable. Space, ventilation, heating, bunks, lockers, washing, berthing, and toilet facilities are structural details peculiar to each ship. Messing, satisfactory issue clothing, lounging, and recreational facilities are further material factors of importance in maintaining morale.

Living compartments.—Compartments of stated location, size, and shape in which it is desired to accommodate a certain number of men give rise to many problems. The number of men that can be accommodated in comfort depends upon reciprocals. Beyond the physical possibility of being able to install so many bunks and accompanying lockers is the livability, the ventilation, and heating of that compartment.

As long as the chemical characteristics of the air are not objectionable from contamination or excessive vitiation, personal comfort or discomfort from ventilation is principally the effect of the temperature, humidity, and local circulation of the supplied air upon the heat regulation mechanism of the body. Clothing, exertion, and diet are modifying factors. While the quality of air taken as a whole for compartment may be satisfactory, if its local agitation about a resting individual is deficient, relative discomfort results. In effect he is enveloped in a capsule of deteriorated air.

In living compartments on board ship it is manifestly impossible to assign a space of 600 cubic feet per capita. Therefore the smaller space available must have sufficient air changes per hour to supply an acceptable amount of air. This reduction of space is limited

by the draft resulting from the necessarily increased rate of air change. Excessive draft is as objectionable in its ways as none at all. When the number of changes per hour exceeds six, perceptable drafts are set up. The greater the excess changes, the more annoying and uncomfortable it is, particularly for those situated close to terminals and this is especially true when the heaters are on. A widespread distribution by many inlets will reduce local draft and give a more even diffusion. Injection via a few terminals causes much noise, strong local draft due to the necessarily high velocity delivery rate and less uniform distribution. When this draft, noise, and heat become too aggravating, men sleeping in bunks adjacent to terminals will shut the damper or plug the opening. While this renders them more comfortable, others in remote areas suffer.

The spotting of lockers in relation to bunks in a compartment has to be considered from two angles. Locker banks scattered through the compartment and intermingled with bunk rows, while a definite convenience to the men, have a tendency to fragment the area and impede uniform air distribution unless a large number of terminals are installed. If the lockers are segregated the bunks can be uniformly ventilated with fewer outlets, but considerable individual inconvenience is a result. However, if a little care is taken a satisfactory compromise can be reached.

Mechanical or artificial ventilation is usually planned as a closed system with forced introduction of air, conditioned as necessary, and utilizing fixed natural escapements, or, if these are inadequate or not suitably situated, positive exhaust ducts. This latter contingency occurs most frequently in compartments not having an overhead escape through a hatch leading to topside. As the top of the watertight doorways is approximately 15 to 18 inches lower than the overhead deck the upper stratum of air is trapped and men in upper bunks are affected. Occurrence of variants, such as the opening of hatches ordinarily kept closed or opening of airports, tends to lower the efficiency of the system unless an alternate selective damper-controlled terminal arborization is provided to meet this alteration.

Special study must be made to adopt the system to conditions of material readiness when many of the escapements ordinarily available are closed during considerable periods of time.

The capacity of the ventilating and heating equipment should be sufficient to supply the full needs of all compartments served while running at an ordinary efficient operating rate. Provision for natural ventilation or local air renovation in case of machinery failure must be provided for in vital operating areas.

The heating of smaller compartments is usually not difficult. Large thwartship areas, particularly in severe weather, are difficult to heat

and ventilate uniformly with single air conditioning and heating units. Supplementary radiators outboard or separation of inboard and outboard systems should improve this deficiency.

Wild heat is frequently a very troublesome complication in living quarters. In most ships insulation of the side to reduce sweating or condensation helps incidently in keeping out some of the external heat or conserving interior heat, but there is apparently little or no serious effort made to control the escape of internal wild heat from machinery or boiler rooms into living spaces. Complicating the heat is usually a concurrent influx of fume contaminated air from the same areas.

Proper insulation of sides, bulkheads, and where necessary, the deck and overhead would keep these areas cooler in warm weather and permit more uniform heating and ventilation in winter. Access hatches to machinery spaces should if possible be not located in living compartments or if this is unavoidable, should be kept closed when not in use for passage and an exhaust vent should be located immediately adjacent in order to trap the fumes before they become diffused throughout the compartment.

The newer ships are all provided with adequate individual locker and group overcoat locker space which enable the crew to keep their clothing presentable and their other belongings in relative security. The widespread use of the ship's laundry should be encouraged by the most frequent turnover and the lowest possible prices.

Sanitary facilities.—The number of toilets, urinals, washing places, and scuttlebutts is determined by definite ratios to the number of men in the crew. These facilities should be readily accessible to and equitably distributed among the living compartments with suitable provision made for working areas, and be available insofar as possible during periods of material readiness. A bulkhead mirror with a small shelf below should be installed over each washing space.

Pernicious and often ridiculous restrictions of water, ventilation, heating, lighting, and sanitary facilities have been permitted in the name of a false conception of operating and engineering efficiency. It was very gratifying to read a recent letter from the commander battle force condemning these practices and ordering them eliminated.

Morale facilities.—Provision should be made in the living spaces for the convenience of men off watch. Properly illuminated areas with seats and a table for reading or writing should be installed as permanent fixtures as the ship's library is seldom sufficiently large and usually not readily convenient to all compartments. These items have been installed in this ship and have proved very satisfactory.

Where space is available separate messing and sleeping compartments are desirable. The cafeteria system if efficiently operated is best in many ways.

The lower deck berthing compartments on the *Ranger* showed the following averages: (1) 131 cubic feet of free space per man with a maximum of 176 cubic feet and a minimum of 68 cubic feet. (2) Ventilation input of air of 2,218 cubic feet with extremes of 1,500 cubic feet and 2,850 cubic feet per man per hour. (3) Rate of 17 changes of air per hour with variables of from 10 to 27 changes.

The superstructure berthing compartments have appreciably more space per man, adequate mechanical ventilation, and are capable of natural ventilation at all times short of a toxic gas attack. The sole disadvantage except from a relative sanitary-convenience deficiency, is a tendency for excessive heat in the Tropics, or during the summer due to the flight deck being the overhead in the three compartments on the highest level.

Generally, the lower deck living compartments are badly pocketed due to irregular area, through trunks, machinery, or the concentration of lockers in solid banks, between sections of bunks.

An acceptable minimum of 1,800 cubic feet of air per man per hour, with 125 cubic feet of free space per man and a consequent atmospheric change of 14 times per hour, has been found practical by partially vacating the compartments with inadequate space and ventilation. The tailor and cobbler shops have been shifted, and a number of bunks in underprivileged compartments have been removed in this desirable area and to new locations in other suitable vacant spaces. Without the squadrons personnel aboard these compensatory measures have improved conditions markedly. The above averages hold good with ships crew and all squadron personnel aboard and in the compartments as at least one section of the crew is on watch at all times while at sea, even with practically all squadron people turned in at night, the mean of the above figures for free space and air are increased 20 to 25 percent per man. The squadrons are only aboard at intervals and when not abroad the quoted figures are then increased by 30 to 35 percent per man.

Ventilation, with humidification and heating of the supplied air in cold weather, is accomplished by thermo-hygro-statically controlled automatic mechanism. Air is delivered at near deck level by outlets placed, mainly peripherally in relation to the usually central natural escapements of hatches, and where none such are effectively available, a positive exhaust duct is provided. The system is apparently designed to operate most effectively with outboard ports closed. The opening of ports shortcircuits the escape of vitiated air by designed routes, and leaves stagnant inboard conditions.

Even with the ports closed, the fragmentation of the compartment, by obstructive locker banks, and equivalent irregularities, prevents an equitable distribution of the supplied air. A superior design would seem to be an unobstructed bunk space with lockers around the bulkheads, and air delivery ducts spotted in proper relation to natural air escapements, or positive exhaust vents. If it is considered desirable to keep the lockers intermingled with the bunks, additional outlets suitably placed are needed. As these outlets would be in excess of the rated capacity of the blower, a suitable dampering mechanism capable of being locked in place on each outlet would permit optional distribution of air to meet varying conditions.

As a result of the small average cubic footage per man and the relatively small number of outlets, in order to supply sufficient air, the delivery velocity is high. This velocity produces a turbulent flow with increased friction-resistance in the ducts tending to heat the air. In warm weather this is particularly noticeable. The turbulence also causes considerable noise.

As the hygro-thermostat control device can only be placed in one situation, and as there is considerable variation in remote areas, the heating and humidification is not uniform. Thus, in cold weather, if the people near the shell have comfortable heat and humidity, the inboard people are too hot, and the air is relatively dry, or if the inboard sleepers are happy, the outboard unfortunates are cold and relatively damp.

A possible solution would be supplementary outboard radiators or a split, dual-control machine, with proportioned separate air modification chambers, and duct arborization for outboard and inboard areas, respectively. It is believed that this alteration could be made without significant increase in size or weight of the present installation. Thus, each hygrothermostat would control its proper area, and reciprocals could be established for each compartment in the light of experience.

The only present practical means to remedy this defect have been undertaken. Thermometers have been installed in each compartment. A patrol has been formed which visits each compartment at hourly intervals. This detail manually adjusts each machine in order to regulate heat and humidity within comfortable limits. They also inspect air ducts to see that dampers are not shut. The present thermometers are difficult to read. It is advisable that easily readable wet and dry bulb thermometers be substituted. This type will permit both thermal and humidity curves to be plotted, for the guidance of the lower deck patrol, in regulating the ventilating machines. Relocation of certain bunks, lockers, and ventilating ducts is gradually being accomplished.

The engine testing stand is on the open space of the hangar deck. Fumes will be carried overboard by the propeller steam.

Twenty-seven dental lavatories are installed in the various crew washrooms. Very few persons use them, preferring to complete their toilet before the mirrors and shelves above each washing trough space and regular lavatory. The major use seems to be for casual washing of small items such as socks, etc. The fixtures are fragile and frequently out of order due to breakage. As they are not used they are not necessary. These items have been removed.

It is recommended that these fixtures be dropped. Further, in view of the experience on board this vessel, installation of these units on future ships seems an inadvisable expenditure.

The cafeteria system of food service is used. There are five steam tables strategically placed in the messing compartments. Men coming in pass the racks containing compartmented china plates, the only item of mess gear he has to carry, go to the steam table, and thence to a convenient mess table. The serving line is so routed that the distance to a table is short and unimpeded. Cups, bowls, cutlery, with pitchers and pots containing beverages and soups, are placed on all the mess tables just before meals are piped.

Thus, hot foods are served hot, and eaten hot. Late comers get the same food as the earlier men. Mass likes and dislikes can be ascertained. Due to the consequent control of waste, a better ration is supplied.

The men pronounce it to be the best feeding ship they have served on. A thousand men can be served in from 12 to 18 minutes, depending upon the number of courses. The food is of excellent quality, wholesome variety, tastefully prepared, and properly served.

This, a great morale feature, is the result of several factors—a splendid commissary officer, backed by a competent and hardworking staff, and the carefully thought out arrangement, and management of this cafeteria system.

As a result of experience on the *Ranger* the following items should be given consideration in the plans and during construction of new vessels or the modernization of older ships.

Medical department.—

(*a*) Operating suite: Surgical scrub-up sinks; proper shelfs or tables for the set-up at operations; steam heating; more suitable operating light; better arrangement of furnishings in order to utilize the available area to the best advantage; and, the desirability of assigning an inboard location.

(*b*) Surgical dressing room: Adequate size and ready accessibility; adjacent to ward; surgical scrub-up sink, and dressing locker to be made standard equipment; desirability of outboard location; and adequate equipment for handling initial phases of emergencies.

(*c*) Dispensary: Make electric still, drainboard, stand-up desk and large stock-bottle rack, standard equipment; locate for accessibility.

(*d*) Venereal treatment room: Higher backed troughs; paper towels standard equipment; ready accessibility.

(*e*) Dental Office: Ready accessibility.

(*f*) Ward: Sitz-bath standard equipment.

(*g*) X-rays:

(1) Mobile X-ray unit: Provide machine which will not be restricted as to location and mobility by low overhead.

(2) Dental X-ray: Provide a machine, that efficiency of which will not be impaired by marked changes in temperature.

(*h*) Wide no-sill doors in ward, operating room, surgical dressing room, X-ray room and intermediate passageways.

(*i*) Diet pantry on larger ships.

(*j*) Battle dressing stations: Location, size, and equipment, in relation to casualty covering, and accessory accommodations of living spaces.

Extradepartmental activities.—(*a*) Investigate the feasibility of separating outboard and inboard ventilation, in order to minimize heating diffierentials in hot and cold weather; and, the spotting of supply and exhaust terminals in such relation to available escapements that uniform atmosphere conditions can be obtained.

(*b*) Attempt to provide living compartments in which the distribution of cubic feet of space and of air supplied per hour will be uniform and adequate.

(*c*) Control the escape of wild heat and contaminated air from the boiler rooms, machinery spaces, laundry, galley, into living compartments.

(*d*) Elimination of dental lavatories.

(*e*) Investigate the feasibility of using the cafeteria system of messing.

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NARCOLEPSY

With Report of Three Cases

By H. O. Cozby, Lieutenant, Medical Corps, United States Navy

Narcolepsy is relatively and absolutely rare. Moreover, it is adequately described only in literature for the most part inaccessible. For these reasons it seems pertinent to give some description of the

symptom-complex with a report of three additional cases of typical narcolepsy.

Definition.—Narcolepsy, as defined by Adie (1), is a condition characterized by "the occurrence of attacks of irresistible sleep without apparent cause, and curious attacks on emotion in which the muscles relax suddenly, so that the victim sinks to the ground fully conscious but unable to move."

History.—The infantile fat boy with hypersomnia of Dickens' Pickwick Papers (circa 1837) must have been painted from life. However, Westphal in 1877, Fischer in 1878, and Gélineau in 1880 were the first (2) in medical literature to describe short attacks of irresistible diurnal sleep. Gélineau gave us the term "narcolepsy" in 1880, speaking of the symptom-complex as a "new species of neurosis"; his patient would suddenly go to sleep under emotional excitement and his legs would give way when he burst out laughing ("riait aux éclats"). In 1886 Foot (3) proposed the term "hypnolepsy" for the attacks of sleep; however, the sleep manifestations are now generally called the narcoleptic attacks. Loewenfeld (4), in 1902, laid emphasis on the double symptomatology and again called attention to the sudden attacks, under the influence of emotion, of loss of postural tone not associated with loss of consciousness. Henneberg (5) further investigated these attacks of weakness and tonelessness and referred to them as cataplexy or the cataplectic attacks, the terms now in use. Two early American reports were those of Morton (6) in 1884 and Ewen (7) in 1893. From a military viewpoint Major Ewen's case is especially interesting as it occurred in a soldier who was charged with falling asleep on sentry duty.

Etiology and pathology.—From Gélineau to the present, much confusion and lack of discrimination has existed in the use of the word "narcolepsy." It has been applied to all variations and degrees of somnolence, hysteria, neurosis, and epilepsy. Loewenfeld (4) and especially Adie (1) regarded idiopathic narcolepsy as a disease sui generis. Among many are Wilson (8), Weech (9), Worster-Drought (10), and Cave (11) who recognize the idiopathic form but use the diagnosis in a symptomatic sense and believe the condition to be a syndrome or symptom-complex. Wilson (8) prefers to speak of the group as "the narcolepsies." From the first observers through Gowers (12) and many others (13) the close relationship between narcolepsy and epilepsy has been emphasized and it has often been classified as an anomalous or borderland epileptic and convulsive type of reaction. Clinically, however, the two conditions are not readily confused.

Narcolepsy, idiopathic, and symptomatic, is not common and the total recorded cases in the medical literature of the world is considerably less than 200. Adie (1) in 1926 reported 6 per-

sonal cases with a précis of 15 cases in the literature. Wilson (8) in 1928 reported 5 personal cases and found 38 in the literature, a total of 43. Levin (14) in 1929 added the cases in the literature up to 66 and reported 5 cases of his own. The records of the Mayo Clinic (11) showed 42 cases during the years 1919 to 1928, inclusive; subsequently 25 additional cases were reported (15).

Narcolepsy affects males to a striking extent. Combining the series of two writers (8 and 11) the ratio is found to be 71 males to 15 females. The average age at onset was 23½ years for Adie's (1) 21 cases and 24 years for Cave's (11) 42 cases. The symptoms of the disease usually reach their maximum intensity a short time after onset and remain throughout life, though remissions are described. Doyle and Daniels (15) mention cases in which the symptoms have persisted for more than 40 years. The prognosis, therefore, as regards life, is good.

The pathology of idiopathic narcolepsy is unknown, for no case has been reported as coming to autopsy. Most writers implicate the region of the hypothalamic centers as the most probable site of the pathology. Sleep and muscular tonus are among the functions probably initiated and regulated by the hypothalamic structures; we know that the red nucleus, the basal ganglia and the thalamus are related to muscular tonus and the emotional life. Symptomatic narcolepsy has been reported as occurring with wide variety of pathological states; e. g. cerebral tumors, abscesses, and new growths, head injuries, epilepsy, circulatory disturbances including hemorrhage and the arteriopathies, endocrine and metabolic disturbances including diabetes insipidus and hyperinsulinism (16), and those toxic or inflammatory and infectious conditions of the nervous system involving the structures surrounding the third ventricle, especially epidemic encephalitis (17). Wilson (8) called attention to the similarity between the cataplectic attack and periodic family paralysis. Numerous observers, notably Wilson (8), Worster-Drought (9), and Cave (11) have reported abolition of deep tendon reflexes, loss of muscle tone and an extensor plantar response (Babinski) during the narcoleptic and the cataplectic attacks.

Symptomatology.—The two aspects of the symptomatology, characteristic and stereotyper, are (a) the narcoleptic attack, an irresistible and overpowering desire for sleep to which the patient ordinarily succumbs, and (b) the cataplectic attack, a sudden loss of postural tonus upon emotional stimulation not attended by loss of consciousness and during which the patient sinks helplessly to his knees or into his chair. The depth of consciousness varies and transitional forms are common. These, depending upon the degree of consciousness, seem to be midway between sleep and cataplexy. Numerous

observers believe that a diagnosis of narcolepsy may justifiably rest on the sleeping attack without cataplexy. Wilson (8) says, "All gradations are met with, from narcolepsy without cataplexy, to cataplexy without narcolepsy."

Treatment.—Among the many drugs used in the treatment of narcolepsy none were satisfactory until the use of ephedrine sulphate (18) in 1930. Subsequently (15) 50 cases from the Mayo Clinic series treated with ephedrine sulphate showed complete relief in 20, marked improvement in 17, and moderate improvement in 8. The average dose was 25 mg three times daily, increased in a number of instances to 50 mg and in one instance to 75 mg thrice daily. In two of my patients ephedrine was used with considerable sypmtomatic relief while taking the drug. Prinzmetal and Bloomberg (19) have recently reported the use of benzedrine in the treatment of nine cases of narcolepsy with complete relief in seven and almost complete relief in two. They conclude that benzedrine profoundly stimulates the higher cerebral centers and is approximately three times more effective than ephedrine in the treatment of narcolepsy. I have not used benzedrine in the treatment of narcolepsy but in one patient with undue drowsiness and daytime somnolence it has worked efficaciously to keep him awake and mentally alert.

CASE REPORTS

Case 1.—W. T. H., high-school student, white male, age 15. Examined at the United States Naval Hospital, Washington, D. C., on June 17, 1929. He had been referred for consultation with a tentative diagnosis of epilepsy or encephalitis lethargica. He complained of undue sleepiness and dizzy spells.

The family history and past history were negative.

The present illness began abruptly some 6 months before. While relating a story at a bridge game the patient's head momentarily fell forward on his chest and almost on the table. Upon regaining consciousness in a minute or two he insisted that he had "just gone to sleep." Since then he has had from one to six daily sleep attacks. They are irregular and not infrequently come on at meals. He has great difficulty in studying because of the sleep attacks. He goes to sleep in odd places and at odd times. In the attacks of sleep his eyes close and he seems limp, as though in a faint or asleep. He does not answer for a minute or two if left alone but if touched or shaken he seems to rouse more quickly. He has never become pale, cyanosed, had any aura, shown any alteration of respiration, bitten his tongue, hurt himself, or manifested any evidence of the tonic or clonic phase of the convulsive state. The patient himself described the attacks as follows: "Sometimes things seem confused and mixed-up and I feel sleepy and drowsy. But I know when they ask me a question and what they are doing. I just can't answer or say anything or move. Then I get awfully sleepy several times a day and just can't keep my eyes open. So then I really go to sleep. If I don't go to sleep I get awfully nervous. When I get nervous I feel funny inside, all dizzy and faint and weaklike in my stomach." The attacks of weakness frequently occur independently of the sleep attacks; they are also brought on readily by excitement or anger. Sometimes in a movie he gets "weak, shakes, and

trembles." The weakness attacks are very irregular; sometimes he goes for several days without an attack and will then have four or five attacks within an hour or two.

The physical and neurological examinations were negative throughout. The blood Kahn, urinalysis, feces examination, and complete blood count were negative. An X-ray of the skull was negative. The basal metabolism rate was minus 4 percent.

Treatment with luminal and bromides was unsuccessful. A ketogenic diet was abandoned for lack of cooperation. He continued to have irresistible diurnal sleep attacks and attacks of weakness and dizziness during a period of two months out-patient observation.

Case 2.—H. A. S., seaman, first class, United States Navy. White male, single, age 26 with 5½ years naval service. Admitted to the United States Naval Hospital, Washington, D. C., on December 8, 1931, with a diagnosis of syphilis (central nervous system). He complained of "falling to sleep at any time."

The family history was negative.

The past history was negative for head injury, influenza, or any febrile illness simulating encephalitis lethargica. During his early childhood he had pertussis and measles. At the age of 10 he had mumps followed by swelling of the left testicle. In September 1929 he had generalized spots and blotches on his body and a month later the blood Kahn was reported four plus by the U. S. S. *Relief*. There was no history of a known primary. He stated that he received eight intravenous injections of neosalvarsan in November and December 1929. The health record showed an admission for syphilis on May 13, 1930, at which time he had a three plus blood Kahn, and was complaining of an eruption on his body, hoarseness, and "canker sores" in his mouth. Subsequent to this admission in May 1930 he received during the next 18 months two courses of neosalvarsan (a total of 9 grams), one course of tryparsamide, and two courses of bismuth salicylate.

The present illness began abruptly nearly 5 years before. In January 1927 he went to sleep at breakfast suddenly and without warning, and his head momentarily fell forward into a plate of eggs. In training with the ship's race crew at the time and not having been ashore he had slept well the night before. Without being able to resist he suddenly "just went to sleep without being sleepy." He waked almost immediately and felt all right except for a numb feeling in the back of his head. On the same day while holystoning the deck he went to sleep several times for short intervals, "maybe half a minute up to a minute or two." At first the sleep attacks occurred many times a day; later they decreased somewhat in frequency, and he now has five or six attacks, sometimes only two or three daily. He was admitted to the sick list with a diagnosis of psychoneurosis, neurasthenia, on April 20, 1927, and discharged to duty May 5, 1927. An excerpt from the health-record entry is significant: "Complains of persistent sleepiness. For the past 6 months has gone to sleep during the day while in some place where he may injure himself. * * * Numerous shipmates corroborate his statements." He falls asleep in all sorts of places and under all types of conditions. He has had attacks while crossing the streets in traffic, in street cars, on the subway, in the home of friends, on board ship, while walking on deck, while working, while painting over the side on the stages, while in the foretop, etc. If the sleep attack comes on while he is standing his knees sag and his head droops forward, but he has never fallen. Sometimes he goes sound asleep and at other times just feels very dull and drowsy. When he feels drowsy he is dimly aware of things going on around him. Following the sleep attacks he

always wakes feeling refreshed. If he tries to fight off the attacks and succeeds in doing so he becomes very nervous and restless and "gets the shakes all over."

About a year after the onset of the sleep attacks when about to have a fight with a shipmate, at the climax of the quarrel and in the position of readiness to strike a blow he suddenly found his arm and general body musculature relaxed and powerless and unable to respond to the demands of the situation. The patient stated that as a result thereof, "I got poked twice in the eye before I could hit back." He did not lose consciousness or go to sleep. Even before this dramatic attack he had noticed that in laughing he would lose control of his muscles and "get weak all over." He described these as "nervous attacks" in distinguishing them from the "sleep attacks." He is sure that while he may objectively give an observer the impression of being asleep in the "nervous attack" he never loses consciousness to any degree. He says, "The muscles are just all gone and without any strength. I couldn't lift my little finger to save my life if I had to do it quick." Any intense emotional experience, excitement, anger, laughter or being tickled, will bring on one of these attacks of tonelessness and loss of power. He has in consequence learned to restrain these emotions as much as possible.

The general physical examination showed hypertrophied tonsils, relaxed inguinal rings, and small testicles, the left testicle being definitely atrophic. The height was 68 inches and the weight 161 pounds. The neurological examination was negative throughout. The blood Kahn and blood Wassermann were negative. The spinal fluid showed normal dynamics, Kahn and Wassermann negative, globulin negative, cell count 8, Lange colloidal gold curve negative. An examination of the feces, a complete blood count, and an X-ray of the skull were negative. The basal metabolism rate was minus 5 percent and minus 8 percent on successive examinations.

Dr. Walter Freeman, Washington, D. C., saw the patient on December 15, 1931, and concurred in the diagnosis and in the view that the narcolepsy antedated the syphilitic infection.

The patient was observed in a narcoleptic attack while riding in an automobile on the same seat with the writer, who was driving. He suddenly slumped down in the seat with limp shoulders, eyes closed, and head on his chest, giving the external appearance of sleep. By the time the car could be drawn to the side of the road and stopped he was awake.

The treatment administered was ephedrine sulphate, grs. 3/8, three times daily. During a period of 24 days while taking the drug he reported only three narcoleptic and two cataplectic attacks. Then, while away on leave for 7 days, the drug was withdrawn. He reported only 2 or 3 attacks during the first 2 days but in the succeeding 5 days he had from 8 to 20 attacks daily. He was invalided from the United States Navy on January 26, 1932.

Case 3.—T. H. C., fire controlman, first class, United States Navy. White male, single, age 29 with 10 years' naval service. Admitted to the United States Naval Hospital, Canacao, P. I., on March 20, 1933, with diagnosis undetermined (epilepsy). He complained of drowsiness, lassitude, brief periods of diurnal sleep, and spells of weakness.

The family history was negative.

The past history was negative for any febrile disorder of encephalo-meningomyelitic type and for venereal diseases. He had had the usual childhood diseases. After several attacks of tonsillitis in the service he had had a tonsillectomy in 1929.

The present illness began nearly 20 years before when he was about 10 years of age with a sleeping spell or attack. In school when about to recite one day

he "just went to sleep suddenly." At the time this was accounted for as a dizzy spell. However, as he continued to have the attacks at intervals it was feared that he had epilepsy and he was taken to a physician. As he had never had a convulsion or bitten his tongue the doctor was loath to make a diagnosis of epilepsy.

Sometime later, probably a year after the onset of the sleeping attacks, he began to experience attacks of weakness. These attacks were definitely distinguished from the sleeping attacks by the patient. Since the onset of the weakness attacks he has never been free from attacks of either kind though they have varied greatly in frequency and severity. The weakness attacks are without any sensation of sleepiness, drowsiness, or dulling of the mental faculties. They consist of sudden, brief, extreme powerlessness, weakness, relaxation of the muscles, inability "to move a muscle, not even a finger or a hand." The spells of weakness have come on most frequently under excitement or emotional situations. Sometimes he has gone for a week or more without an attack of weakness; usually he has one or more almost every day; several days ago he had four attacks within an hour. Laughing, being tickled, "balled-out", in a brawl, or angered nearly always brings on an attack of weakness and powerlessness.

The sleeping attacks come on without warning and last "10 or 20 seconds up to 1 to 15 or 20 minutes." During these attacks he is at times vaguely aware of things going on about him and a sharp command or touch on the arm will often "snap me right out of it." The sleeping attacks come on without reference to the amount of sleep he may have had the night before. He has been able to stay awake and fight the attack sometimes, though not always, by walking about and keeping busy. To fight off one of the attacks does not really help much because it makes him feel "fog-bound and loggy." To go ahead and sleep makes him feel normal, clear-headed, refreshed, and as "though relieved of a gas or pressure on the stomach." The spells of sleeping and the attacks of weakness seem to alternate in some degree; when he has the attacks of sleeping he has not been so liable to the attacks of weakness. He has gone to sleep in all manner of places and at the most inopportune times; e. g., while eating, while standing on the street and in a street car, while in the subway and thereby being carried past his station, at inspection, while watching a show, while with his girl, etc. The attacks of sleeping vary in frequency; sometimes none in a day, usually two or three and sometimes six or even more.

The physical and neuroligical examinations were negative throughout. The blood Kahn, feces examination, urinalysis, and spinal fluid examination were entirely negative. An X-ray of the skull was normal. The basal metabolism rate was plus 2 percent.

The treatment consisted of ephedrine sulphate, grs. ⅜, at 6 a. m., 10 a. m., and 2 p. m., daily, for 12 days. During this period he reported a decrease in the number and severity of both narcoleptic and cataleptic attacks. No attack was actually observed by a medical officer though several were witnessed by other patients and the ward corpsmen. On April 24, 1933, he was transferred to the U. S. S. *Henderson* for further transfer to a naval hospital in the United States.

REFERENCES

(1) Addie, W. J.: Idiopathic Narcolepsy, A Disease Sui Generis; With Remarks on the Mechanism of Sleep, Brain 49: 257 (Sept.) 1926.

(2) Westphal, C.: Zwei Krankheitsfalle, Arch. f. Psyhiat. u. Nervenkrankh. 7: 631, 1877. Fischer, F.: Epileptoide Schlafzustände, Ibid. 8: 203, 1878. Gélineau: De la narcolepsie, Gaz. d. hôp. 53: 626, 1880.

(3) Foot, A. W.: Narcolepsy, Brit. M. J. 2: 1166 (Dec. 11) 1886.

(4) Loewenfeld, L.: Ueber Narcolepsie, Munchen med. Wchnschr. 49: 1041 (June 24) 1902.

(5) Henneberg, R.: Ueber genuine Narcolepsie, Neurol. Zentralbl. 35: 282, 1916.

(6) Morton, W. J.: A Case of Morbid Somnolence, J. Nerv. & Ment. Dis. 11: 615, 1884.

(7) Ewen, C.: A Case of Narcolepsy, Boston Med. & Surg. J. 128: 569, 1893.

(8) Wilson, S. A. K.: The Narcolepsies, Brain 51: 63, 1928.

(9) Worster-Drought, C.: Narcolepsy, Brit. J. M. Psychol. 3: 267, 1923.

(10) Weech, A. A.: Narcolepsy; A Symptom Complex, Am. J. Dis. Child. 32: 672 (Nov.) 1926.

(11) Cave, H. A.: Narcolepsy, Arch. Neurol. & Psychiat. 26: 50–101 (July) 1931.

(12) Gowers, W. R.: The Borderland of Epilepsy, J. & A. Churchill, London, 1907.

(13) Purves-Stewart, J., Cohen, H., Stewart, G., et al: Part of Discussion on the Narcolepsies, Brain 51: 126, 1928. Jelliffe, S. E.: Narcolepsy, Hypnolepsy, Pyknolepsy, M. J. & Rec. 129: 269–313, 1929. Jelliffe, S. E., and White, W. A.: Diseases of the Nervous System, Lea & Febiger, Philadelphia, 5th Ed., 1929.

(14) Levin, M.: Narcolepsy (Gélineau's syndrome) and other varieties of morbid somnolence, Arch, Neurol. & Psychiat. 22: 1172–1200 (Dec.) 1929.

(15) Doyle, J. B., and Daniels, L. E.: Narcolepsy: Results of Treatment with Ephedrine Sulphate, J. A. M. A. 98: 542 (Feb. 13) 1932.

(16) Harris, S.: Narcolepsy associated with hyperinsulinism, J. A. M. A. 100: 321–328 (Feb. 4) 1933.

(17) Spiller, W. C.: Narcolepsy, Occasionally a Post-Encephalitic Syndrome, J. A. M. A. 86: 673 (March 6) 1926.

(18) Daniels, L. E.: Symptomatic Treatment for Narcolepsy, Proc. Staff Meet. Mayo Clin. 5: 299–300 (Oct. 22) 1930.

(19) Prinzmetal, M., and Bloomberg. W.: The Use of Benzedrine for the Treatment of Narcolepsy, J. A. M. A. 105: 2051 (Dec. 21) 1935.

OBSERVATIONS ON CHROMATOID BODIES IN THE CYSTS OF ENTAMOEBA HISTOLYTICA[1]

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About a year ago, one of us (J. F. B.) noted that when cysts of *E. histolytica* were kept in water there would be seen a larger proportion with chromatoid bodies than when the cysts were allowed to remain in the fecal mass. While attempting to find the cause of this peculiarity it was discovered that chromatoid matter apparently occurs in two forms: As microscopically demonstrable chromatoid

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bodies, and as a microscopically invisible substance which has been termed latent chromatoid matter.

It is well known that *E. histolytica* cysts in evacuated fecal material gradually lose their chromatoid bodies. As early as 1912, Hartmann (1) noted that they were consumed during the development of the cyst and its subsequent life. Dobell (2) observed that the mature living cysts lost their chromatoid bodies in the course of a few days, and assumed that they were being absorbed. It will be shown that the loss of these primary chromatoid bodies is not entirely a process of consumption or absorption, but mostly a change from a manifest to a latent form of chromatoid matter from which a formation of secondary chromatoid bodies can be produced by transferring the cysts from the feces into water, saline solution, or, probably, any other aqueous solution which would not be destructive to the cysts.

MATERIAL AND METHODS OF EXAMINATION

In order to convey the true significance of our results the essential data on the material used and the methods of examination employed will be given. Twenty cyst-containing fecal specimens from 10 different cases of *E. histolytica* infection were studied. These cases and the specimens were selected. To guard against mistakes in identification of cysts in wet unstained smears no case who had concomitant infections with *E. coli*, *I. butschlii*, or *G. lamblia* was used. Large races of *E. histolytica* with cysts measuring from 12 to 14 microns were selected in order to facilitate the observations on the nuclear development, the number, size, and shape of the chromated bodies, and the changes of degeneration, all of which are not so easily seen in the smaller races. In order to make it possible to find the necessary cysts without prolonged search only stools showing 25 or more cysts under a 7/8-inch coverglass were used. The fecal specimens were stored in cardboard ice cream containers with moistened blotting paper inserted in the lids to prevent drying. The various liquid mixtures were made in the proportion of about 10 gms of feces to 100 cc of the liquid.

The stools were first examined within 5 minutes to 1 hour after evacuation and then at intervals as required by the experiment until most of the cysts or all had degenerated or disappeared. In 8 series the cysts were examined only in wet unstained smears while in the other 12 series wet-fixed stained smears (iron-hematoxylin method) were also used. One hundred cysts or more were observed at each examination except toward the end of the experiments, when sometimes only a few cysts remained. When both wet and stained smears were used an equal number of cysts, usually 50, were studied in each preparation. In all, 34,280 cysts were observed in

wet unstained smears and the presence or absence of chromatoid bodies, their number per cyst, and their characteristics noted. Records were also kept of the morphological changes of degeneration in the cysts as they grew old under the various conditions of the experiments. In the stained smears, 15,770 cysts were studied and a corresponding record made, as well as notes on nuclear development and glycogen vacuoles.

The relative value of wet and stained smears for a study of this nature may be mentioned in this connection. The presence of chromatoid bodies and their size and shape can be best studied in stained smears. In wet smears the chromatoid bodies in not a few cysts with glycogen or large vacuoles will not be seen even though the cysts may be examined most carefully, and chromatoid bodies in the shape of small granules, spicules, or filaments may escape detection even in cysts with the clear cytoplasm usually present after absorption of the glycogen. Our records show that stained smears of young cysts with abundance of glycogen will reveal up to 20 percent more cysts with chromatoid bodies than wet smears; under all conditions they reveal more chromatoid bodies per cyst. In old specimens with many cysts in advanced degeneration and disintegration the wet smears are more accurate for the determination of percentage of cysts with chromatoid bodies, since in the stained smears the degenerate cysts stain poorly or not at all and readily escape notice.

MANIFEST AND LATENT CHROMATOID MATTER

In order to illustrate the transition of chromatoid bodies into latent chromatoid matter and the development of secondary chromatoid bodies, graphs have been made of three representative series of observations on these changes. *Series 12* (fig. 1). This series is presented to show the changes in chromatoid matter from one of the earliest stages encountered in cysts from fecal material. The cysts were very young and mostly immature, 82 percent being uninucleate and binucleate. Practically all contained chromatoid bodies; in the first 3 smears, which were made 1, 2, and 3 hours after the evacuation, only 2 of the 300 cysts observed were without chromatoid bodies. As shown in the lower heavy line (F) there is then a rapid decrease until at 48 hours all these primary chromatoid bodies have changed into latent chromatoid matter. This is the loss of chromatoid bodies observed by previous workers. The upper heavy line (W–1) represents the percentages of cysts with chromatoid bodies in cysts which were transferred to water one hour after evacuation, at the time of the first examination. In this mixture of water and feces there is no such transition of primary chromatoid bodies into latent chro-

matoid matter but the chromatoid matter remains manifest, and as such gradually decreases and finally disappears as the cysts begin to degenerate. The other lines show the return of latent chroma-

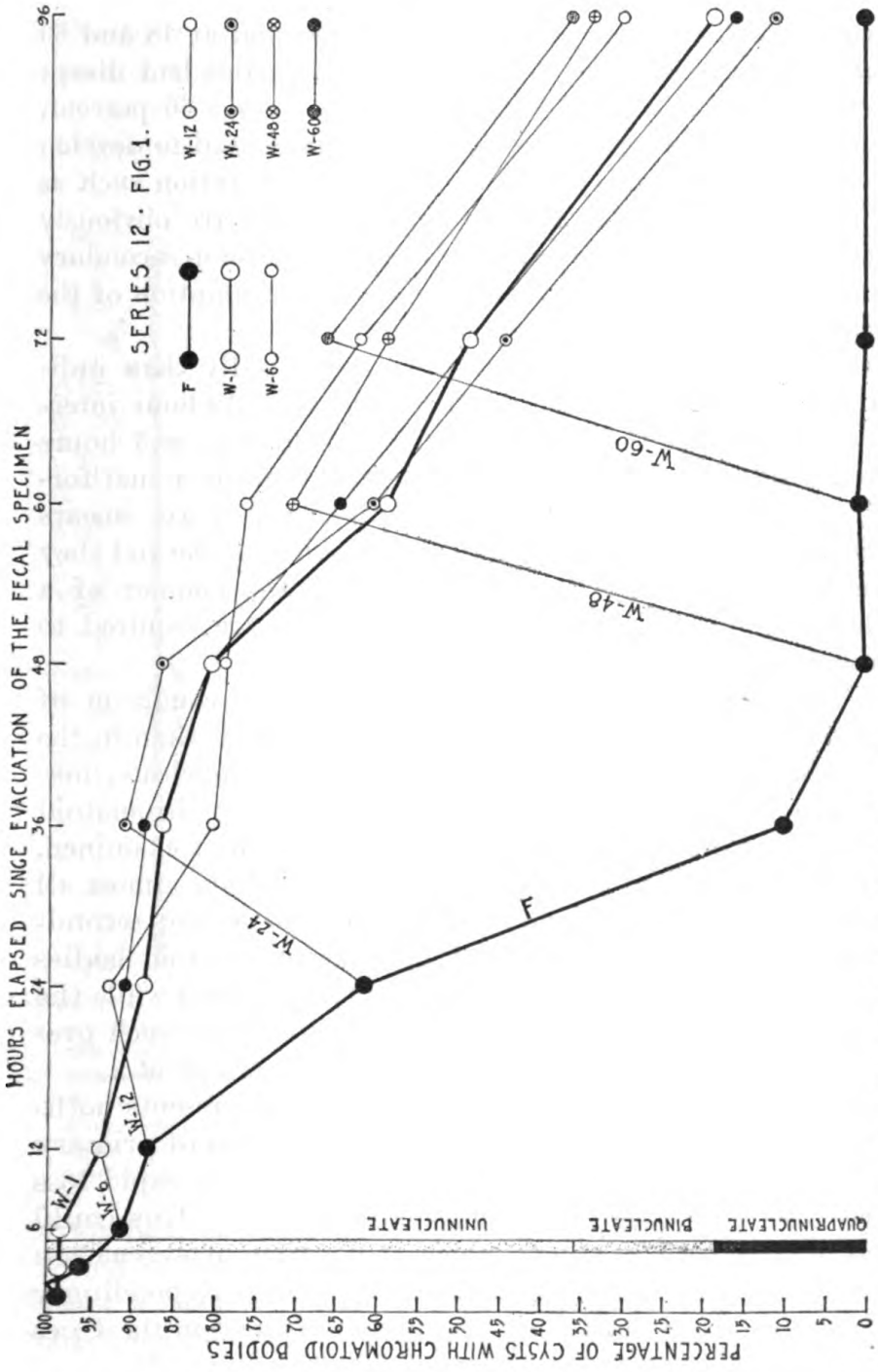

FIGURE 1.—A graph to illustrate changes in chromatoid matter in young cysts in which there has been practically no transition of primary chromatoid bodies into latent chromatoid matter before evacuation. Line F., transition of primary chromatoid bodies into latent chromatoid matter in cysts remaining in the fecal material. Lines W–1, W–6, W–12, W–24, W–48, and W–60, cysts transferred from the feces to water at 1, 6, 12, 24, 48, and 60 hours, respectively, after evacuation of the feces; the increase in percentages of cysts with chromatoid bodies represents the transition of latent chromatoid matter into secondary chromatoid bodies. All specimens stored at room temperature.

toid matter to a manifest form, the secondary chromatoid bodies, in cysts transferred to water at 6, 12, 24, 48, and 60 hours after evacuation. Note that secondary chromatoid bodies do not form in all

cysts which have lost their primary chromatoid bodies; for example, at 6 hours when primary chromatoid bodies have disappeared in 8 percent of the cysts, secondary ones form in only 2 percent, at 24 hours secondary chromatoid bodies form in 30 percent although 36 percent had lost their primary chromatoid bodies, and at 48 and 60 hours when in all cysts the primary chromatoid bodies had disappeared, secondary chromatoid bodies develop in 70 and 66 percent, respectively. Most of the cysts which in this manner fail to develop secondary chromatoid bodies show changes of degeneration such as granulation of the cytoplasm and loss of lustre and are obviously dead; others appear to be alive and their failure to develop secondary chromatoid bodies may indicate that a metabolic consumption of the latent chromatoid matter had taken place.

The secondary chromatoid bodies form more rapidly than indicated by the graph which records the percentages at 12-hour intervals. Actually most of them form during the first 2 or 3 hours (at room temperature). During this rapid transition the actual formation of secondary chromatoid bodies may be seen in wet smears by the persistent observer. Beginning as a fine thread-like rod they increase in length and thickness very much in the manner of a wax candle being dipped. From 20 to 30 minutes are required to complete the formation (fig. 4).

Series 15 (fig. 2). In this series a more common condition of the chromatoid matter will be seen. The cysts are older than in the previous series, 97 percent having reached the quadrinucleate, mature stage. In 14 percent of these cysts the primary chromatoid bodies already had disappeared when the cysts were first examined, 10 minutes after evacuation (line F). As shown in W–⅙ almost all of these cysts had enough latent chromatoid matter to develop secondary chromatoid bodies. Transition of primary chromatoid bodies into latent chromatoid matter apparently then had occurred while the cysts were retained in the colon. In most fecal specimens such pre-evacuation transition takes place to a greater or lesser extent.

The graph of this series has the usual pattern and presents nothing essentially new. Note, however, the quick transition of primary chromatoid bodies into latent chromatoid matter and the rapid loss of the latter and of the secondary chromatoid bodies. This could be expected to occur in these cysts which at the time of evacuation were relatively old. Degenerative changes appeared correspondingly early, for example, at 48 hours very few cysts remained in the feces and of these 70 percent were in a state of disintegration. It is not assumed, however, that the age of the cysts was the only factor in the early degeneration of the cysts in this stool. Equally mature cysts, and probably as old, have been seen to last much longer in other specimens of feces stored under the same conditions. Biological

variation in various strains, differences in the putrefactive process of the feces, and probably other conditions are undoubtedly factors influencing degeneration in the cysts and incidentally the changes in chromatoid matter.

Series 14 (fig. 3). This series is presented to show the exteme to which the chromatoid matter may disappear before evacuation of the cysts. The cysts were obtained from a formed dry stool which obviously had been retained a long time in the colon. When first

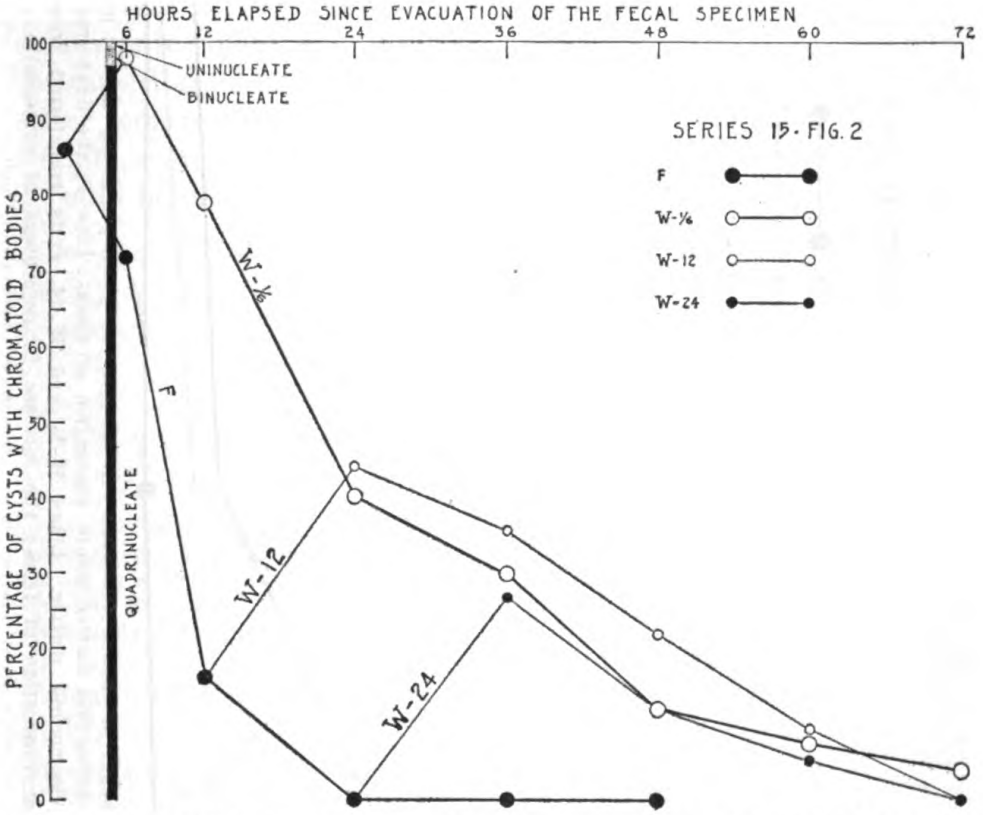

FIGURE 2.—A graph to illustrate the changes in chromatoid matter in older cysts in which some transition of primary chromatoid bodies into latent chromatoid matter has taken place before evacuation of the feces. Line F, transition of primary chromatoid bodies into latent chromatoid matter in cysts remaining in the feces. Lines W–½, W–12, and W–24, cysts transferred from feces to water at 10 minutes, 12 and 24 hours respectively after evacuation of the feces; the increase in percentage of cysts with chromatoid bodies represents the transition of latent chromatoid matter into secondary chromatoid bodies. All specimens stored at room temperature.

examined, one-half hour after deposition, chromatoid bodies were found in only two cysts (2 percent), in one a short rod, and in the other a slender rod and a thin filament. Transferred to water at this time (W–½), only 28 percent of the cysts developed secondary chromatoid bodies; 24 hours later, 23 percent still had enough latent chromatoid matter to develop secondary chromatoid bodies (W–24). When the pattern of this graph is compared with series 15 it appears reasonable to assume that the chromatoid matter in the cysts of series

14 at the time of evacuation was in the same stage as the chromatoid matter in the cysts of series 15, 24 hours after deposition, at which time the primary chromatoid bodies had disappeared and latent

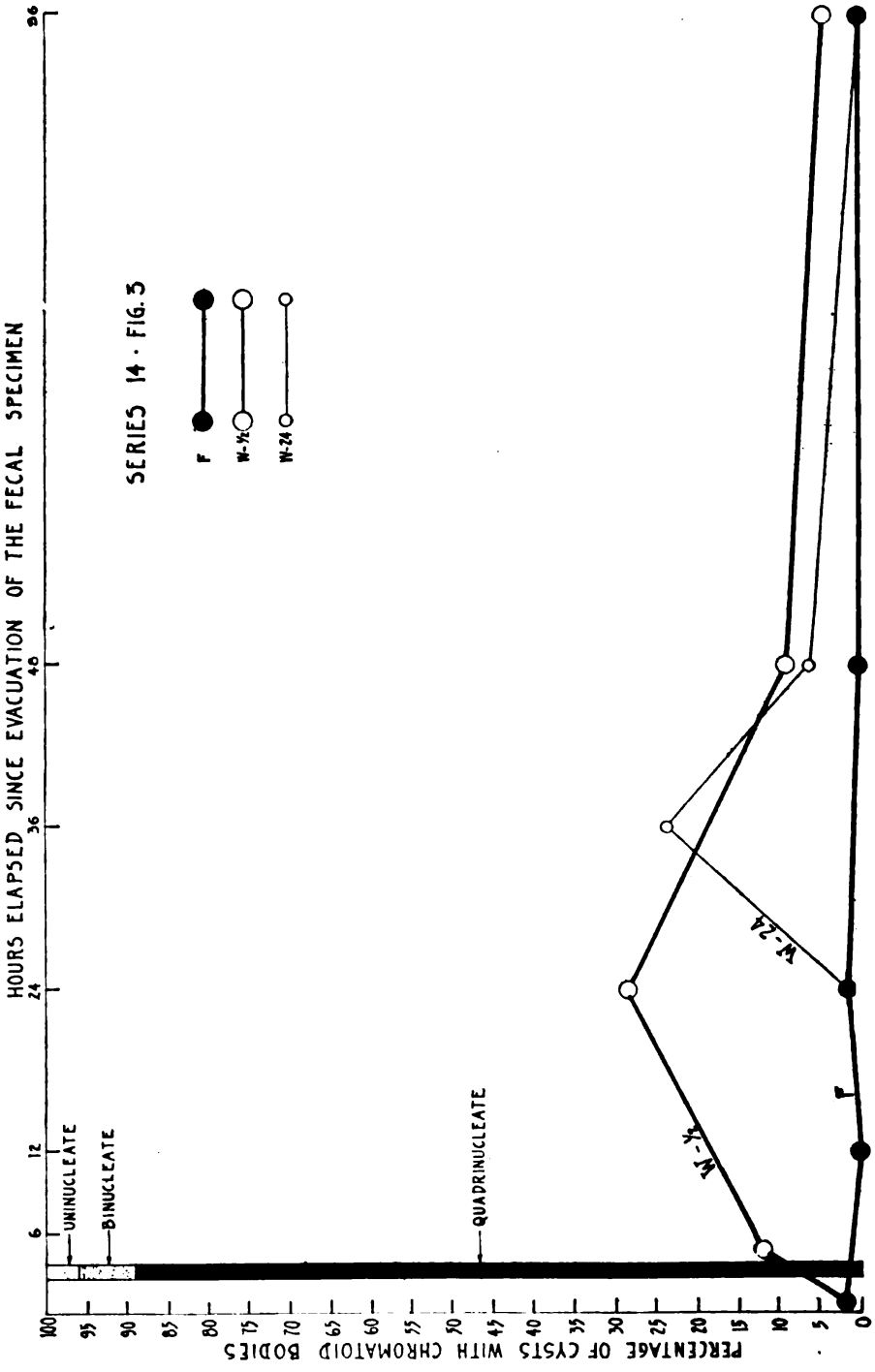

FIGURE 3. A graph to illustrate changes in chromatoid matter in old cysts in which practically all the primary chromatoid bodies have changed into latent chromatoid matter before evacuation of feces. Line F, the final transition of primary chromatoid bodies into latent chromatoid matter. Lines W-½ and W-24, cysts transferred from feces to water one-half hour and 24 hours after evacuation of feces; the increase in percentage of cysts with chromatoid bodies represents the transition of latent chromatoid matter into secondary chromatoid bodies. All specimens stored at room temperature.

chromatoid matter was present in 27 percent of the cysts. The character of the stools from which the cysts of the two series were obtained supports this assumption; the stool used in series 15 was formed but

soft, while the stool for series 14 was formed and dry, and obviously a stool of constipation which well may have been retained 24 hours longer in the colon.

CHANGES IN CHROMATOID MATTER UNDER VARIOUS CONDITIONS

Temperature.—In the three series of observation discussed above all specimens were kept at room temperature, 28° C. to 32° C. The changes in chromatoid matter also have been studied in cysts stored in incubator at 37° C., and in refrigerator at 10° C. Except for variations in time the changes were essentially the same. At 37° C. the transition of primary chromatoid bodies into latent chromatoid matter required from 10 to 12 hours, at 28° C. to 32° C. from 24 to 48 hours, and at 10° C. from 11 to 12 days. Latent chromatoid matter could be demonstrated (by transfer of cysts to water and noting development of secondary chromatoid bodies) for 24 to 48 hours in cysts kept at 37° C., for 60 to 72 hours in cysts at 28° to 32° C., and for 17 to 18 days at 10° C. Secondary chromatoid bodies lasted a short time beyond these periods due, apparently, to the longer life of the cysts in water.

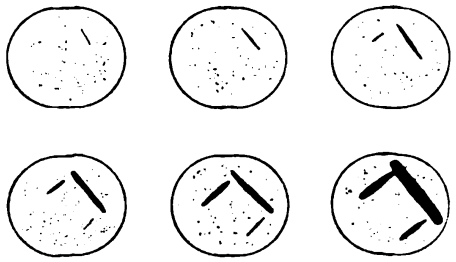

FIGURE 4.—The formation of secondary chromatoid bodies in an *E. histolytica* cyst observed in a wet smear with 4 mm. objective and 6X ocular .X1000. The sketches were made at intervals of 5 minutes.

The transition of latent chromatoid matter into secondary chromatoid bodies is strikingly accelerated at 37° C. and much delayed by refrigeration. The change is also more complete at higher temperatures, as indicated by the development of more and larger secondary chromatoid bodies.

Reaction.—During the early stages of this work the findings seemed to indicate that acidity and alkalinity influenced the changes in chromatoid matter. Comparative observations were then made on cysts in neutral, acid, and alkaline mixtures of feces and water. Hydrochloric acid, 1 percent, and sodium hydroxide, 1 percent, were used for adjusting the reaction which was roughly determined by litmus poper. It was found that slight deviations from the neutral point had no apparent influence on the changes in chromatoid matter. In strongly acid mixtures latent chromatoid matter developed into more abundant and often larger secondary chromatoid bodies than in neutral mixtures, while in corresponding strongly alkaline mixture the cytoplasm of the cysts became opalescent and lifeless, the nuclear chromatin coalesced, and secondary chromatoid

bodies failed to develop, all apparently due to injury to the cyst wall allowing alkalinization of the cytoplasm and rapid degeneration.

SUMMARY OF THE CHANGES IN CHROMATOID MATTER IN *E. HISTOLYTICA* CYSTS

The results recorded above and some incidental observations make it possible to trace chromatoid matter from the time of its first appearance in the newly formed cysts until its disappearance in the disintegrating cysts. As already has been shown, changes in chromatoid matter take place before as well as after evacuation of the cysts, and since most of the stools containing cysts are formed, having been retained in the colon for a considerable time, these changes are usually well advanced even when the cysts are examined immediately after deposition. In order to see all events in one batch of cysts it is necessary to select a stool which has been evacuated with a minimum delay in the bowel. Since such stools will be soft or mushy they usually contain only, or mostly, trophozoites, but occasionally patients with subacute amoebic colitis will pass a shower of cysts in a mushy stool. These young cysts, particularly the uninucleates and binucleates, if examined immediately, will present chromatoid matter in the earliest phases observable in human infections.

The young cysts have an abundance of chromatoid matter, manifest in the most variable form and greatest numbers per cyst seen in any stage. Most cysts have four or more, and not a few have their cytoplasm crowded with chromatoid bodies. More than 30 have been counted in some. With four or less in a cyst they usually have the characteristic rod shape, but when more numerous they frequently are in the form of minute filaments, spicules, wedges, or granules. In cysts with large glycogen vacuoles, which will be found in the majority of these young cysts, the chromatoid bodies are usually small and numerous and crowded against the periphery; with less glycogen they frequently are packed into thick nondescript masses.

When the cysts are allowed to remain in the fecal mass these primary chromatoid bodies gradually disappear as they change into latent chromatoid matter. The time required for this process varies with the temperature at which the stool is kept, as noted above. During this period of transition into latent chromatoid matter the chromatoid bodies invariably become fewer per cyst, the larger rod-shaped ones being the last to disappear. At any time during and subsequent to the disappearance of the primary chromatoid bodies and until the cysts begin to show morphological changes of degeneration, the latent chromatoid matter returns to a manifest form (secondary chromatoid

bodies) when the cysts are transferred to water. This transition also takes place in saline solution, Ringer's solution, in liquids used for culturing *E. histolytica*, such as Locke albumen for the Boeck-Drobhlav medium, and the horse serum and saline for covering agar slants, and probably in any aqueous solution which is not injurious to the cysts.[2] The formation of secondary chromatoid bodies is accelerated when the cysts are kept at 37° C. and delayed at lower temperatures. When observed in wet smears at room temperature the transition of latent chromatoid matter into secondary chromatoid bodies, once started, has been seen to require only from 20 to 30 minutes. The secondary chromatoid bodies vary in number, size, and shape according to the amount of latent chromatoid matter present in the cysts at the time of their formation. With an abundance of the latter they become long thick rods or bars, brilliantly refractile in wet smears, while with a scantier supply they form as thin rods or filaments, or broad rods or bars of a very light density which in wet smears lack the bluish luster and appear glasslike. In general the secondary chromatoid bodies show less variation in shape and frequently are diagramatically typical of the characteristic chromatoid body of *E. histolytica*. Broad, thick bars extending along the whole diameter of the cyst are particularly distinctive.

As cysts grow old there is a steady loss of chromatoid matter whether latent as in cysts kept in feces or manifest as in the cysts transferred to liquids. When latent this loss becomes evident in the decreasing number and lessened density of the secondary chromatoid bodies developing upon transfer of the cysts to water; when manifest they can be seen to decrease in size and in number per cyst. The final disappearance of chromatoid matter is incident to morphological changes of degeneration observable in the unstained cyst (granulation of the cytoplasm with loss of lustre and coalescence of the peripheral nuclear chromatin) or it may precede these obvious changes of disintegration by a few hours.

COMMENT ON SOME OF THE RESULTS RECORDED BY PREVIOUS OBSERVERS

Origin of chromatoid bodies.—Hartmann (1) held that the chromatoid bodies have their origin in the nucleus. James (4), after a prolonged study of their staining reactions and their morphological characteristics, came to the conclusion "that they are derived from the cytoplasm by a process of condensation in the latter, and have nothing whatever to do with nuclear chromatin." Dobell (2) states that he had not found that staining reactions proved their cytoplasmic

[2] Yorke and Adams (9) may possibly have observed the transition of latent chromatoid matter into secondary chromatoid bodies when they noted an "initial increase" in percentage of cysts containing chromatoid bodies (from 27 to 56 percent in 3¼ hours) during cultivation on Locke-egg-serum medium.

origin. Whether they are formed from the chromatin of the nucleus or are secreted in the cytoplasm, he considered uncertain, and all that he could say with certainty was that they appeared in the cytoplasm.

Having observed their actual formation we can state with certainty that in their earliest visible form they appear in the cytoplasm without any microscopical evidence of any relationship to nuclear chromatin and that the process of formation aptly can be described as "a process of condensation" of some substance in the cytoplasm.

Function of chromatoid bodies.—The function of chromatoid bodies never has been definitely determined. To Hartmann (1) it appeared that they acted as *Reservestoff* since they would be consumed during the development of the cysts and the following resting stage. Dobell (2) also considers it probable that, like the glycogen, they represent reserve material of some sort. He holds the same opinion in 1927 (3), then stating that "it seems reasonable to suppose that glycogen and chromatoids are reserve substances (carbohydrate and protein, respectively) which the amoebae store in their cysts to enable them to live outside the body, when they are no longer free and able to capture food." Not a few writers subsequently have expressed the same opinion.

Although the disappearance of the primary chromatoid bodies, which phenomenon has been the basis for the food reserve theory, apparently does not represent consumption but merely a transition to another form (latent chromatoid matter), there is, nevertheless, a gradual decrease in the total amount of chromatoid matter, whether latent or manifest, a decrease which may be due to consumption in metabolic processes of the living cysts rather than to destruction incident to degenerative changes. A change in chromatoid matter which does not make it appear as stored food is the transition of latent chromatoid matter into the solid form of secondary chromatoid bodies when the cysts are incubated (liquid culture media, or solid media with a superimposed liquid). One would rather expect that the chromatoid matter would remain in the latent form and thus be more readily available for consumption in the enhanced metabolism of incubation.

Frequency of occurrence of chromatoid bodies.—As indicated by the results presented above, the frequency of occurrence of chromatoid bodies in cysts from feces, even when examined immediately after evacuation, may vary from 100 to 0 percent, the variation depending upon the time elapsed since the cysts were formed. In stools which have been evacuated with a minimum delay the cysts may have formed so recently that all may still contain some of their primary

chromatoid bodies, while in hard dry stools, which may have been retained in the colon 12 to 24 hours or even more and thus having permitted the primary chromatoid bodies to change into latent chromatoid matter, all cysts may be without chromatoid bodies. Usually stools are passed before the majority of the cysts have lost their primary chromatoid bodies.

Smith (5) found chromatoid bodies present in 27 percent, absent in 65 percent, and doubtful in 8 percent of 1,162 cysts examined in iodine solution. He considers it possible that very small chromatoid bodies might have been overlooked and that only counts from stained preparations would give absolutely accurate results. It has been our experience that even chromatoid bodies of average size readily may escape detection not only in iodine solution but even in saline, "in which medium", as Smith states, "the chromatoid bodies are more distinctly seen than in iodine." Furthermore, Smith gives no information as to the consistency of the stools, nor as to the time after evacuation that they were examined. Without these data his figures have no significance. Dobell (2) describes the formation of chromatoid bodies as part of the normal development of the cysts of *E. histolytica*, but also notes that "sometimes they are completely absent in cysts at all stages of development." The last statement is not understood to mean that cysts form and develop without at any time having chromatoid bodies, but that sometimes chromatoid bodies are completely absent in cysts which at the time of examination were in all stages of nuclear development. This condition is seen occasionally in cysts immediately after evacuation (see series 14) and frequently 24 hours or more later. Uninucleate and binucleate cysts without chromatoid bodies, although immature in their nuclear development, are thus relatively old cysts with delayed nuclear development in which the primary chromatoid bodies already have changed into latent chromatoid matter.[3] This conclusion is based on the observation that the various changes in chromatoid matter take place irrespective of nuclear development. An example of this can be found in series 12; No. II.

When the cysts were first examined, 1 hour after evacuation, 64 percent were uninucleate, 18 percent binucleate, and 18 percent quadrinucleate and all had chromatoid bodies; 48 hours later chromatoid bodies were completely absent in all cysts although 64 percent still were uninucleate and binucleate, and when transferred to water secondary chromatoid bodies appeared in cysts at all stages of nu-

[3] It is, of course, possible that immature cysts may be passed before chromatoid bodies have developed. That this may occur is suggested by an observation made by Cleveland and Sanders (6) who noted that in cysts produced in cultures chromatoids sometimes appeared much later than they did at other times. They have drawings of uninucleate and binucleate cysts in which chromatoids have not appeared.

clear development. Kofoid et al. (7) state that chromatoid bodies are found in a majority of the cysts, and Craig (8) finds that they occur in at least 50 percent of all cysts of *E. histolytica*. Rightly, Craig also has noted that "these bodies are most numerous in the uninucleate and binucleate cysts." A very different observation is recorded by Yorke and Adams (9): "Careful examination of freshly passed faeces", they state, "showed that these bodies were comparatively rarely present in the uninucleate cysts, but were commonly found in the bi- and quadri-nucleates", and again in discussing the variations in cysts passed at different times: " * * * on one occasion the cysts may be practically all uninucleate with much glycogen, and but few of them containing chromatoid bodies, whereas on another occasion the vast majority may be quadrinucleate with chromatoid bodies but little glycogen." These are indeed the findings when cysts are examined in iodine, which method apparently these workers used. Even the most careful examination of cysts stained with iodine will not reveal the small chromatoid bodies frequently present in uninucleate cysts with glycogen vacuoles. Often they are not seen even in unstained cysts in wet smears. Actually uninucleate cysts have as much chromatoid matter as binucleates and quadrinucleates of about the same age.

PRACTICAL APPLICATION

The change of latent chromatoid matter into secondary chromatoid bodies has a practical application in the laboratory diagnosis of amoebiasis. Not infrequently one encounters specimens of feces with cysts which should be suspected of being a small race *E. histolytica* but which cannot be identified in wet smears. For example, there may be numerous cysts of *E. nana* or of *C. mesnili*, and among these a few rounded cysts, perhaps a little larger and perhaps slightly different in their cytoplasmic structure, but not labeled with a chromatoid body. Smears in iodine solutions may not be of much help since the nuclei in the cysts of a small race of *E. histolytica* have such fine peripheral chromatin rings that their appearance in iodine often does not differentiate them from *E. nana*. Similarly a few small cysts of *E. histolytica* in a shower of cysts of *I. bütschlii* may very readily escape detection unless they happen to have a characteristic chromatoid body. In wet-fixed stained preparations the differentiation can be made, of course, but when the cysts are very few, an unduly long search may be required. Not infrequently the simpler procedure will be to macerate a portion of the feces, about the size of a bean in a test tube of water and after 4 hours or later examine the sediment, or a concentrate thereof, and look for cysts with chromatoid bodies. Should concentrations be used, a method which does

not require any chemical which is destructive to chromatoid matter must be selected. In this connection it may be mentioned that the sugar solution used in the concentration method by Yorke and Adams (9) does not affect the chromatoid bodies in any visible degree.

SUMMARY

Chromatoid matter in *E. histolytica* cysts apparently occurs in two forms, manifest and latent. This assumption is based on the observation that in cysts which have lost their chromatoid bodies while in the fecal material, a new set of chromatoid bodies may develop when the cysts are transferred from the feces into water. The hypothetical substance into which the primary chromatoid bodies are converted and out of which the secondary chromatoid bodies are formed has been termed latent chromatoid matter.

The transition of primary chromatoid bodies into latent chromatoid matter takes place in the cysts as they grow old in the fecal material, before as well as after its evacuation and apparently without any relation to nuclear development.

The formation of secondary chromatoid bodies has been observed and appears to be a process of condensation of some substance in the cytoplasm. They have essentially the same appearance as primary chromatoid bodies but tend to be more characteristically rod shaped and bar shaped.

Chromatoid matter, whether manifest or latent, decreases as the cysts grow old and disappears when they begin to show morphological changes of degeneration, or shortly before this stage.

The knowledge of these changes makes it possible to correlate or reasonably explain some of the data on the occurance of chromatoid bodies recorded by previous workers.

The generally accepted theory that chromatoid bodies constitute a food reserve appears rather doubtful in view of some observations on these changes. Further studies on the transition of manifest chromatoid matter into latent and its return to the manifest form may reveal its significance in the metabolism of the cysts.

The change of latent chromatoid matter into secondary chromatoid bodies has a practical application of considerable importance in the laboratory diagnosis of amoebiasis.

REFERENCES CITED

(1) Hartmann, M.: Untersuchungen über parasitische amöben. 1. *Entamoeba histolytica* Schaudinn. Arch. f. Protisenk., 24: 163, 1912.

(2) Dobell, C.: The amoebae living in Man. William Wood & Company, 1919.

(3) Dobell, C.: Further observations and experiments on the cultivation of *Entamoeba histolytica* from cysts. Parasit., 19: 288, 1927.

(4) James, W. M.: A study of the Entamoebae of Man in the Panama Canal Zone. Ann. Trop. Med. & Parasit., 8:133, 1914.

(5) Smith, A. M.: Measurements of and observations upon the cysts of *Entamoeba histolytica* and *Entamoeba coli*. Ann. Trop. Med. & Parasit., 12:27, 1927.

(6) Cleveland, L. R. and Sanders, Elizabeth P.: Encystation, multiple fission without encystment, excystation, metacystic, development and variation in a pure line and nine strains of *Entamoeba histolytica*. Arch. f. Protist., 70:222–266, 1930.

(7) Kofoid, C. A., Korhnauser, S. I., & Swezy, Olive: Criterion for distinguishing the Endamoeba of amoebiasis from other organisms. Arch. Int. Med. 24:35–50, 1919.

(8) Craig, C. F.: A manual of the parasitic protozoa of Man. J. B. Lippincott Company, Philadelphia, 1926.

(9) Yorke, W. & Adams, A. R. D.: Observations on *Entamoeba histolytica*. 1. Development of cysts, excystation, and development of excysted amoebae, in vitro. Ann. Trop. Med. & Parasit. 20:279, 1926.

TERATOMAS OF THE TESTICLE—GENERAL CONSIDERATIONS AND CASE REPORT[1]

By MELVILLE J. ASTON, Commander, Medical Corps, United States Navy

Testicular neoplasms are comparatively rare (1), the incidence being less than 1 percent of all tumors. The precosity of these tumors is well recognized, yet they occur more frequently between the ages of 20 and 50 years, during the period of greater sexual activity.

Testicular tumors are practically always unilateral. Pearson (2), from a review of the literature, reporting only 47 cases of bilateral involvement, including one case of his own. The two testes are involved with equal frequency.

It is rather generally believed that undescended testes are especially predisposed to malignant degeneration, although (3) MacKenzie and Ratner hold that there are no substantial facts in proof of this belief. Hinman (4), however, studying 649 cases of malignancy of the testes, found 12.2 percent in undescended testicles. It is estimated that normally placed testicles occur from about 600 to 1,000 times to every cryptorchid.

The role of trauma or of preexisting testicular disease in the production of testicular neoplasms is open to question.

Pathology.—Concerning the definite pathological nature of neoplasms of the testicle, a marked difference of opinion exists. Theoretically, some teratomas should be more benign. Yet from a practical standpoint it is considered advisable to regard all testicular neoplasms as malignant unless otherwise proven.

[1] Read before the Norfolk County Medical Society on Oct. 7, 1935.

According to Hinman (4), the common tumors of the testicle are (*a*) the teratomata in which mixed tumors all three germ layers (epiblast, hypoblast, and mesoblast) are preserved in varying proportions, and (b) seminomata which are tumors composed of one specific type of cell in accordance with the theory of Chevassu whose name is commonly linked with this type of tumor.[1]

Ribbert and Ewing hold that these seminomata are in reality teratomata which exhibit a one-sided development.

Most pathologists regard the seminoma as a type of teratoma testis.

Hinman (4) believes that it is of great importance clinically to differentiate, if possible, between the mixed type or teratoma and the seminoma, or tumor of mono cellular type, as the teratoma is generally regarded as radio-resistant, while the semionoma is believed to be radiosensitive.

A very rare neoplasm of the testicle is the chorionepitheloma, identical in type to chorionepitheloma of the uterus. Sarcoma also is a very rare tumor and many of the cases previously reported as such are now considered to have been seminomas. Sarcomas may criginate in the connective tissue of the testicle or in the tunics. Only a few purely benign tumors are on record. These are usually fibromas or adenomas.

Metastases occur usually early by way of the primary lymphatics to the retro-peritoneal lymph glands.

The lungs, liver, spleen, long bones, and kidneys may be involved by early general metastasis through venous extension.

Symptoms.—Early testicular tumors exhibit local enlargement and are usually painless. Later in the course of the disease, pain is experienced in the scrotum, inguinal regions, abdomen, back, and legs.

The rapidity of tumor growth varies. Weight loss is a prominent symptom. A feeling of general weakness is common. Loss of sexual power, vomiting, and constipation sometimes occur. Other symptoms are those which are common to malignant neoplasms in general.

Diagnosis.—Palpation of the testicle reveals a smooth, hard, symmetrically enlarged organ, varying considerably in size.

The scrotal skin may be slightly reddened.

Late in the disease when the tumor is quite large, nodulations and fluctuant areas of cystic degeneration are occasionally present. The scrotal skin in the large tumors is smooth and stretched, and large veins may be seen beneath it.

The epididymis is usually normal, but in large tumors it is often distorted in shape.

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[1] Read before the Norfolk County Medical Society on Oct. 7, 1935.

Usually the vas deferens is normal, as is also the spermatic cord. However, in some cases the cord shows definite enlargement and the induration indicative of malignant infiltration. Hydrocele may accompany the large tumors.

The inguinal glands are involved only when direct extension of the tumor to the scrotal tissue or skin take place. These instances are rare.

An important aid in diagnosis of teratoma testis has been acquired by the development of Aschheim-Zondek pregnancy test. This is a biologic test for the anterior pituitary sex hormone known as prolan A.

The urine of patients suffering from teratoma testis contains prolan A, while no report of the presence of this hormone in the urine of a normal healthy man has been made.

Ferguson (5) states that the amounts of prolan A in terms of mouse units per liter of urine vary in different types of teratoma testis, and further states that "the quantitative excretion of prolan A is a definite indication of the structural type of the tumor." The amounts of hormone are further influenced by the presence or absence of metastases, and by the reaction to irradiation in accordance with the degree of radio-sensitivity of the malignant growths.

A clinical cure is obtained when, following surgical and irradiation therapy, there is no clinical evidence of the disease, and repeated examinations of the urine show an output of less than 400 mouse units of prolan A per liter of urine. Ferguson (5) considers that the excretion of these smaller amounts of prolan A may be due to a physiological imbalance between the secretion of the remaining testicle (which may have been damaged by irradiation), and the pituitary, with the resultant excretion of the hormone over long periods of time.

Differential diagnosis.—Hydrocele of the tunica vaginalis testis, gumma, hematocele, (traumatic and nontraumatic), spermatocele, chylocele, and tuberculosis must be considered in differential diagnosis.

In hydrocele, light is transmitted unless the sac is thickened considerably or calcified, or the hydrocele contents are opaque. Operation, which is indicated in the treatment of hydrocele, will enable one to make a definite diagnosis after inspection of the testis. Paracentesis with evacuation of the hydrocele fluid followed by palpation of the testicle, may be considered.

Gummata more frequently simulate testicular tumors than any other condition. The Wasserman or Kahn blood test and antileutic therapy particularly potassium iodide, may be helpful aids in diagnosis. When doubt exists, it is preferable to remove the gumma than to delay unduly in the removal of a malignant growth.

Traumatic hematocele is characterized by the history and evidence of recent injury, by its solid doughy consistency and opacity of its contents. Traumatic hematocele may be produced by the rupture of a hydrocele.

Nontraumatic hematoceles are due to hemophilia, scurvy, syphilitic endarteritis, severe grades of anemia, arteriosclerosis and malignancy of the testicle.

Spermatocele is characterised by the presence of a small cyst in the substance of the globus major of the epididymis, or attached to it. The cyst is freely movable and the testicle can be well outlined as a separate body.

Chylocele is identified by its tropical origin, being a local expression of filariasis. Associated symptoms of filariasis may be present and the parasite Filaria sanguinis hominis may be identified.

The chylocele fluid is easily recognized by the characteristic upper fat layer which develops after settling.

Tuberculosis offers difficulties in diagnosis only in the uncommon case with extensive involvement of the testicle which forms one mass with the epididymis. In tuberculosis of the epididymis however, the seminal vesicles and prostate are frequently involved, while in tumors of the testicle no change is noted in the vesicles or prostate.

Prognosis.—The prognosis in these cases is determined for the most part by the type of tumor present, the extent of the malignant invasion and by the response to treatment.

In most cases the course of the disease is steadily progressive with an average duration of less than 2 years.

Treatment.—Hinman (4) believes that the treatment of these tumors should be determined by the type of tumor under consideration, and by the presence or absence of metastases which are evident on clinical study. Emphasis is placed upon the belief that seminoma type tumors are radiosensitive while the teratoma are radioresistant.

Ferguson's (5) investigations however, have led him to believe that "the diagnosis of radio resistance on histological grounds is not a safe criterion on which to select treatment." He considers that the biologic response of the tumor to the therapeutic use of irradiation is the only safe criterion of radiosensitivity and is at the same time an accurate prognosic index.

Methods of treatment to be employed are (1) orchidectomy with radium or X-ray therapy; (2) radiotherapy alone and (3), the radical resection of the tumor with its primary lymphatic area.

Radical resection should never be done in cases which present clinical evidence of abdominal mestastases. These are inoperable cases in which life may be prolonged, and the malignant process checked by the use of radium packs and X-ray therapy.

In cases without clinical evidence of metastases, in which, after orchidectomy, the pathologist reports a mixed type tumor, Hinman (4) advocates the radical resection of the tumor with its primary lymph zone.

In pure seminomas with or without metastases, whether radical resection should be done or X-ray or radium therapy alone, be used in the opinion of some surgeons is still an open question.

Hinman's (4) statistics show that of 100 cases in which the radical operation was performed by American surgeons, 17 are alive and clinically free from evidence of the disease after 5 years.

Dean's (6) study at the Memorial Hospital, of the results of treatment by irradiation of 154 unselected cases of teratoma testis, showed that 44 or 29.2 percent of these were alive and without evidence of the disease after 5 years.

Case Report

R. S. Y., Quartermaster, third class, United States Navy. Aged 26. Admitted to Norfolk Naval Hospital August 15, 1935, from the U. S. S. *Wyoming*. Diagnosis undetermined (teratoma, right testicle).

(1) Pains in back, hips, and right lumbar region.

(2) Painless swelling of right testicle.

(3) Constipation.

Five months prior to admission patent experienced dull, aching pains in the flanks and over the region of the right kidney; attacks of pain were intermittent and unaccompanied by any urinary symptoms; constipation was chronic and very troublesome. The attacks of pain gradually became more frequent and more severe, yet the patient did not seek medical treatment until about 2 weeks prior to admission to this hospital. At this time he noticed an increasing painless enlargement of the right testicle. During the past 10 weeks the patient states that he has lost about 25 pounds in weight, although his appetite has remained quite good. While aboard the U. S. S. *Wyoming*, shortly before hospitalization, the patient passed in the voided urine, an organized blood clot about 2½ inches long, the diameter of which corresponded to that of the open urethra.

Irrelevant.

Negative.

(1) There is evidence of marked loss of weight.

(2) There is present an uniform smooth, hard enlargement of the right testicle, moderate in size and only slightly tender. The right epididymis is enlarged and firm and distorted in shape. The right spermatic cord is considerably enlarged and definitely indurated to a point slightly above the external inguinal ring. The cord and epididymis are not tender. The left testicle is moderately atrophied. There is enlargement, slight in extent, of the inguinal lymph nodes on both sides. There is present no penile sore or evidence of urethral discharge. The prostate and seminal vesicles are negative.

In the lower right abdominal quadrant at the mid-right rectus region is an irregular, very firm, and slightly tender mass which seems to be continuous with the right rectus muscle.

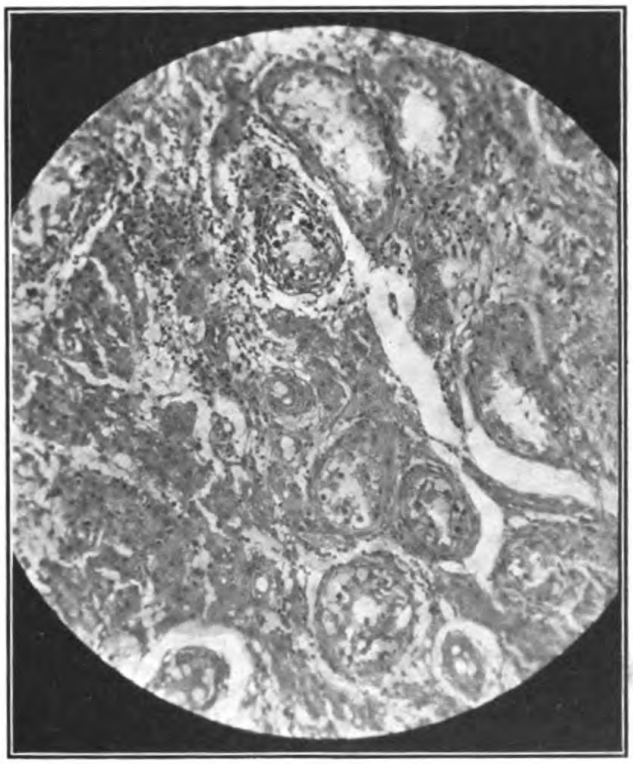

FIGURE 1.—LOW POWER PHOTOMICROGRAPH OF A SECTION OF THE TESTICLE SHOWING INFILTRATION OF THE TUMOR CELLS, ESPECIALLY IN THE LOWER HALF OF THE FIGURE.

Considerable lymphocytic infiltration may also be noted.

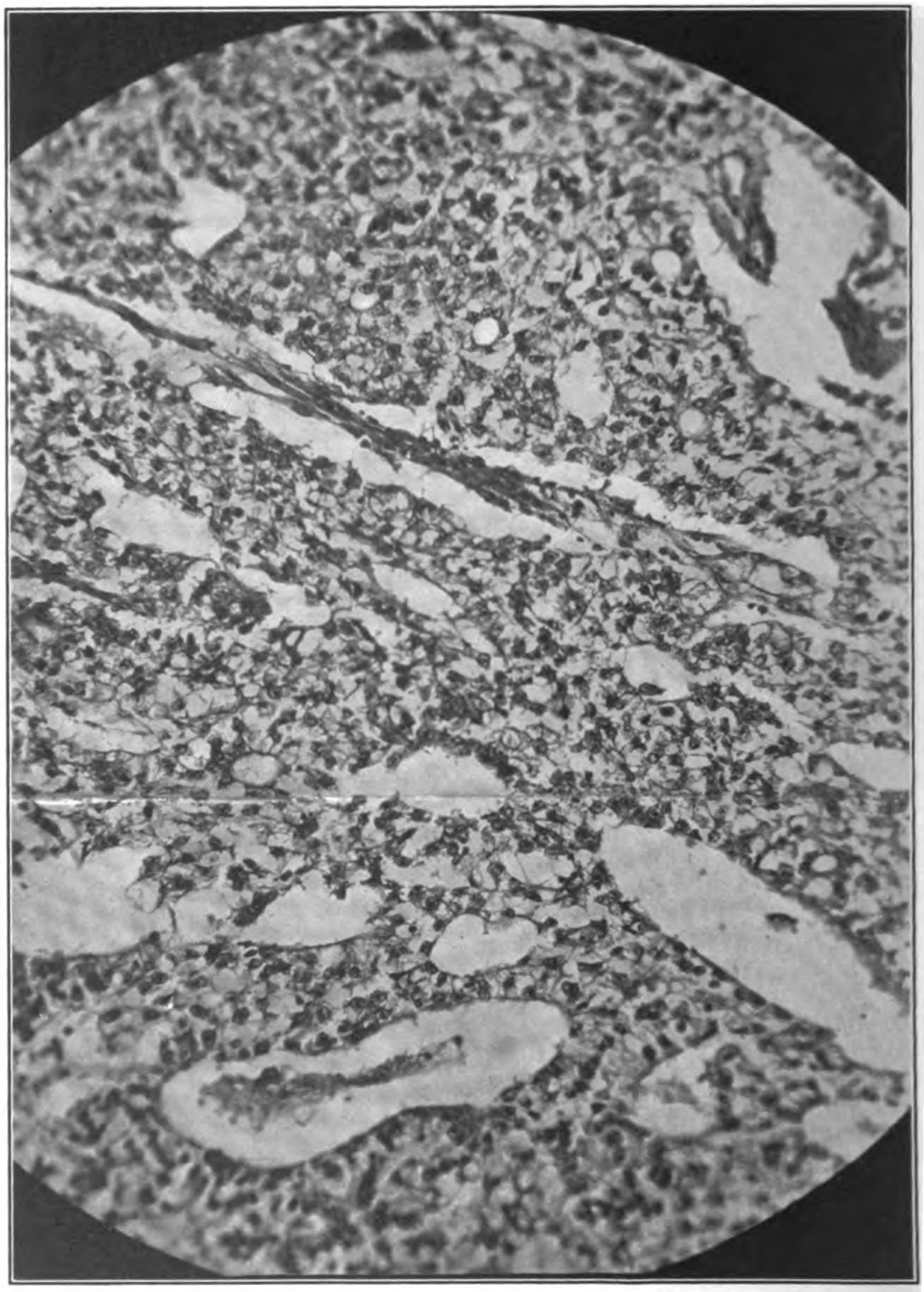

FIGURE 2.—LOW POWER PHOTOMICROGRAPH OF ANOTHER PORTION OF THE TESTICLE SHOWING TUMOR CELLS WITH A VERY LOOSE AREOLAR TYPE OF STROMA.

Laboratory and X-ray reports.—

(1) Urinalysis negative.

(2) Kahn blood test negative.

(3) White blood count 14,000 with normal differential count, red blood count 4,380,000, hemoglobin 80 percent (T).

(4) X-ray examinations of lungs, bony pelvis, and long bones show no evidence of metastases.

(5) Aschheim-Zondek test positive.

Diagnosis: Teratoma, right testicle.

On the day following admission the patient experienced very severe pain in the right testicle and cord; pain was practically continuous and was little influenced by moderate doses of morphine. It was decided therefore to remove the right testicle, epididymis, and involved portion of the cord primarily for the relief of local pain, and in advance of X-ray or radium therapy. At operation August 20, 1935, a moderately large tumorous testicle with enlarged edematous, misshapen epididymis and about 4 inches of enlarged, indurated spermatic cord were removed under spinal anaesthesia. The superficial veins of the tunica vaginalis testis were engorged and tortuous; upon gross examination the testicle and cord appeared to be frankly malignant.

Tissue study of specimen at United States Navy Medical School showed an embryonal carcinoma, right testicle.

On September 12, 1935, deep X-ray therapy instituted. On September 15, 1935, quantitative and qualitative Aschheim-Zondek test was negative.

Cystoscopy reveals a bladder which shows no evidence of infection, ulcer, tumor, foreign body, stone, or diverticulum. The ureteral orifices are normal in appearance, position, and location. The left ureter exhibits a normal urinary efflux, no urine however is observed coming from the right ureter. An ureteral catheter was passed up the right ureter for a distance of 12 centimeters. At this point a very definite obstruction was encountered. No urine was obtained by uretheral catheter. A pyelogram of the right kidney was made using five cc of 12½ percent sterile solution sodium iodide; the pyelogram shows a hydronephrosis of the right kidney with questionable extravasation of the urine and the dye into the soft tissues around the pelvis and down the right ureter. There is noted a tortuous constriction of the right ureter. The pyelogram and ureterogram suggest malignancy. Pressure of retro-peritoneal gland metastases on the right ureter is a considered etiological factor in the production of ureteral constriction and hydronephrosis.

Pain in the pelvic region and thighs has continued and has become more severe in character. The clinical course of the patient has been steadily downward.

Palpable masses in the lower and upper abdomen have developed, the right kidney now can be palpated.

Aschheim-Zondek test negative on September 28, 1935.

To date the amount of X-ray therapy totals 5,560 R units applied to both inguinal regions, upper and lower abdominal regions, post lumbar, lateral, anterior, and posterior kidney regions.

The factors used are 200 K. V. 8 M. A.

Filters ½ mm copper—2 mm aluminum.

Distance—50 cm—Portal 12 by 12 cm.

Roentgenograms October 1935, 7, in search of metastases show a metastatic lesion in the base of the left lung. The pelvis, lumbar spine and long bones are negative for visible metastases.

Aschheim-Zondek qualitative and quantitative test negative this date.

While the negative quantitative and qualititative Aschheim-Zondek tests would appear to indicate a favorable response to Roentgen therapy, subjective symptoms have steadily become more severe.

It is possible that in this case we have had to deal with two unassociated type of malignancy, one type which has involved the testicle and cord and the other type representing the kidney lesion. What seems more likely however, is that the renal malignancy represents a metastatic invasion, the source of which is the seminoma of the testicle. The testicle from all accounts is seldom the seat of a metastatic malignant process.

Pain which has been most severe over the region of the right kidney is in all probability due not only to the questionable metastatic malignancy of the kidney, but also to the associated hydronephrosis with marked impairment of kidney drainage.

The wound of operation has completely healed and shows no evidence of local malignancy.

On October 9, the patient was subjected to an injection of absolute alcohol in the subarachnoid space. The injection was made between the third and the fourth lumbar vertebrae, 2 cc of alcohol being used. Following this injection no relief of lumbar region pain was experienced.

Subarachnoid injections of alcohol were repeated twice at weekly intervals. Little or no relief of pain in legs, hips, and lumbar regions was experienced. Pain and discomfort are in a measure controlled by morphine injections to the extent of 4 to 5 grains daily. Patient is steadily losing ground. Weight loss and emaciation is progressive. There has developed a moderate degree of jaundice. Palpation over the region of the liver elicits definite tenderness. Retroperitoneal masses are easily palpated.

Jaundice has deepened. Patient continues to lose ground.

Patient vomiting frequently today; very weak and almost moribund.

Death occurred at 2:37 a. m., this date.

Report of autopsy, November 25, 1935—general.—Patient showed marked emaciation with deep generalized jaundice. Palpation of the abdomen showed in the region of the right inguinal scar a palpable mass which was freely movable. There was another palpable mass in the region of the right kidney and gall bladder about the size of a large grapefruit. The right testicle was absent. The left testicle showed beginning atrophy.

The body was opened through the usual Y shaped incision. The heart was stopped in systole, the great vessels were normal. The lungs showed moderate degree of hypostatic pneumonia with multiple metastatic nodules varying in size from one-half centimeter in diameter to about 3 centimeters in diameter, the center of many of these nodules show beginning necrosis and degeneration. The abdomen shows in the right lower quadrant a metastatic nodule about the size of a small lemon which is soft and friable and attached to the caecum. There is another small nodule on the dome of the bladder about the size of an acorn, which is very firm and hard. All of the retroperitoneal lymphnodes are enlarged and involved in the progression of the metastatic process. The right kindey shows a large metastatic lesion about the size of a grapefruit which has caused pressure atrophy of most of the kidney substance and in growing has surrounded and compressed the common duct causing an obstructive jaundice. This large metastatic nodule has also impinged on the duodenum causing subacute obstruction. The center of this large metastasis shows marked degeneration and necrosis. The liver shows many nodules varying from the size of an acorn to that of an orange, the larger of these nodules are becoming necrotic. The liver in general is about two times normal size. The remainder of the autopsy is essentially negative.

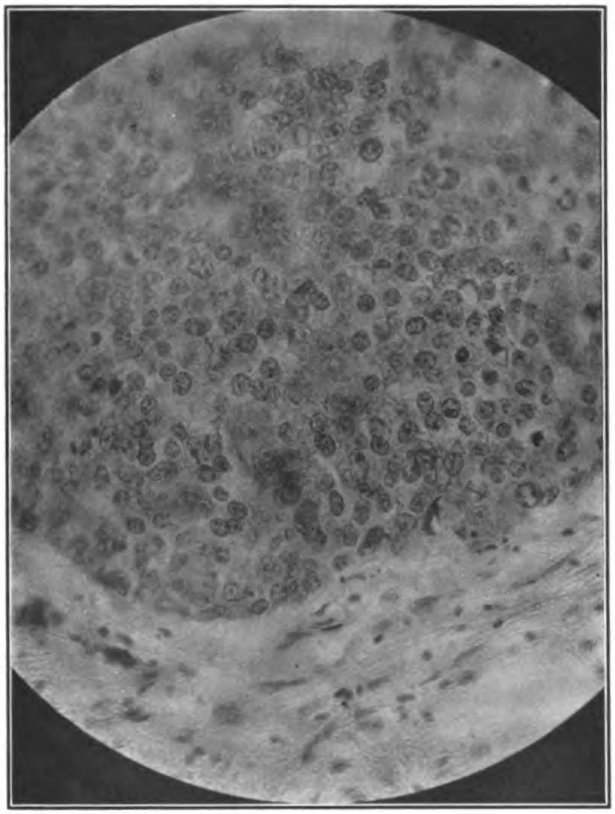

FIGURE 3.—HIGH POWER PHOTOMICROGRAPH OF A MASS OF TUMOR CELLS AS SEEN IN FIGURE 1.

Note the polyhedral shape and large hyperchromatic nuclei. Mitotic figures are also present.

SUMMARY

1. Some general consideration of testicular tumors are submitted with a case report.

2. The case is characterized by insidious onset and by rapid and extensive metastatic invasion.

3. Later in the course of the disease, after Roentgen therapy, the Aschheim-Zondek tests of both qualitative and quantitative nature were negative although the malignant invasion was progressive. This is a point of interest.

4. Examination by autopsy revealed the characteristic invasion of the retroperitoneal glands along with the mediastinal and pre-aortic groups.

5. Subarachnoid injections of alcohol used with such success by various physicians in combating intractable pain of various pelvic malignancies were unsuccessful in this case.

REFERENCES

(1) Young, H. H.: Practice of Urology.

(2) Pierson, E. L. Jr.: A case of bilateral tumors of the testicle with some notes on effects of castration of the adult male. Journal of Urology, September 1932.

(3) Mackenzie, D. W., and Ratner, M.: Malignant growths in the undescended testis. Review of the literature and report of two cases. Journal of Urology, October 1934.

(4) Hinman, F.: Tumors of the testis, 5-year cures following radical operation. Surgery, Gynecology, and Obstetrics, February 15, 1935.

(5) Ferguson, R. S.: Clinical evaluation of the quantitative excretion of prolan A in teratoma testis. Journal of Urology, March 1934.

(6) Ferguson, R. S.: Studies in the diagnosis and treatment of teratoma testis. American Journal of Roentgenology and Radium Therapy, March 1934.

VINETHENE ANESTHESIA [1]

By J. Connolly, Lieutenant, Dental Corps, United States Navy, and R. E. Baker, Lieutenant, Medical Corps, United States Navy

Encouraged by the enthusiasm with which Dr. Edward Beach, professor of anesthesia at the University of Pennsylvania Graduate School of Medicine, recommended to us the use of vinethene [2] (di-vinyl oxide, di-vinyl ether, vinyl ether, vinesthene) in certain types of cases, we decided to give this agent clinical trial. Favorable reports in relation to its use have been had by him from various parts of the country with the exception of the Southern States and it has been partly with a view to the determination of its efficacy

[1] From the Surgical and Dental Departments of the U. S. Naval Hospital, Charleston, S. C.

[2] Vinethene generously supplied by Merck & Co., Rahway, N. J.

in a southern climate that we agreed to try it. In addition, however, we were led to believe that this agent would fill a distinct void in the spectrum of anesthetic agents available for the special surgical and dental conditions which exist in the Navy. It is recommended for use in operations which require general anesthesia not exceeding one-half hour, particularly where quick induction and a rapid recovery are desirable. We were particularly interested in the fact that it could be given by the open-drop method as facilities for the administration of nitrous oxide are not available at this hospital.

Historical.—Semmler, in 1887, described di-vinyl oxide as a liquid obtained from di-vinyl sulphide, a constituent of the volatile oil of the leek, Allium Ursinum.

In 1924, Stehle, Bourne, and Lozinsky, examined a related compound, ethylene oxide $\begin{matrix} CH_2 \\ | \\ CH_2 \end{matrix}\!\!>\!O$, and found that it presents no possibilities as an anesthetic. These findings were confirmed by Johnston in 1931.

In 1930, Leake and Chen, believing that di-vinyl ether (vinethene) should have anesthetic properties because of its close chemical relation to ethyl ether and ethylene, obtain an impure sample of this material from a Princeton student. Although not a pure sample, it was nevertheless found to have anesthetic properties. Ruigh and Major, working in the laboratories of Merck & Co., prepared pure di-vinyl ether, and with this material, in 1931, Leake, Guedel, and Knoefel, working in the laboratories of the University of California Medical School, conducted experiments on mice and dogs, demonstrating its anesthetic efficiency. They determined the minimal anesthetic concentration and the minimum lethal dose and compared them with those for ether and chloroform on the same group of animals. They concluded that it had definite advantages over ether or chloroform and that it was deserving of clinical trial.

Gelfan and Bell first used this agent on humans. In January 1933 they reported the results of its administration on three separate occasions. They reported no signs of excitement or struggling during induction and recovery, no salivation or sweating, and no nausea or vomiting. They stated that it was not unpleasant to inhale, was sweetish in taste, and that it lacked the burning pungency of ethyl ether. It did not apparently irritate the respiratory passages and the induction was impressively smooth, prompt, and even, and the recovery rapid. They, too, felt that this agent was worthy of a clinical trial and evaluation.

Leake reports that di-vinyl oxide, produced by Ruigh and Major in the Merck laboratories, was first used for surgery in the University

of California Hospital in San Francisco, for the removal of a gall bladder from an obese patient.

In 1934, Goldschmidt, Ravdin, Lucke, Muller, Johnston, and Ruigh conducted extensive physiological, pathological, and clinical studies on the dog, the monkey, and man, and report 461 patients anesthetized with vinethene. They used the open-drop method in 90 percent of these cases. The patients varied in age from 5 months to 82 years and in weight from a few pounds to 274 pounds. The following types of operations were performed: Tic douloureaux, brain tumor, middle ear disease, thyroidectomy, gastrostomy, gastro-enterostomy, appendicectomy, herniorrhaphy, mastectomy, and other operations on the soft parts and the bony skeleton. There were two operations for cholecystectomy. In only 2 percent of these patients did any real excitement occur. The induction time was short, 3½ minutes being required for sufficient relaxation for laparotomy and less than that for extra-abdominal lesions. In some patients, good relaxation was obtained in 2 minutes. The length of time patients were anesthetized varied from 10 minutes to 2 hours and 51 minutes. There were no deaths in their series. They noted that the amount of vinethene necessary for the maintenance of anesthesia was, on the average, about 2 cc per minute. They noted no evidence of renal irritation or liver damage. They showed that the anesthetic potency of vinethene is four times that of ether and one and three-tenths greater than that of chloroform. They found liver necrosis in some dogs after prolonged vinyl ether anesthesia, but no liver necrosis occurred in monkeys. A footnote on their reprint states that "since this paper was written the number of patients subjected to di-vinyl ether anesthesia has exceeded 2,000. Four patients have come to autopsy within 5 days after operation and in two of these evidence of a central lobular necrosis of the liver was found."

Dr. Beach, in September 1935, told us that the number of administrations of vinethene at the University of Pennsylvania would, at that time, approximate 10,000.

In March 1934, Bourne reported the administration of vinethene in 152 parturient women and described further experiments using normal dogs, dogs with chloroform damaged livers, and partially starved dogs. He later reported 500 more administrations in obstetrical cases, making a total of 652 administrations. One death occurred, as mentioned below.

With the first 50 obstetrical patients, the bromsulfalein dye test for liver function was carried out on four, including a very "toxic" woman of the renal type. These were compared with similar tests made after chloroform administered under similar conditions. These tests showed that after vinethene anesthesia, very little dye

retention occurred, whereas, the administration of chloroform was followed by marked dye retention, indicating definite liver impairment.

As a result of the experiments carried out on dogs he reached the following conclusions:

1. Vinyl ether (vinethene) anesthesia in normal dogs does not alter the liver function appreciably. In those cases in which cyanosis is a feature of the anesthesia, moderate liver damage occurs, which is not due directly to the drug but to the associated anoxemia.

2. Vinyl ether anesthesia does not enhance the liver function damage produced previously by the inhalation of chloroform, not does it delay the period of recovery from this damage.

3. The effect of vinyl ether anesthesia on the liver function in partially starved dogs is not appreciably different from that produced in normal animals.

Bourne, in a later article, quotes the experiments of Molitor with rabbits. Molitor introduces cannulas into the bile duct and duodenum of rabbits and observes the effect of anesthetic agents and anoxemia on the rate of bile flow. He found that there was no change in the usual flow of bile when vinyl ether was used, even in a 2-hour administration and profound anesthesia.

He further quotes experiments by Molitor (confirming similar experiments by Peoples and Phatak) in studying the effect of vinethene on rabbit's intestine. He showed that vinethene does not interfere with the automatic movements of the intestines, whereas, ether will stop these movements. He also found that vinethene does not affect uterine contractions and this agrees with Bourne's clinical observations.

Bourne and Sparling, in collaboration with Dr. A. F. Foss, report the use of vinethene in 143 dental cases with most gratifying results. They believe it very useful in difficult dental cases as an addition to nitrous oxide.

In January 1935, Sir Francis Shipway reported a total of 314 administrations of vinethene. In one of these cases it was combined with nitrous oxide and oxygen for 1 hour and 32 minutes during an operation for cholecystitis. The patient, a 73-year-old man, jaundiced at operation, made a perfect recovery. On two other occasions he also gave it combined with avertin and nitrous oxide and oxygen for operations on the gall bladder. His observations agree with those of Goldschmidt, Ravdin, Lucke, Muller, Johnston, and Ruigh.

Marvin, in December 1935, reports the use of vinethene in several types of operations, including dental cases.

Stumpf, in September 1935, reporting some 50 cases in which he has used vinethene, writes as follows: "From our experience we may say that vinyl ether should have a very definite place among anesthetic agents. It is outstanding as a pleasant rapid anesthetic with

quick recovery and safety comparable to that of ethyl ether. This should appeal to the average physician when the expert anesthetist and his gas apparatus are not available."

Physical and chemical properties.—Di-vinyl ether, or di-vinyl oxide, has the chemical formula of $CH_2:CH.O.CH:CH_2$ and is a clear, colorless, liquid having a specific gravity of 0.77. The boiling point is 28.3° C. Its vapor pressure is greater than ether; that is, it is more volatile than ether. It has a sweetish ethereal odor resembling that of ethylene, to which it is closely related chemically. It is more inflammable than ether and is relatively unstable in the chemically pure state as it forms resin-like polymers and decomposes with the formation of formaldehyde and formic acid in the presence of light and air. It is readily decomposed by traces of acid (this may be prevented by a faint trace of alkali).

It decolorizes $KMnO_4$ solutions and his reaction may be used as a means of detecting its presence in certain biological fluids.

As prepared for anesthesia, under the name of vinethene, it is said to contain 0.01 percent phenyl-alpha-naphthylamine to prevent polymerization and decomposition, and 3.5 percent absolute alcohol to prevent freezing on evaporation.

Mortality.—We have been able to locate the detailed account of three deaths occurring after the administration of vinethene in which the anesthetic may have had some part in producing death. Two of these occurred at the Johns Hopkins Hospital and were quoted by Bourne through the kindness of Dr. Dean Lewis. The third was one of Bourne's patients, an obstetrical case. The complete details of all three cases are given by Bourne in his paper on vinethene in the Journal of the American Medical Association of December 21, 1935, and the reader, if interested, is urged to review them. We should only like to quote Bourne's remarks, as follows: "Having now obtained more exact knowledge of the pathologic changes concerning the two deaths at the Johns Hopkins Hospital just mentioned, and with Professor Waugh's report before me, I feel justified in the belief that vinyl ether was not the cause of the liver damage in any of these three cases. Although I do admit that it probably did enhance liver impairment in the first two anesthesias, which were unusually long, it is very likely that the results would have been the same had ethyl ether been the anesthetic. This opinion is expressed because it has already been clearly shown from repeated experiments that vinyl ether does not effect any more impairment of hepatic function than does ethyl ether. In these experiments there were no complicating factors and the conditions were under absolute control. Who can say with any sense of assurance that the livers in these three clinical cases were not diseased before the administration of vinyl ether?"

In view of the fact that this agent had been recommended to us for use only for short operations (not exceeding one-half hour), we decided to try it first on dental patients. To date we have administered vinethene on 20 separate occasions and have tabulated our results below. We realize that this is too small a number of cases to make any definite conclusions as to its value but we are of the opinion that our observations may induce others to give this agent a trial. We have given it to dental patients on 17 occasions, twice for the incision of an ischio-rectal abscess, and once for the manipulation of a compound, comminuted, fracture of the tibia and fibula. In all cases in which we have used it, the anesthesia was satisfactory for the purpose for which it was given. We had some difficulty with our first patients in getting them "under" but this was due to our inexperience with a new anesthetic and our fear of overdosage. Because of the rapid recovery, there are technical disadvantages in its use in dentistry, but these can be overcome with experience. Gauze mouth packs, to which a string is attached, furnish a useful means of closing the mouth to the passage of air while a small inhaler is kept over the nose. However, caution must be observed in order to prevent placing of the pack too far posteriorly or the soft palate will be forced against the pharyngeal wall and shut off the nasopharynx. The pack also aids in absorption of blood and saliva although we have used a suction machine in most of the dental cases.

Vinethene is more useful in extractions of the anterior teeth for the reason that extraction of molar teeth requires the gauze pack to be placed too far back in the mouth.

The anesthetic was considered agreeable by the patients in all cases. Two of our patients were given vinethene a second time and one patient (Mrs. B.) received this anesthetic on three separate occasions without untoward result.

It was noted on several different occasions that, although the patient might cry out toward the end of the operation, that after complete recovery, he would have no recollection of pain, showing that analgesia was present even when the anesthesia was incomplete.

SUMMARY

1. A brief historical review of the development and use of vinethene is presented.

2. Observations on its use on 20 separate occasions are made.

3. Vinethene appears to be a safe anesthetic which may be given by the open-drop method and which will provide a rapid induction and quick recovery.

4. We believe it valuable for dental operations and minor surgical operations, as a substitute for nitrous oxide, when that is not available.

Vinethene data

Case no.	Initials	Rate	Date	Operation	Amount used	Induction period	Total time of administration	Recovery period	Blood pressure	Pulse	Nausea	Cyanosis	Spasmodic laughter	Excitement
					cc	*Min.*	*Min.*	*Min.*						
1	A. T.	C. C. C.	Sept. 25, 1935	Extraction of 1 tooth.	25	15	16	2	Slight increase.	Slight increase.	None	None	None	Slight.
2	C. A. J.	C. C. C.	Sept. 26, 1935	do	18	4	5	½	No change.	do	do	do	do	None.
3	L. P.	BM. 2c.	Oct. 9, 1935	Extraction of 2 upper molars.	25	1½	3	½	Slight increase.	Slight decrease.	do	do	do	Do.
4	W. R. M.	C. C. C.	Oct. 16, 1935	Extraction of 1 tooth.	(1)	11	4	4	No change.	No change	Slight ether	do	do	Marked (ether).
5	W. R. M.	C. C. C.	Oct. 21, 1935	Extraction of 5 teeth.	40	6	6	2	Slight increase.	Slight increase.	None	do	do	None
6	W. E. K.	SC. 3c.	Oct. 24, 1935	Extraction of 1 molar.	50	9	9	2	No change.	do	do	do	Present.	Do.
7	Mrs. B.	Civ.	Oct. 28, 1935	Extraction of 4 teeth.	35	5	5	3	Slight increase.	do	Very slight	do	None.	Marked.
8	A. M. M.	MM. 2c.	Nov. 6, 1935	Extraction of 3 teeth.	25	4	4	2	do	do	None	do	do	None.
9	H. R. S.	Lieut.	Nov. 25, 1935	Extraction of 2 impacted molars.	40	4	7½	1½	do	do	do	do	do	Do.
10	J. S. B.	C. C. C.	do	Extraction of 4 teeth.	35	2½	5	1	do	do	do	do	do	Do.
11	J. W. U.	Pvt.	Dec. 20, 1935	Extraction of impacted lower third molar.	40	2	4	3	do	do	do	do	do	Do.
12	W. N.	C. C. C.	Dec. 27, 1935	Extraction of 1 tooth.	15	4	4	2	No change.	do	do	do	do	Do.
13	Mrs. B.	Civ.	Dec. 30, 1935	Extraction of 8 teeth.	35	5	8	2	do	do	do	do	do	Slight.
14	H. D. M.	Sgt. (F. N. R.).	do	Incision of abscess, ischio-rectal.	25	2¾	2¾	1½	Not taken.	Not taken.	do	do	do	None.
15	H. D. M.	Sgt. (F. N. R.).	Jan. 1, 1936	do	20	2	4	2	do	do	do	do	do	Do.
16	I. S. W.	C. C. C.	Jan. 17, 1936	Extraction of 3 molars.	65	2	4	2	Slight increase.	Slight increase.	do	do	do	Do.
17	Mrs. B.	Civ.	Jan. 20, 1936	Extraction of 10 teeth.	30	7	7	2	No change.	do	do	do	Present.	Slight.
18	W. G. N.	C. C. C.	Jan. 23, 1936	Extraction of 3 molars.	50	9	10½	1½	Slight increase.	do	do	do	None.	Do.
19	N. M. N.	C. C. C.	do	Extraction of 1 molar.	65	6	10	1	do	do	do	do	do	Do.
20	I. S.	Civ. (P. W. A.).	Feb. 8, 1936	Manipulation of fracture, left tibia and fibula.	20	1	2	2	do	do	do	do	do	Do.

[1] 25 cc and ¼ pound ether.

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REFERENCES

(1) Dr. Edward Beach, professor of anesthesia, Graduate School of Medicine, University of Pennsylvania.

(2) Leake, C. D. The Role of Pharmacology in the Development of Ideal Anesthesia. Journ. A. M. A., vol. 102, no. 1, Jan. 6, 1934, pp. 1–4.

(3) Council on Pharmacy and Chemistry (A. M. A.), Vinyl Ether. Jour. A. M. A., vol. 102, no. 1, Jan. 6, 1934, pp. 44.

(4) Leake, C. D., Knoefel, P. K., Guedel, A. E. The Anesthetic Action of Di-Vinyl Oxide in Animals. Jour. of Pharm. and Exper. Ther., pp. 5–16, January 1933, vol. XLVII, no. 1.

(5) Johnston, J. F. A. On the Anesthetic Action of Furan. Jour. of Pharm. and Exper. Thera., vol. XLIII, no. 1, September 1931, pp. 85–88.

(6) Goldschmidt, S., Ravdin, I. S., Lucke, Baldwin, Muller, G. P., Johnston, C. G., Ruigh, W. L. Di-vinyl Ether. Experimental and Clinical Studies. Jour. A. M. A., Jan. 6, 1934, vol. 102, pp. 21–26.

(7) Marvin, F. W. Clinical Use of Vinethene. Analgesia and Anesthesia, November–December 1935, 257–262.

(8) Jackson, D. E., Herzberg, M. A Study of Analgesia and Anesthesia with Special Reference to such substances as Trichlorethylene and Vinesthene (Divinyl Ether) Together with Apparatus for their administration, etc. Anesthesia and Analgesia, September–October 1934, pp. 198–204.

9. Wesley Bourne. Vinyl Ether Obstetric Anesthesia for General Practice. Jour. A. M. A., vol. 105, no. 25, Dec. 21, 1935, pp. 2047–2051.

(10) Gelfan, S., Bell, I. R. The Anesthetic Action of Divinyl Oxide on Humans. Jour. of Pharm. and Exper. Ther. Vol. XLVII, no. 1, pp. 1–3, January 1933.

(11) Bourne, W., Raginsky, B. B. Vinyl Ether (Vinesthene) Anesthesia in Dogs—Effects upon Normal and Impaired Liver. Brit. Jour. of Anesthesia, vol. XII, no. 2, January 1935, pp. 62–69.

(12) Bourne, W., Sparling, D. W. Some Aspects of Vinyl Ether (Vinethene) Anesthesia. Anesthesia and Analgesia, January–February 1935, pp. 4–7.

(13) Shipway, Sir. F. E. Vinethene. Brit. Med. Jour., Jan. 12, 1935, pp. 82–84.

(14) Bourne, W. Di-vinyl Oxide Anesthesia in Obstetrics. The Lancet, Mar. 17, 1934, pp. 566–567.

(15) Knoefel, P. K., Guedel, A. E., Leake, C. D. Experimental Observations on the Anesthetic Properties of Di-vinyl Ether. Proc. Soc. Exper. Biol. and Med., November 1931, pp. 139–140.

(16) Stumpf, E. H. Clinical Experiences with Vinyl Ether. Jour. Amer. Inst. of Homeopathy, vol. XXVIII, no. 9, pp. 546–548, September 1935.

(17) Rosenthal, S. M., White, E. C. Clinical Application of the Bromsulfalein Test for Hepatic Function. Jour. A. M. A. 84:1112. Apr. 11, 1925 (quoted by Bourne).

(18) Peoples, S. A., Phatak, N. M. Proc. Soc. Exper. Biol. and Med. 32: 378. November 1934 (quoted by Bourne).

(19) Lundy, J. S., Tovell, R. M. Some of the Newer Local and General Anesthetic Agents. Northwest Medicine, 33:308. September 1934.

PELVIC SURGERY AND GYNECOLOGY

By JOSEPH L. SCHWARTZ, Lieutenant Commander, Medical Corps, United States Navy

Perhaps it seems a far cry to present a discussion of gynecological diseases, since the average medical officer in the Navy only infrequently comes in professional contact with such conditions. However, the out-patient department of a naval dispensary is a fertile field for such diseases and to the medical officer assigned to this detail a wealth of gynecological and surgical material is available.

As an example, the following figures taken from the reports of the naval dispensary, Long Beach, Calif., for a 12-month period reveals the following:

Office consultations	26,753
Major operations	196
Minor operations	386
Confinements	315

GONOCOCCUS INFECTION OF THE FEMALE GENITAL TRACT

It is necessary to consider the pathological sequence in the development of the disease in order to gain a concept of the proper treatment.

Extending by direct contiguity of surface, the gonococcus passes along the endometrium to invade the mucosa and muscularis of the fallopian tube with the production of a catarrhal or purulent discharge. The escape of the infective contents into the pelvis sets up a peritoneal irritation, which, in the process of healing, is productive of moderately firm adhesions, binding the uterus and adnexa to the adjacent and neighboring structures, with the result that we find, especially in the chronic case, the uterus, tubes, ovaries, sigmoid, cecum, appendix, loops of small intestines, and omentum, fixed in the pelvis by a mass of these adhesions.

If the virulence of the organism is low a catarrhal salpingitis is the result, with little structural alteration of the tube or surrounding tissues. With repeated catarrhal attacks the fimbriated end of the tube becomes occluded, either as a result of adhesions of the serosa with an inversion of the fimbriae into the lumen of the tube or as a result of adhesions of the fimbriae to the ovary or to neighboring structures, the mouth of the tube becomes sealed. Inflammatory swelling of the uterine end of the tube in conjunction with occlusion of the fimbriated end, renders a sac which becomes distended with the secretion of the mucosa, resulting in a hydrosalpinx.

A virulent infection produces a suppurative process resulting in a thickened, edematous, engorged infiltrated tube discharging pus-a-pus-tube. If the uterine and fimbriated ends of the tube become

occluded a pyo-salpinx results. In the presence of a pus-tube with the fimbriated end of the tube adhering to the ovary, a rupture of a Graafian follicle leaves an open path for the invasion of the gonococcus into the ovary, and the soft hemorrhagic tissue of the corpus luteum forms an excellent culture medium for the resulting abscess of the ovary. Frequently an abscess of the ovary communicates with the adjacent pus-tube by a narrow channel—a tubo-ovarian abscess.

The ovary is very commonly involved, not necessarily in abscess formation, but mostly in a sclerocystic process. These cysts of the ovary rarely attain any great size, averaging 1 to 3 centimeters. The cysts are atresic follicles that have failed to rupture owing to the thickening of the tunica albuginea. If maturation precedes the cyst formation the result is a lutein cyst. These are usually small single cysts with clear contents, although occasionally hemorrhagic.

Tubo-sigmoid abscess is observed very infrequently; we have noted the occurrence on three occasions. The fimbriated end of the tube becomes intimately adherent to the sigmoid, the serosa and the muscularis of the sigmoid are destroyed, and in this defect the tube is embedded. Through a small perforation in the mucosa of the sigmoid, a communication is maintained between the lumen of the tube and the bowel, with pus discharging from the tube into the bowel.

The earliest manifestation of gonococcus infection, and at times the only manifestation, may be an acute Bartholin gland infection, an acute Skene duct infection, or acute endocervicitis. However, any combination of these infections may be present, but the acute endocervical gland infection is the common one.

In acute catarrhal salpingitis, the abdominal signs and symptoms may be either vague or misleading. In the usual case of acute salpingitis, the onset is rather stormy, the fever high, the leucocyte count is high with a proportionate increase in the polymorphonuclear cells, nausea, vomiting, and abdominal pain is a prominent symptom, while abdominal rigidity is absent. In the subacute infection an adnexal mass or thickening may be made out on bimanual vaginal examination, the temperature and leucocyte count may approach normal limits, but the accelerated blood sedimentation time indicates the presence of active infection. Great stress should be placed on the sedimentation rate in salpingitis as it gives one a definite indication of the presence or absence of active infection and no case should go to surgery, unless some abdominal complication makes surgery urgent, until the sedimentation time is within normal limits.

At times patients present themselves with symptoms referable to the lower genital tract only, with no infection evident in the pelvic

adnexa. Again, patients are seen with an acute, subacute or chronic infection in whom no history of any symptoms referable to infection in the lower genital tract, can be obtained. Yet sight of these sources, that play such a major role in the production of chronic pelvic inflammatory disease, must always be kept in mind. Clinical and laboratory studies by numerous observers over a period of time convinces one that gonorrhoea persists in the female because of the continued presence of the gonococcus in the endocervical glands as well as the glands of the lower genital tract—urethral, Skene's and Bartholin glands. Studies carried out by Curtis on cultures of thoroughly ground tubes have shown that the gonococci do not persist in the tubes, but that they disappear from the tubes within a period of 2 wekes after the temperature and leucocyte count has returned to normal. His coincidental studies of the gross pathological changes in the tube has further demonstrated that a single infection seldom results in a marked increase in the thickness of the wall of the tube, and that greatly thickened tubes that are found in patients who have recovered from an acute infection is evidence of previous infection.

Treatment.—Acute salpingitis, alike acute gonococcus infection of the urethra in the male, is a self-limited disease and under a proper regime the greater percentage of patients will recover and will remain well, unless reinfection takes place. If the foci responsible for reinfection are eliminated and if she can be protected from reinfection by the male carrier of the gonococcus, we can expect an ultimate clinical cure with no signs or symptoms of residual pelvic pathology.

In the acute stage, bed rest, the application of heat to the abdomen in the form of hot compresses or a heat cradle, and the use of exdouches. Vaginal douches under pressure predispose to tubal reinfection. Vaccines are of value.

As the condition becomes subacute and in the chronic case, we avail ourselves of the use of the Elliott method or hot vaginal douches and the employment of vaccines. Patients are to be instructed to take the douches flat on their backs, with the hips elevated and to use at least 2 gallons of solution as warm as can be taken comfortably. We have tried out various vaccines and various foreign proteins and have found that protein shock therapy, by giving increasing doses of typhoid vaccine intramuscularly at frequent intervals, in conjunction with the Elliott method or the use of hot douches properly taken, has given our patients eminently satisfactorly results.

However, no case will remain cured under this regime alone. As has been indicated reinfection will inevitably ensue unless the foci of reinfection in the lower genital tract are eradicated. Nor can we expect to cure a patient harboring these infections, by the removal

of the tubes, or body of the uterus and tubes. Many patients will return with a recurrence of symptoms because of failure to eradicate the foci before operation.

Of the many methods employed in treatment, a very common one directed to the cervix is the topical application of drugs or medicated tampons. The use of tampons in infection of the cervix is mentioned to be condemned, since the tampon acts only to prevent the escape of discharge from the cervix, while the medications employed acts only to coagulate the mucopurulent discharge and is only rarely of beneficial effect. Besides, it is necessary to apply medication to the cervix continuously and over a period of time without assurance that the medication will penetrate the endocervical glands where the gonococcus is harbored.

An admirable means of treating infections of the cervix is by means of the small spatula-tip cautery, heated to white heat. Linear cauterizations 3 to 6 mm deep and about one-half cm apart are made, beginning at the everted edematous eroded mucosa about the external os and extending well up into the canal. Nabothian cysts are punctured with the cautery at this time also. The cauterization is repeated after an interval of 6 to 8 weeks and two or three cauterizations will usually suffice to produce an excellent healing. Often it is necessary to dilate the cervix, by passing a series of small Hegar dilators, before cauterization. Following the completion of healing it is well to again pass dilators into the cervix, in order to prevent the formation of an obstructive lesion of the cervix.

In the extensive infections of the cervix associated with hyperplasia and marked cyst formation, the operative removal of the infected cervical tissue by the Sturmdorf operation becomes necessary.

Skene duct infection is difficult to eradicate. Passing a probe, into the ducts, on which lunar caustic has been fused is effective in many cases. Often it becomes necessary to destroy the ducts with the cautery, by passing a curved needle eye first into the duct for its full length, bringing it out through the tissues and laying the duct open with the needle acting as a guide. This can be done as an office procedure under local anaesthesia.

Bartholin gland infection in the acute stage, with inflamed edematous tissues over a gland distended with pus is treated by incision. In the chronic infection the gland may be removed by dissection. Dissection of the gland is facilitated by first aspirating the contents and then injecting melted paraffine that is stained with methylene blue. Upon hardening, the paraffine filled gland makes dissection less difficult. The excision of a Bartholin gland is a procedure not to be done in the office. A simple and effective

method of destroying the secreting membrane of this gland is to aspirate the contents and with the needle in situ, a solution of quinine-urethane is injected and aspirated repeatedly, leaving a few drops before withdrawing the needle. Another method is to incise the gland, evacuate the contents and instill a sclerosing solution after the method described by Cutler and Zollinger.

Operative intervention.—Surgery is reserved for the chronic cases presenting residual pathology with symptoms referable to the adhesions involving the ovaries, intestines, omentum and so forth, and for those patients who have not obtained relief after prolonged and properly directed treatment. Certainly in the acute infection surgery is never indicated or justifiable unless complicating pathology such as an acute intestinal obstruction makes surgery necessary. Parenthetically, diagnostic errors occasionally lead to surgery, as in the cases that present clinical and laboratory findings suggestive of acute appendicitis, where on opening the abdomen we find a pair of slightly engorged and edematous tubes, which on massaging exude creamy pus. These cases however are very infrequent. Again we occasionally encounter a case presenting abdominal pain, rapid pulse, an adnexal mass, a leucocyte count within normal limits, a moderately accelerated sedimentation time and a history of amenorrhea followed by menorrhagia, to find not the expected ectopic gestation but a subacute salpingitis.

The avoidance of surgery in the acute cases is stressed, since conservative or reconstructive surgery in these cases is not possible. Certainly in the presence of tubes and ovaries that are edematous, friable, and distended with pus, tissues will be mutilated and sacrificed that may conceivably be retained when the infection has subsided entirely. Difficulty will be experienced in separating adherent edematous small and large intestinal loops and the anatomical distortion making injury to the intestinal loops or ureter a potential danger. The end results of surgery in the acute or subacute case is not all that may be desired, and we invoke the potentiality of postoperative intestinal obstruction as well as the invaliding syndrome of the prolonged surgical menopause.

In the chronic infection, with the sedimentation rate normal, conservative surgery is feasible, freeing adhesions may be done without much danger of injury to the intestines, it may be possible to free the fimbriated ends of a chronic hydrosalpix and with the uterine end of the tube free, sterility may be overcome. There is much less danger of interference with ovarian function. Conservation of the ovaries should always be practiced. However, where one ovary appears hopelessly diseased its removal should be practiced rather than a resection. If both ovaries and both tubes are hopelessly

involved, the fundus of the uterus should also be removed. In removing the tubes it is always necessary to also remove a wedge of the fundus at the cornu.

All raw surface should be thoroughly peritonealized. Using the round ligaments for this purpose also serves to maintain the uterus in normal position. Omental transplants are to be used where necessary or the sigmoid can be used to cover over raw surfaces. Drainage of the pelvis is rarely necessary. The appendix is removed routinely unless prohibited by the condition of the patient.

The anaesthetic of choice in pelvic surgery is spinal enaesthesia preceded by fairly large doses of nembutal, as a basal anaesthesia. Often patients are seen whose symptoms are the result of having their attention directed to the pelvis, by the unintentional remarks on part of the examining physician.

It should be recalled that in the nullipara, about 20 percent are found to have the uterus in either retroversion or retroflexion, while in the multipara both retroflexion and retroversion are very common. Without the association of definite pelvic pathology or marked relaxation of the pelvic floor, these uteri are not productive of symptoms. The cause for backache in the presence of uncomplicated retroversion or retroflexion should be sought in an endocervitis, sacro-lumbar arthritis, sacro-iliac slip, poor posture, overweight, poor muscle tone, focal infection, and so forth.

The uncomplicated retroversion or retroflexion requires no treatment. With retroflexion or retroversion in the presence of a relaxed pelvic floor, either as a result of subinvolution or laceration of the levator muscles, if the uterus can be brought well forward without difficulty, the adjustment of a suitable pessary is the preferable form of treatment. Knee chest position and similar exercises are valuable adjuncts in treatment.

Cases in which the adjustment of a suitable pessary and the knee chest position does not give relief, and those cases complicated by ovarian or tubal pathology, are amenable to and suitable for surgery. with a relaxed pelvic floor a perineal repair is necessary, in conjunction with a uterine suspension of the Crossen-Gilliam type.

As noted in the foreword the outpatient department offers many and varied interesting surgical and gynecological experiences. The following is a tabulation of some major abdominal and pelvic operations personally performed while on duty at naval dispensaries.

Operation	Number	Indication	Remarks
Amputation breast, radical	5	Carcinoma	No follow-up after 2 years.
Excision branchial cleft	2	Fistulous tract	
Decompression osteoplastic flap.	2	Subdural hematoma	1 death, patient with secondary lues.
Thyroidectomy	3	Colloid	1 patient had 2 previous operations and thyrotoxicosis followed eclampsia.
	4	Adenoma	
	6	Toxic	
Cholecystectomy	8	Cholecystitis	1 death.
	5	Cholelithiasis	
Suture duodenum	5	Perforated ulcer	1 death. Parotitis postoperative. Ill 20 hours before admission.
Gastro-Jejunostomy, posterior.	2	Ulcer duodenum carcinoma stomach with obstruction.	Patient with carcinoma died, urinary suppression.
	1		
Gastrostomy	1	Congenital atresia oesophagus.	1 death. Child 3 days old died sixteenth day. Pneumonia.
Intestinal resection and anastomosis—enterostomy.	1	Intestinal obstruction; gangrene Meckels diverticulum.	Noted postoperative 4 days after appendectomy—child.
Intestinal resection and double-barrel enterostomy.	1	Gunshot, wound, abdomen	Death, fourteenth day.
Resection Meckels diverticulum.	2	Noted at operation	
Enterostomy	3	Intestinal obstruction	Condition of all cases critical.
	2	Perforation intestine by ascaris lumbriocoide.	1 death in intestinal obstruction. 1 death in perforation.
Appendectomy	71	Acute appendicitis	1 death. Previous drainage for suppurative appendicitis 1 year before. Ill 24 hours developed obstruction postoperative.
	35	Chronic appendicitis	
Nephrectomy	2	Pyonephrosis	
Do	1	Nephrolithiasis	
Caesarian section, low cervical.	9	Disproportion	1 death (abruptio) due postoperative.
	6	Preeclamptic toxaemia	Pulmonary edema.
	1	Abruptio placenta	
	1	Asthma	Asthmatic developed in third month. No evidence or history of allergy. Relief, postoperative.
	2	Placenta previa	
Salpingectomy or salpingo-oophorectomy or salpingoplasty.	154	Chronic pelvic inflammatory disease. Salpingoplasty in 8 cases.	1 case of postoperative obstruction. 2 conceived after salpingoplasty.
Supravaginal hystero-salpingectomy.	18	Chronic pelvic inflammatory disease.	
Supravaginal hysterectomy	26	Fibroid uterus	Includes 1 case on which pan-hysterectomy was done.
	4	Persistent menorrhagia	
Celiotomy	18	Ectopic pregnancy	
	2	Hemorrahge from corpus luteum.	
	15	Intestinal obstruction	
Oophorectomy	17	Cystadenoma or dermoid	
	8	Intraligamentous cyst	
	2	Papillary cyst-adenoma	Postoperative X-ray therapy.
Suspension of uterus	26	Retroversion or retroflexion with persistent symptoms.	
Celiotomy with marsupialization of lesser peritoneal cavity.	2	Pseudo-pancreatic cyst	1 case developed after intra-abdominal injury. No cause determined in other case. Each contained over 1,500 cc of enzyme containinr fluid.
Herniorrhaphy	24	Inguinal	Includes 1 strangulated femoral hernia and 1 strangulated hernia.
	8	Femoral	
	6	Umbilical	
	5	Incisional	
	2	Epigastric	
Total number	506		
Deaths	10		

NOTE.—Appendectomy done in course of other operation not noted.

Operative procedures involving cervix, perineum, etc., done in course of pelvic surgery not noted.

REFERENCES

Boyd: Surgical Pathology.

Curtis, Arthur H.: Indications for Surgical Intervention in Pelvic Lesions. Journal A. M. A., vol. 89, no. 15. Oct. 8, 1927.

Matthews, Harvey B.: Electric Cautery versus Sturmdorf Operation. Journal A. M. A., vol. 87, no. 22. Nov. 27, 1926.

Cutler and Zollinger: American Journal Surgery, vol. XIX, p. 411. March 1933.

Polak, John Osborn, and Tollefson, Donald G.: Clinical significance of the Sedimentation Test. Journal A. M. A., vol. 90, no. 3. Jan. 21, 1928.

Crossen: Operative Gynecology.

DeLee: Principles and Practice of Obstetrics.

ANOMALIES OF DEVELOPMENT OF THE LUMBAR SPINE

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In the examination of the spine by means of the X-ray, peculiarities of structure and development are often noted. Sometimes these are due to lack of development and sometimes they may be the result of a fault in posture. This is especially true of the lumbar spine. It has been said, and it is believed to be true, that there are more changes from individual to individual in this region than in any portion of the bony skeleton.

In these examinations one should never be satisfied with an anteroposterior view alone but should always have a lateral as well. Indeed it is believed the latter is the more important of the two because only in the profile can the examiner see the fainter lines.

Examples of the diversities mentioned are easily called to mind: six lumbar vertebrae instead of five, differences in the length of the transverse processes of the same bone, double or triple transverse processes on one or both sides. Then there are the more important changes such as spondylolisthesis, scoliosis, and retardation of development of the bodies of the vertebrae. And to the last three it is intended to confine the discussion in this paper.

It should always be borne in mind that where some of these differences are found other peculiarities may also be noted if a thorough study of other bones is made. This is particularly true of developmental defects, and the examiner will often be rewarded by an examination of other bones, especially the skull.

It is necessary in an explanation of what follows to describe the growth and anatomy of the vertebral column and of the individual bones.

The vertebrae are gradually differentiated in embryonic life from the mesodermal somites, the apical portion of which is a scleratogenous segment. These cells rapidly proliferate, gradually migrating along the neural tube and by union with their fellow on the opposite side finally form a continuous sheath. This is the membranous vertebral column. Ossification of the arch and of the body takes place entirely independent of one another. That of the arches begins in the atlas and extends from there downward, while ossifi-

cation of the bodies begins in the lower dorsal vertebrae and extends upward rapidly and downward less so.

Each vertebrae has three centers of ossification, one or possibly two for the body and one for each side of the neural arch. The ossification of the body begins about the third month of embryonic life and that in the arch about the end of the second.

At the age of puberty five epiphyseal centers are formed. One epiphyseal plate is above and one below the body, one on the tip of each transverse process and one on the end of the spinous process. Fusion of these centers with the body and the arch takes place about the twenty-fifth year.

The lumbar vertebrae are supplied with blood from four or five pairs of branches from the lumbar artery, and from the vertebrae the blood passes through the tributaries of the lumbar veins and from thence into the inferior vena cava.

In spondylolisthesis the fifth lumbar vertebrae is apparently dislocated forward and downward. In a lateral view of a normal child the anterior line of the lower lumbar vertebrae and the sacrum forms an **S**-curve with the superior convexity pointing forward and downward and the inferior convexity pointing backward and upward. In the condition under discussion the lower portion of the upper arc is greatly accentuated, in some cases it becomes almost an angle with the anterior surface of the fifth lumbar forming the apex.

An examination of the sacrum in these cases will disclose the fact either that the lower convexity of the **S**-curve is pointing more upward, making the superior articular surface lie at a greater angle, or that the anterior portion of the first sacral body is more compressed than normally. In either case the result is the same. The superior surface of the sacrum affords no substantial base for the support of the spine above.

A close examination of the fifth lumbar in these cases will reveal the fact that the body of the bone has slipped forward, while the articular processes of the arch are held in practically the normal position. This means that the neural arch is elongated and becomes oval in shape.

This has been found to be due to a congenital lack of ossification of the neural arch. In this condition each arch results from two ossification neuclei instead of one with a lack of fusion between resulting in a membranous union. This being true it is easily seen how the weight of the body above pressing down on this bone with but little support below, and with the posterior portion of the bone held firmly, causes an elongation of the membranous arch.

Of course, there are other causes for this condition, but, except for cases of trauma, this seems to be the principal one.

The symptoms are more or less indefinite. Objectively the only evidence calling attention to this area is a peculiar "dance step." In the normal, the pace is long and the heel meets the ground first. This results in a jar the effect of which is carried direct to the spine. In the characteristic "dance step" the pace is short and the ball of the foot approaches the ground first, thus easing the body onto the other foot. The result of which is to eliminate the jar which is so distressing.

Subjectively these cases often have pain in the back often severe or they may have a feeling of tension. Formerly spondylolisthesis was thought to be very rare, but now due to X-ray examinations some writers say it is found in about 5 percent of those who come to the roentgenologist.

Treatment has little to offer after the patient has attained his growth. In infancy, however, by means of braces it can in great measure be corrected. Exercise should also be employed to strengthen the muscles along the anterior portion of the spine.

The second of the conditions referred to above may be briefly described as scoliosis. This of course involves a multitude of causes. But the particular condition referred to is believed to be due to a difference in the length of the transverse processes of one side from those on the opposite side.

The curve of the scoliosis is most pronounced in the middle of the lumbar spine, the curve gradually lessening as it approaches the dorsal vertebrae above and the sternum below. This scoliosis is accompanied by a rotation of the bodies of the vertebrae on their axes. This rotation also lessening as it approaches the upper limit of the curve also decreasing below until at the extremities the bones attain their normal position. The rotation is always toward the side of the convexity.

In an examination the roentgenologist finds the scoliosis and the rotation as described above. The shadow of the spines of the dorsal vertebrae will be in the midline. Then as the examiner traces downward, the spines will gradually deviate more and more to the right or left as the case may be until at the center of the curve they will be far over to one side. Then continuing downward they will come more and more toward the midline until they have assumed their normal position. Now if the position of the patient is changed so as to rectify the rotation of the bones and a second film made, in many cases the length of the transverse process of each bone will be found to vary in length from its fellow on the opposite side. That the transverse process of one or more of the bones on the concave side will be longer than those on the convex side. This results in a difference in the muscular leverage of the two sides. On the side hav-

ing the longer transverse processes the muscular pull being greater the processes are brought nearer together and a scoliosis results.

The differentiating point between this condition and a scoliosis due to faulty position on the table is the rotation of the bodies. To prove this, if the examiner will take a film of a normal lumbar spine in a faulty position the film will show the spines of the vertebrae in their normal position with reference to the midline throughout the curve although there is a scoliosis present.

Just what the modus operandi is of this change in development is hard to determine. That it begins in infancy and childhood seems to be certain. It may be that the child being more or less conscious of the extra pull on the one side leans slightly over thus accentuating the curve. Or it may be that the greater pull on one side is alone responsible.

Whatever the cause may be the result is the same—a lack of flexibility of the spine.

The percentage of these cases is also hard to determine because of the indefiniteness of the symptoms. This might be expected as the change from normal is not very radical. In the writer's experience pain on one side of the back is the only symptom. This is not much hardly more than a discomfort, but is much increased after unaccustomed or prolonged exercise, or when the patient is physically subnormal. It is believed this symptom is due to muscular strain.

Here again the treatment is more or less unsatisfactory. Exercise for the development of the muscles on the convex side with the aid of a splint to lessen the rotation of the faulty bodies is all that can be accomplished. It is needless to say that the younger the patient the more perfect the result.

The last condition—retarded development, is best illustrated by describing a case which came to the writer. It was that of a boy 15 years of age whose second teeth were just beginning to erupt. The front upper teeth had not appeared. He was rather small for his age but otherwise seemed perfectly normal. Mentally he was very bright but the absence of the front teeth made him reluctant to meet strangers and engage in the pleasures that normal young people have. Examination of the skull showed the absence of the posterior clinoid process of the sella. The epiphyses of the bones elsewhere were much more separated than they should be at that age. This was especially true of the space between the ilium and the ischium and pubes. An antero-posterior view of the spine revealed nothing very unusual but the lateral view was much more productive of interest. The most noticeable thing was a spondylolisthesis. But above this the bodies of the vertebrae were distinctly underdeveloped. In profile the bodies instead of being slightly concave were convex. And the epiphyseal disks above and below could hardly be seen they

were so rudimentary. They appeared as a very faint dot widely separated from the body. No other changes could be found.

These cases of subnormal development range all the way from a slight retardation to an actual lack of a sufficient number of fingers and toes. In one case that came to the writer's attention a child of 5 years of age was lacking an entire hand with the exception of three carpal bones. Thhe other hand was very rudimentary and by X-ray showed most of the carpal, some of the metacarpal, and two fingers with two phalanges present only. Mentally the child was normal.

It is not intended to class all these cases of lack of development under one heading because it is believed they differ according to cause. We do know that in many instances this extreme lack of development begins in embryonic life. Often it is due to syphilis or it may result from the administration of ergot to bring about abortion, the circulation of the blood of the foetus being interfered with.

These cases are entirely different from those where there is only slight retardation. Here it is probably due to a glandular subnormality. Just why a child should be sent into the world lacking this important element we do not know as yet.

In the case of retardation mentioned above an orthopedic surgeon in consultation advised the application of a cast to the entire trunk. This of course might correct the spondylosisthesis, but it was a question how it would effect the development of the vertebral bodies. Whether the movement and exercise of the spine might hurry the growth or retard it. Of course calcium was ordered, but it is always a question how much of this is changed into bone. In the opinion of the writer it is believed that in all these cases glandular treatment is by far the best. There must be a lack of the secretion of the pituitary body and by its administration this lack can be supplied and the child brought to a normal stage of development.

The point in the discussion of these conditions is really a plea for early and more frequent examination of children by means of the X-ray. Physicians and insurance companies urge their adult clientele to have frequent examinations. Isn't it just as necessary that children whose skeleton is undergoing development should be examined frequently by the roentgenologist to be sure that nature is functioning properly so that the child may grow to healthy manhood and womanhood? Correction of these deformities is so much easier in early life than later when the body has become fully set in its mould.

Success in life depends so much on a healthy body. Socially and economically we are becoming so complex that it is believed the complexity must react on the human body and men who are not at "concert pitch" will go to the wall, try as they may.

PHRENIC EXERESIS [1]

By HARDY V. HUGHENS, Lieutenant Commander, Medical Corps, United States Navy

The phrenic nerve arises chiefly from the fourth cervical nerve with a few filaments from the third and a branch from the fifth, although this branch is occasionally derived from the nerve to the subclavius. The phrenic nerve descends to the root of the neck running obliquely across the front of the scalenus anticus and beneath the sternomastoid, the posterior belly of the omohyoid muscles, and the transversalis colli suprascapular vessels. It next passes over the first part of the subclavian artery, between it and the vein, and as it enters the thorax, across the internal mammary artery near its origin. A branch of this artery accompanies the nerve along its course through the thorax. The nerve supplies the diaphragm by giving off some branches to the thoracic surface, but principally by perforating the muscle and distributing its filament to its undersurface. Each nerve supplies filaments to the pericardium and pleura, and near the thorax is joined by a filament from the sympathetic and, occasionally, by one from the ansa hypoglossi. From the right nerve one or two filaments pass to join in a small ganglion with phrenic branches of the solar plexus, the suprarenal gland and inferior vena cava. From the left nerve filaments pass to join the phrenic plexus of the sympathetic, but without any ganglionic enlargement.

When permanent immobilization of the diaphragm is desired phrenicotomy is unsatisfactory, due to the fact that: 1. Occasionally a branch of the subclavius joins the phrenic below where the nerve is sectioned. 2. On the right there is the ganglion formed from the branches of the phrenic and solar plexus. 3. On the left due to filaments joining the phrenic plexus of the sympathetic. One or all of these conditions may be present. Following a phrenicotomy there may be a temporary paralysis of the hemidiaphragm, but later its function is resumed.

Phrenicoexeresis was first suggested by Stuertz in 1911 and somewhat later advocated by Schepelmann. It was developed, however, by Sauerbruch and his followers, who found that a more radical removal of the nerve was necessary in order to obtain a lasting paralysis of the diaphragm. The operation, as generally advocated today, consists of removing as much of the nerve as possible, at least 10 to 15 cm. By exercising care in exposing and withdrawing the nerve, its filamentous attachment to the diaphragm gives way and the portion distal to the point of severance is avulsed.

[1] Read at staff conference at Norfolk Naval Hospital, Portsmouth, Va., Nov. 11, 1935.

Preliminary considerations.—The procedure in performing phrenic exeresis is simple. However there is ample opportunity for encountering trouble in the area involved in the operation. Branches of the large arteries of the neck may easily be injured, especially if the position of a vessel vary from the normal, and dangerous hemorrhage may occur. Sauerbruch up to 1930 reported three such accidents occurring during the performance of a simple phrenicotomy. Two of the patients died, the third was saved by the ligation of the subclavian. During the evulsion of the nerve it is possible to rupture one of the superior diaphragmatic vessels allowing blood to enter the mediastinum. Injuries to veins have been given as cause of embolism. The pioneer Sauerbruch lost three patients from the latter cause. There is a lot of dynamite in the lateral triangle of the neck and anything in it may be damaged. The vagus has been cut. As phrenic exeresis is now performed the mortality is practically nil and should not be considered when the procedure is indicated. Nehil and Alexander of Ann Arbor reported 654 operations without causing, or in any way contributing to, a death. H. R. Decker reported 222 phrenic nerve operations done at the Presbyterian Hospital, Pittsburg. He says that in no case was the interruption followed by an unfavorable course so closely that it could be ascribed to the operation. He further comments:

> The operation itself is relatively simple, but requires exact technique. It is performed in an anatomic field fraught with sufficient potential danger to demand the skilled and experienced rather than the occasional operator.

During 1933 there were 102 phrenic exeresis performed at Fitzsimons General Hospital, Denver, Colo. In his annual report the chief of the enlisted men's tuberculosis service, Maj. W. C. Pollock, said:

> We have never seen any complication following operation of such moment as to cause any hesitation in recommending the procedure.

The most common symptoms complained of by the patient following phrenic exeresis are digestive disturbances and cardiac flutter. These symptoms are caused by shifting of the mediastinum and by the interference of nerve fibers from the phrenic to the pericardium and disturbance of the solar and sympathetic plexuses. These symptoms usually disappear after a short time.

Curti is credited with performing in 1924 the first bilateral phrenic exeresis. Since that time numerous workers have performed the operation. H. Schwatt, medical director of the sanitarium of the Jewish Consumption Relief Society at Spivac, Colo., reported one case in which he did a bilateral exeresis on a woman suffering with advanced pulmonary tuberculosis with questionable benefit to the patient. One bilateral phrenic exeresis was done at Fitzsimons Gen-

eral Hospital in 1933. Up to August 1933, 14 other workers each reported one instance of performing the operation for uncontrollable hiccough complicating far advanced pulmonary tuberculosis, and Brunner had recourse to blocking of both nerves with cocaine for the same symptom occurring after thorocoplasty. In 1934 Campbell at Fitzsimons performed exeresis on one side and blocked the other with alcohol in the case of a marine with severe hiccough. I observed this case and know that the hiccough was stopped and the tuberculous lesions were improved by the procedure. The hiccough in this case was precipitated by unilateral intrapleural pneumolysis. Bilateral phrenic exeresis is a heroic procedure and is only resorted to in desperate cases.

Preparation.—Usual preparation of the operation field. Patient is placed on his back with a sandbag or roll of cotton or gauze under his neck.

Anesthesia.—Infiltration method using 1 percent novocane.

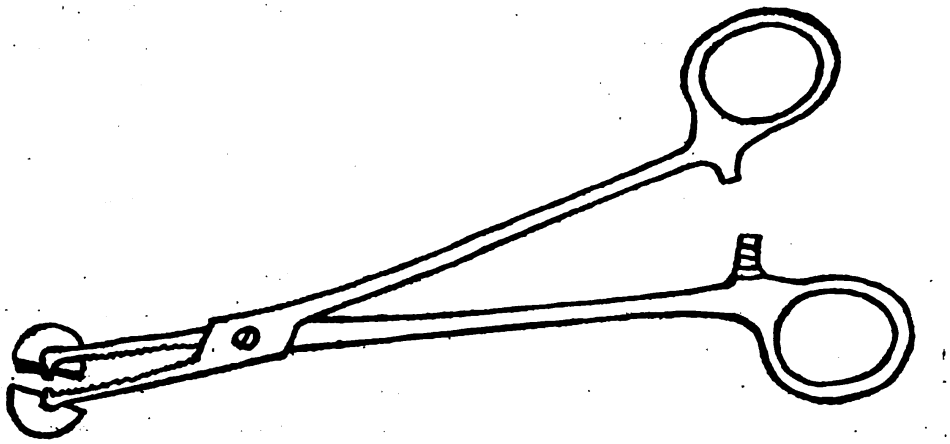
Technique.—A transverse incision 5 to 7 cm in length is made approximately 5 cm above the clavicle. The anterior end of the incision is just over the posterior border of the sternomastoid. The subcutaneous fat is bluntly dissected and the platysma is opened vertically about the center of the incision or slightly away from the posterior border of the sternomastoid. The muscle is gently retracted and by blunt dissection the fatty layer is separated down to the thin facia over the scalenus anticus. The facia is opened in the same manner. Normally the phrenic nerve is found to run diagonally across the lower third of the scalenus anticus. Occasionally it passes through the muscle. Rarely it passes into the thorax with the subclavius and may not be seen in the operation field. Upon finding the nerve it is separated from its attachment to the muscle and freed down well under the structure toward its entrance to the thorax. Some authorities advise against pinching the nerve for purpose of identification, stating that it should be recognized by its course. However, I have always seen it pinched, gently, and I do the same and am gratified when the patient complains of pain in the shoulder. The nerve, having been identified, it is picked up with an artery forcep and quickly cut. It may be injected with novocane, but the injection gives as much pain as clamping and cutting. The nerve may then be twisted out by use of the forcep. Shown here is an Oschner forcep with a wheel three-fourth inch in diameter on the end. I copied this form the one Maj. Dan Campbell had made at Fitzsimons General Hospital, Denver.

In using this instrument there is no danger of the nerve slipping over the end and consequently causing it to be pulled in two before a sufficient amount is avulsed. The twisting is done gently as the

patient is encouraged to breath a little more deeply than normally, until the nerve is felt to give away at its diaphragmatic attachment. This is the perfect exeresis. It sometimes breaks short of the desired minimum 10 or 12 cm. If the break is distal to the ganglion permanent paralysis of the hemidiaphragm will be obtained.

Indications for phrenic exeresis are fairly well limited to the field of treatment of pulmonary tuberculosis, the procedure being one of the important steps in pulmonary collapse therapy. Its indications in pulmonary tuberculosis:

1. Instances in which it is not possible to establish artificial pneumothorax, (a) due to the obliteration of the intrapleural space; (b) at points where artificial pneumothorax cannot be established for a considerable time after diagnosis has been made, due to the lack of equipment; (c) where exeresis is demanded by the patient instead of pneumothorax.



Parenic avulsion forceps.

2. In pulmonary hemorrhage when it is not possible to induce pneumothorax or check the hemorrhage by other means.

3. As an adjunct to artificial pneumothorax therapy; (a) where apical adhesions have prevented closure. Frequently paralysis of the diaphragm on the affected side will relax these adhesions and rapid closure of cavitation follows; (b) in central involvement, paralysis of the diaphragm often serves as the needed splint for completing the closure of the cavity.

4. To diminish cough and at the same time cause sputum to be raised easier, according to Major Pollock. While Decker says:

> Its value as a public-health measure in reducing sputum and incidence of bacilli cannot be overestimated.

My impression is that the reduction of sputum is due to gradual cavity closing.

5. Preexpansion procedure in pneumothoraces. This is desirable because the lung, having been compressed over a long period of time,

should not be required to fill the space that the normal lung once filled. Paralysis of the hemidiaphragm reduces the pleural space as much as one-third.

6. Prior to thorocoplasty. This is desirable in certain instances but should not be done, for instance, when the thorocoplasty is done primarily for closure of upper lobe cavitation. It is performed preceding upper phase thorocoplasty where cavitation involves middle lobe or extends into the lower lobe, and when lower phase thorocoplasty or unroofing is done.

The only other instances in which phrenic exeresis is justified is in lung abscess and bronchiecstasis. It is only resorted to in these instances when induction of artificial pneumothorax is not possible.

THE NODAL TRIANGLE

By R. A. Nolan, Commander, Medical Corps, United States Navy

The Nodal triangle, an abnormal finding noted in the study of influenza lymphatica[1], or so-called glandular fever, is easily elicited by simple, gentle, digital palpation, palpating fingers running along vessel's course on surface (fig. 2).

The condition when elicited is definite. Sacculated nodes ranging from the size of a pea to a bean are in linear arrangement converging at the inner aspect of the knee joint, but never below this point. These lymphatic dilations or sacculations take on temporarily the nature of the lymphangioma or fibroma. They lie in the subcutaneous tissue just below the corium; the skin is normal in appearance and shows no local signs of inflammation over nodal triangular lines.

By palpating over these sacculations with pressure, pain is elicited along the nodal chain.

The number counted along the great saphenous vein in one male adult was 54; the number counted along accessory saphenous was 32. In a child, aged 2, a total of 42 nodes made up the triangle.

This sign appears in the thigh within 1 week following initial introductory symptoms and coordinates with the white cell anarchy, the symptomatology, the coryza and conjunctivitis, and the presence of acetone in a large percentage of urines.

Results.—The results obtained by phrenic exeresis in pulmonary tuberculosis, lung abscess, and bronchiecstasis are due wholly to pulmonary immobilization. In hiccough results are due to paralysis of the diaphragm. All kinds of percentages of beneficial results and cures are given by various workers. It is obvious that no two workers' reports can be compared since the type of cases in which the

[1] Influenza lymphatica, author's caption.

procedure is used is a matter of personal judgment. It is certain that when care is exercised in selecting the cases in which to use the procedure, the productioin of diaphragmatic paralysis is of great value in pulmonary compression. It is fairly well established that there is a rise from 1 to 7 cm following evulsion of the nerve. The maximum rise may be reached any time from 1 week after operation. In his series of 170 cases studied for periods up to 3 years, Schwatt states that, "it appears that elevation of the diaphragm bears no consistently direct relation to the time elapsed after operation."

The writer's small series of 11 cases of phrenic exeresis were performed as an adjunct to collapse thereapy in pulmonary tuberculosis. There was nothing unusual in the type of cases. The average rise in the hemidiaphragm followed avulsion of the nerve and no complications occurred.

This sign with symptoms will persist and mask the true diagnosis if rest in bed is not instituted and the various types of maltreatment avoided. (See reference 1 and 2.)

With this sign, and using information obtained from blood counts, the Bunnell agglutination test, the clinical evidence noted in 3,000 or more cases (see reference 2), and daily consultations with brother medical officers, permit me to use my imagination in attempting to describe the reason for the pathologic formation of this so-called nodal triangle.

Lymphatic vessels may become abnormal due to mechanical injury or obstruction. It is also caused by bacterialogic factors, for instance, in elephantoid fever, which finally results after many attacks in true elephantiasis; also the reaction of lymph vessels (lymphangitis) is daily seen in dispensaries due to local infections. There is also evidence to suppose that lymph vessel walls may lose their tone and dilate, causing an interference with the osmosis that exists between the venous blood and the lymph system. This factor may account for the muscular asthenia especially noted in the lower limbs of those affected with this condition. It is further thought the return flow of lymph from the lower extremities to the torso would be impeded, causing further dilatation. This dilatation of lymphatic vessels, with lymph stasis, resulting in increased intervalvular, intravascular pressure, can produce the nodal, sacculated, cord-like, doughy feel, which go to make up the diagnostic sign. (Such a condition is aptly described in Stelwagon's Diseases of the Skin, in his chapter on lymphangioma.)

It is my feeling that the organism or virus causing influenza is alone responsible for producing the picture of influenza lymphatica, and heretofore known as glandular fever epidemicus. It is not the writer's intention to convey the thought that this condition is new.

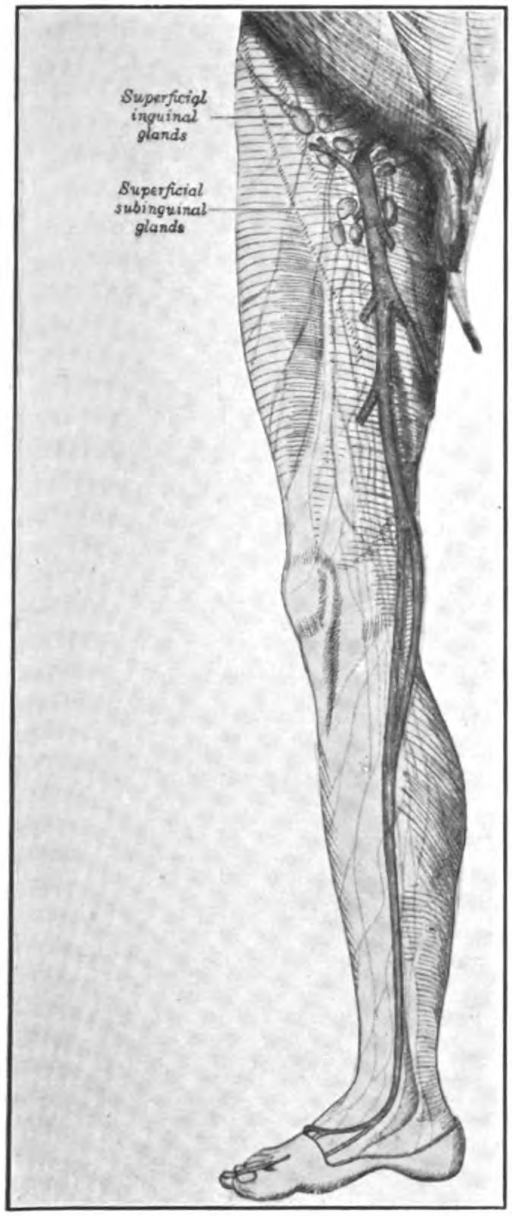


FIGURE 1.—NORMAL SUPERFICIAL LYMPH ANGIOLOGY OF LOWER EXTREMITY TAKEN FROM GRAY'S ANATOMY.

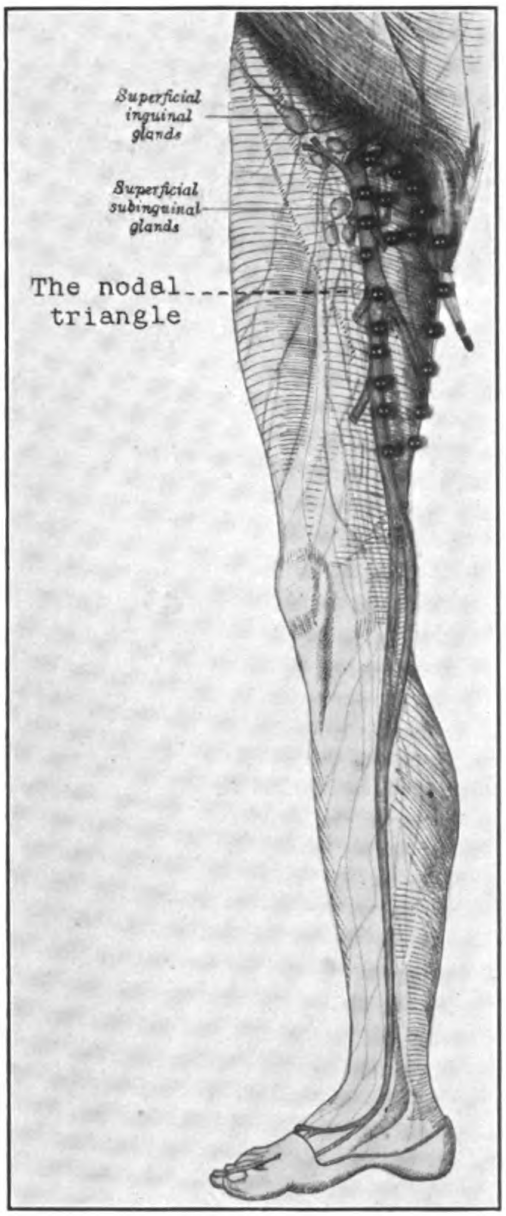


FIGURE 2.—SAME PICTURE SHOWING DEFINITE PALPABLE PATHOLOGY ALONG THE COURSE OF THE GREATER AND ACCESSORY SAPHENOUS VEINS OF THE THIGH.

Note triangulation in diagram.

It is as old as influenza, and if Osler classified four types of influenza, namely, the respiratory type, the gastrointestinal type, the rheumatoid type, and the nervous type, with the evidence that has been gathered during the past epidemic in juveniles and adults, it is permissible to add to this classification, affecting primarily the lymphoid system and the tissues of the body. I would further state that this type may mask the diagnostic picture of any one of the aforementioned. In other words, one or more types may be present in a given case.

This sign is considered of extreme importance because of the following reasons:

1. For purposes of early differentiation.
2. A possible explanation for muscular asthenia.
3. As another check in establishing the fact that the organism or virus, causing influenza in some juvenile epidemics, has a specific action on the lymphatic system of the body, thereby producing a train of signs and symptoms.
4. When the patient shows by the blood count, etc., an atypical picture of so-called glandular fever, and the cervical, axillary, and inguinal adenopathy is apparently normal, this sign in the lower extremities will furnish the evidence demonstrating adenomatosis in an anatomical location rarely if ever looked for.
5. Its presence is apparently due to a mechanical irritation (muscle squeeze) during the prodromal or acute introductory stage of the influenza virus affecting the lymphatic system.

The following examples of daily blood picture

No. 0.—Note drop from high to low in 6 days and presence of lympho-blasts in female child age 6 years

	Segs	Bands.	S. lymphs	L. lymphs	Mono.	Eos.	Lympho B.	Turk.	Baso.	Total
1	70	23	5		1	1				28, 200
2	24	40	22	7	2	5	7			17, 250
3	28	33	25	4	2	6		2		12, 200
4	41	8	45		4	2				6, 800
5	23	37	21	15	2		1		1	4, 600

No. 00.—Note introductory leukopenia with count low in Pharmacist Mate R. J. P., age 25 years

1	60	17	19						3	4, 400
2	49	13	20	10	4	2		2		3, 500
3	48	9	19	10	5	6		3		5, 400
4	34	7	28	11	6	4	4	6		3, 800
5	35	3	41	14	6	1		1		4, 600
6	43	5	39	8	4	1				5, 700
7	53	3	32	3	6	2			3	5, 600
8	42	2	44	6	5	1				4, 500
9	51	2	31	10	3	3				4, 800
10	53	3	33	1	7	3				4, 200
11	45	3	43	1	6	1		1	1	4, 450
12	51	13	26	1	7	1			1	8, 200
13	57	5	28	3	5	2				6, 200
14	53	3	32	3	6	2			1	5, 600
15	47	4	40		7	2				4, 200

The following examples of daily blood picture—Continued

No. 000.—Male child age 8 years—Juvenile

	Segs	Bands	S. lymphs	L lymphs	Mono.	Eos.	Lympho B.	Turk.	Baso.	Total
1	7		90		2	1				4,000
2	61	17	15		5				2	28,000
3	50	31	7	1 4	5	2				29,500
4	28	58	5	3 1	2			1		36,800
5	42	21	25	2	8	1		1		21,000
6	49	18	22	3	6	1		1		13,200
7	51	18	19	2	5	2			2	15,800

No. 0000.—Lieutenant, Navy

1	35	5	40	5	12	2			1	10,800
2	62	8	20	1	8				1	14,200
3	51		37	2	5	5				8,200
4	53	5	27		10	5				8,700
5	40	7	30	4	15	4				11,200
6	35	8	33	5	13	5	1			6,400

No. 00000.—Seaman, first-class

1	19	19	53		8				1	5,400
2	21	14	59		6	1				6,750
3	19	14	59		7	1				4,750
4	21	7	66		6					6,400
5	9	21	65		5					8,600
6	6	11	72		10					8,400
7	15	24	52		4		4	1		8,400
8	8	14	61		6	1	7	3		8,000
9	8	8	75		3		4	4		10,400
10	4	5	80		5	1	4	1		10,600
11	2	7	81		3	1	4		2	
12	10	12	72		1		5			12,200
13	14	13	67		2		3	1		10,250
14	18	13	38	26	2		2	1		18,200
15	18	15	36	22	4		4	1		17,800
16	43	5	46		6					9,400
17	45	7	37		11					10,300
18	48	2	39		11					7,000
19	41	5	43		7	3			1	5,800
20	17	4	71		6				2	6,100
21	26	4	60		8	2				5,400
22	38	6	51		3	2				8,300
23	38	2	52		6				2	6,350
24	41	4	50		5					6,900
25	32	8	52		2	2			2	6,500
26	37	5	52		4	1			1	7,600

No. 000000.—Aerog., first-class

1	23		74		3					15,250
2	15	14	67		3		1			10,450
3	27	8	53		12					15,400
4	30	12	55		2				1	10,600
5	15	3	73		1		6		2	14,400
6	23	15	60		2					12,400
7	25	12	53		6	1	3			13,600
8	16	10	67		3		3		1	10,400
9	23	10	63		2	1	1			10,850
10	46	6	34	5	5	3			1	10,000

No. 0000000.—Jan. 2, 7, and 10—Mar. 30, 1935

1	87	3	10							16,200
2	80		17		3					8,600
3	42	6	51		1					7,400
4	45	10	38		4	2			1	7,200

No. 00000000.—Seaman, first-class, United States Navy

1	60	8	22		8	3	2-12-35			10,200
2	49	19	8	8	14	1	3- 3-35			12,200
3	59	7	21	1	10	2	3-22-35			7,200

REFERENCES

(1) Early report of so-called glandular fever epidemic in the Naval Medical Bulletin of October 1935. Nolan.
(2) Paper entitled "The Pentatype of Influenza", namely influenza lymphatica, presented at the sixth annual meeting of the western branch, American Public Health Association at Helena, Mont., June 2, 1935. Nolan.
(3) Stellwagon's Diseases of the Skin.
(4) Graphs from Gray's Anatomy.

PHENYL MERCURIC NITRATE IN THE TREATMENT OF OTITIS EXTERNA AND OF THE DERMATOPHYTOSES[1]

By FREDERICK C. GREAVES, Lieutenant, Medical Corps, United States Navy

Otitis externa and the fungus diseases of the skin are two relatively minor disease entities, but they often tax the ingenuity of the medical officer and are responsible for a considerable number of sick days among naval personnel. In spite of the fact that standard treatments exist for these conditions and are discussed in any textbook that treats of these subjects, their routine use is often attended with indifferent success and leaves much to be desired. The opportunity to test the value of phenyl mercuric nitrate in the treatment of these conditions presented itself and the results of those tests are the basis of this report.

Phenyl mercuric nitrate has been known since 1870, but its value in the treatment of disease was not investigated until recently. Weed studied its biological properties at Iowa State College and published his results in a thesis in 1932 (1). He continued his studies at Western Reserve University with Ecker and at various times these men have made additional reports upon its bacteriological and mycological aspects, as well as its toxicity for man and animals (2). Birkhaug (3), investigating its bactericidal powers, determined the following phenol coefficients: Against staphlococcus aureus, 2,259; against streptococcus hemolyticus, 1,440; against pneumococcus, 1,067; against gonococcus, 889; against B. subtilis, 801; and against B. coli, 640. Upon the basis of these results he concluded that phenyl mercuric nitrate surpassed other disinfectants in the following order: Metaphen, 1.4 times; merthiolate, 1.6 times; mercuric chloride, 5.7 times; hexylresorcinol, 9.6 times; mercurochrome, 434 times; and phenol, 1,202 times. Weed and Ecker (2b) found that aqueous solutions, in dilutions as high as 1–125,000, inhibited the growth of fungi with the exception of Anchorion violaceum, which was more resistant.

[1] Presented at the Staff Meeting, U. S. Naval Hospital, Pearl Harbor, T. H., Jan. 17, 1936.

The clinical value of the compound was reported upon by Biskind (4), who used it in treating 70 cases of cervical and vaginal infection. These cases were consecutive and included specific and nonspecific infections and cases resulting from the presence of Trichomonas vaginalis. Treatment consisted of instillations into the cervical canal, tamponade of the vagina and daily douches. An aqueous solution in the dilution of 1–1,250 was used for the cervical instillations and the vaginal tampons and a 1–25,000 solution for the douches. He reported favorable results in all cases except those in which T. vaginalis was the exciting cause and that no ill results or irritating effects were encountered.

Levine (5) reported his results in 205 cases of fungus and yeast infections of the skin. These cases consisted of tineas of the feet, scalp, face, tinea versicolor, tinea circinata and interdigital saccharomycoses. He concluded that Phenyl mercuric nitrate was highly efficacious in the treatment of these conditions, producing cures where other forms of treatment had failed.

The toxicity of the compound was studied by Birkhaug, who found that mice maintained on an aqueous solution of phenyl mercuric nitrate in a 1–2,000 dilution, as the only source of water supply for a period of 10 weeks, showed no ill effects. Weed and Ecker (2c) determined the minimal lethal dose for rabbits, when injected intravenously, to be 1 cc of an aqueous 1–1,250 solution, per 100 grams of body weight, and that the lesions produced were those seen in acute mercurial poisoning.

The council on pharmacy and chemistry of the American Medical Association published a report (6) in which it was stated that phenyl mercuric nitrate was a powerful bactericide and fungicide of low toxicity and that further clinical evidence, confirmation, and the test of time were needed before its value could be accepted as conclusive.

The cases comprising this report in the treatment of which the compound was used consist of 75 cases of otitis externa and 100 cases of fungus infections of the skin. The etiological agent in the otitis externas is doubtful. Bacteriological studies, consisting of smear, hanging drop and culture, failed to show the presence of pathogenic fungi in any case. A somewhat unexpected bacterial flora was found. Staphlococci and streptococci were extremely rare, but all cases showed the presence of a gram negative, motile, pigment producing bacillus of the pseudomonas, or pyocyaneous, group. In culture media, this organism produces an unpleasant, musty odor that is similar to the odor observed in the discharges from the infected ears. It is probable that this organism is an accidental secondary invader in these cases but it seems to be instrumental in prolonging the symptoms and discharge. Apparently, it produces a toxin that sets up an eczematous dermatitis about the orifice of the external auditory canal.

Patients with an eczematous dermatitis complicating an otitis externa have been found, in some cases, to have a marked skin sensitivity to the endogenous protein of this organism, while healthy controls, tested simultaneously, have failed to react to a similar extent.

The history that is frequently obtained from these patients is that they have noticed itching within the canal and that they have attempted to gain relief by scratching with the fingernail or some handy object. Abrasions and excoriations of the canal wall are easily produced and the result is infection, with intense pain and swelling as the outstanding symptoms. In the more severe cases, pain prevents sleep, hearing is interfered with because of the swelling within the canal and the patient is incapacitated for duty. At first there is no discharge but later there is a variable amount of foul, green pus in which there are shreds and flakes of the epithelium lining the canal.

For the treatment of both otitis externa and dermatophytosis a solution containing phenyl mercuric nitrate in a strength of 1–1,250 in 95 percent alcohol was used. One percent castor oil may be added to this solution to prevent excessive dehydration of the tissues. In the acute otitis externa cases, the canal is gently swabbed with the solution or, if the amount of swelling will allow, a small cotton tampon, soaked in the solution, is inserted in the canal and left in place for 30 minutes. This treatment is repeated at daily intervals for 3 or 4 days, or until the process begins to subside. When this occurs discharge usually appears and treatment at this stage consists of gently blotting up the discharge with cotton applicators and the blowing into the canal of a dusting powder containing powdered phenyl mercuric nitrate of 1–1,500 strength in a base of four parts of powdered boric acid to one each of zinc oxide and zinc stearate. Those cases presenting themselves after the acute symptoms have subsided, or in which there is a purulent discharge with little or no swelling, are treated by applying the solution once or twice, subsequent treatment consisting of blotting out the discharge and use of the dusting powder.

The group of fungus diseases of the skin treated with phenyl mercuric nitrate contained 1 case of ringworm of the scalp, 1 of ringworm of the face, 3 of tinea versicolor, 4 of tinet cruris, and 91 of epidermophytosis of the feet. Many of these latter cases were discovered accidently during a physical examination for other conditions and treated as a routine measure, but the majority were under medical supervision primarily because of the epidermophytosis.

The fungus epidermophyton as a rule localizes in the interdigital tissues of the feet, where its presence is manifested by lesions varying from a dry scaliness, with a tendency to cracking and fissure formation, to a mass of white, moist, sodden epithelium. There is

a marked tendency to the production of hypersensitiveness of the skin at a distance from the original lesions, particularly on the feet and legs and on the hands and about the wrists. Secondary eczematous lesions commonly occur in these areas. These secondary lesions, which are known as epidermophytids, rarely, if ever, contain the exciting fungus itself, but are the reactions of a hypersensitive tissue which is rebelling against irritating agents that ordinarily would produce no effects in a healthy skin. Epidermophytids are areas of dermatitis, frequently ecxematous, that become secondarily infected with pyogenic organisms. White and Taub (7) and Strickler, Ozellers and Zaletel (8) emphasized the importance of considering the lesions on the feet as local foci from which sensitizing agents are elaborated. These sensitizing agents may be the fungus itself or its metabolic products.

Two separate conditions must be treated in epidermophytosis with secondary lesions. The foci of fungus infection must be eradicated and the epidermophytids must be treated with as little irritation as possible. Applications of the 1–1,250 alcoholic solution control the former, rapidly and effectively, but the epidermophytids are more resistant. First of all, the secondary pyogenic infection, that seems to be present in every case, requires the institution of evacuation and drainage of collections of pus and the applications of boric acid solution dressings. Following this, the blandest of lotions or cold cream are used to prevent the sticking of dressings to raw surfaces, and dry sterile dressings are substituted as early as possible, since a hypersensitive skin rapidly becomes intolerant to chemical irritation.

The treatment of the cases of simple epidermophytosis without secondaries and the treatment of the ringworms, the cases of tinea cruris and tinea versicolor consisted of the application of the solution to the lesions twice daily. Response to treatment was prompt and uneventful in every case.

The results of the use of the phenyl mercuric nitrate solution in the very acute otitis externas, where there is considerable pain and swelling, are almost spectacular. Immediately the solution comes in contact with the inflamed, swollen, excoriated canal wall there is intense burning pain, due to the high alcoholic content of the solution. This pain rarely lasts longer than 2 minutes, and with its disappearance all pain in the affected ear ceases. The patient immediately becomes comfortable and, in many cases, obtains the first sleep he has had since the onset of the acute symptoms. The use of the compound in the less acute cases and in those of long standing has produced equally satisfactory results, shortening the course of the condition to a matter of days after the treatment is instituted.

One case of chronic bilateral otitis externa that had been present for 15 months and had resisted all treatment, responded and at the end of 2 weeks presented normal canals with wax formation. In none of the cases treated has any irritating effect been observed in the tissues of the canal or of the tympanic membrane.

Its use in treatment of the various fungus diseases of the skin also has proved highly satisfactory. It promptly and completely eliminates fungi from the lesions so that none can be found after the first two or three applications and it caused no irritation or soreness in any of the cases in which it was used.

The phenyl mercuric nitrate used in these cases is manufactured by the Eastman Kodak Co., and occurs as a silky crystalline substance that is sparingly soluble in water, alcohol and glycerine and apparently insoluble in all other solvents. One part of the compound dissolves in approximately 1,250 parts of each of the solvents mentioned. At room temperature, however, solution takes several days for completion but the addition of heat speeds up the process. The solution described above is made as follows: 0.08 grams of phenyl mercuric nitrate are added to 100 cc of 95 percent ethyl alcohol in an Erlenmeyer flask and slowly heated and agitated until the compound dissolves. It is not necessary to boil the alcohol. After the compound has dissolved, 1 cc of castor oil is dissolved in the solution and enough methylene blue is added to give it a distinct blue tint, after which it is filtered and is ready for use.

The use of an ointment has not been very successful. Trying to make an ointment by incorporating the solution in a suitable base and having a satisfactory concentration of the compound in the finished product has proved unsatisfactory and in view of the slight solubility of the compound it would appear irrational to incorporate the solid directly into the ointment base.

Taking advantage of the solubility of the compound in glycerine a 1–1,250 solution in that substance can be made and a glycerite of starch can be produced having a final concentration of phenyl mercuric nitrate of 1–1,500. This substance has the appearance and consistency of K–Y jelly and, while it possesses fungicidal properties, it has no advantages to recommend its use in preference to the alcoholic solution.

CONCLUSIONS

1. Phenyl mercuric nitrate was used in 75 cases of otitis externa and was found to eliminate the pain and discomfort in the acute cases and to shorten the course and hasten recovery in the chronic and long standing cases.

2. Its use in 100 cases of dermatophytosis indicates that it is very efficient in rapidly and effectively eliminating fungi from the lesions.

3. Its use in both otitis externa and dermatophytosis was not attended by ill or irritating effects.

4. From the above conclusions it is believed that its further trial and use are indicated.

REFERENCES

(1) Weed, L. A.: The Biological Properties of Certain Organic-Mercury-Salts, thesis, Department of Pathology, Western Reserve University, May 15, 1932.

(2) Weed, L. A., and Ecker, E. E.: (*a*) The Utility of Phenyl Mercuric Nitrate as a Disinfectant, J. Infect. Dis. 49: 440–449 (Nov.) 1931; (*b*) Bacterial Action of Phenyl Mercuric Nitrate, ibid. 51: 309–314 (Sept.–Oct.) 1932; (*c*) Phenyl Mercuric Compounds—Their action on Animals and their Preservative Values, ibid. 52: 354–363 (May–June) 1933.

(3) Birkhaug, K. E.: Phenyl Mercuric Nitrate, J. Infect. Dis. 53: 250–261 (Sept.–Oct.) 1933.

(4) Biskind, L. H.: Phenyl Mercuric Nitrate: Its Clinical Uses in Gynecology: A preliminary report, Surg., Gynec. & Obst. 57: 261–264 (Aug.) 1933.

(5) Levine, Benj.: Use of Phenyl Mercuric Nitrate in Tinea and Yeast Infections of the Skin, J. A. M. A. 101: 2109 (Dec. 30) 1933.

(6) Council on Pharmacy and Chemistry: Phenyl-mercuric Nitrate and Phenyl-mercuric Chloride, J. A. M. A. 102: 1224 (April 14) 1934.

(7) White, Cleveland, and Taub, S. J.: Sensitization Dermatoses of Nonfungous Nature Following Superficial Infections ("Ringworm") of Extremities. J. A. M. A. 98: 524 (Feb. 13) 1933.

(8) Strickler, A., Ozellers, E. A., and Zaletel, R. P.: Modern Interpretation of Mycotic Infections of the Feet and Hands, Arch. Dermat. & Syph. 25: 1028 (June) 1932.

DANGERS OF PROSTHESIS FOR AVIATION PERSONNEL

By James Lee Brown, Commander, Dental Corps, United States Navy

As a part of the annual physical examinations of officers at the United States Naval Air Station, San Diego, Calif., the dental clinic examined 405 officers. The greater number of these officers are engaged in actual flying. Of this number 345 needed dental attention of some character and 60 required none. Obviously the admission rate is high. Many of these officers were attached to Fleet air personnel and naval air station personnel and a small percentage, of course, are not engaged in actual flying.

One outstanding fact was learned, and it is believed worthy of emphasis. Among the aviators engaged in actual flying it was found that quite a number were wearing removable prosthetic appliances. Among this personnel it was found that few, if any, had been cautioned regarding the removal of these appliances while they were in the air. It would seem timely to invite attention to the hazard involved in wearing such appliances while actually engaged in aviation operations. While this writer has contacted no actual injuries result-

ing from this hazard, it is easy to understand wherein the hazard exists.

An accident, such as a crash, due to a forced or other landing, or in taking off, may or may not be of such a severe character as to cause severe injury or death, or it may be only sufficiently severe to cause considerable shaking up, hard bumps, and minor injuries to the head. An intense blow upon the maxilla or mandible or any part of the head could easily dislodge, fracture, or otherwise injure the entire appliance or parts thereof in such a manner as to force them into the surrounding tissues, the pharynx or trachea, thus producing such a serious condition that even death might ensue before the individual could be properly brought under the care of a surgeon. Rough or bumpy air or other hazards incident to flying might easily be so severe as to cause dislodging or fracture of such appliances which, if lodged in the throat or bronchial tubes, might easily interfere with the control of the ship. It becomes a plausible consideration to foresee that even parts of such appliances may find their way into the bronchial tubes and lungs, thus causing other serious complications.

In view of these considerations it is believed timely to recommend, as a medico-military precaution, that personnel engaged in this and other hazardous occupations be cautioned to remove such appliances while so engaged. It becomes a simple matter to provide one's self with a small, suitable, metal box fitted with some cotton or other material in which they may place the appliance and carry it in their pocket while flying. If it is broken therein, it at least obviates the physical hazards mentioned above.

Fixed prosthesis, particularly the large bridges carrying porcelain pontics, also constitute a hazard almost as grave as the removable appliance. This type of prosthesis has the disadvantage that it cannot be removed by the patient. It therefore becomes questionable in this writer's opinion if extensive fixed prosthetic appliances should be fabricated for the aviation personnel.

EAR SYMPTOMS INCIDENTAL TO SUDDEN ALTITUDE CHANGES, AND THE FACTOR OF OVERCLOSURE OF THE MANDIBLE—PRELIMINARY REPORT

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Every change in our mode of living has brought new symptoms to the medical and dental professions. With the advent of the airplane the Army and Navy found it necessary to establish a school of aviation medicine. Primarily these physicians were to examine applicants who wished to join the flying service of our military forces. The work of this group has steadily grown as the number

of people flying has increased. Today they recognize symptoms related to flying which were given no consideration a few years ago.

Selecting six cases showing malocclusion of the jaw, from a large number of pilots a striking coincidence of ear pain and dizziness was constantly observed with sudden loss of altitude. Each of the six cases was entirely relieved of the symptoms after repositioning of the mandible, thereby increasing the vertical dimension of the jaw, and presumably relieving compression of the Eustachian tubes. No further difficulty has been noted after 3 years.

The following case history is typical of the group and was the first one observed: In April 1933, an airplane pilot, whose teeth were badly worn down and eroded, came in for dental inspection. A survey was made and it was suggested that his teeth be built up to balance the occlusion. By change of the application of force and correction of the wide overclosure we felt it possible to stop the erosion. No mention was made of ear symptoms, and no check was taken of the hearing.

On May 11, 16 gold shoes were placed in his mouth. This increased the vertical dimension of the jaws three-eighths of an inch. That night on his flight out of Kansas City he found it necessary, for the first time in 5 years, to use cotton in his ears. A temporary deafness which he always had for a few hours after landing was absent, and a slight dizziness which he usually experienced when rapidly losing altitude was not felt at any time during the trip.

It had been generally assumed that "flying deafness" is the result of the roar of the motor in the same manner that boilermakers become deaf from the intense vibrations of hammering. That some of the flying deafness cases are of intermittent and temporary nature, and are the result of Eustachian tube compression seems quite clear from our observation of this selected group of cases. The simple physical factor of production of a partial vacuum about the flier in loss of altitude contributes something to the collapse of his Eustachian tubes. Overclosure of the jaws from varieties of malocclusion has been proven to be another most important factor in compression of the tubes in this group, as shown by the improvement when the correction was made.

In further investigating ear symptoms of pilots we found some who had severe pain in the ears when losing altitude. This also was caused by a lack of adjustment of pressure within the middle ear as the atmospheric pressure increased on the outside. Under certain conditions this unequalization of pressure produces dizziness. The fact vertigo has been seldom reported may be answered by the fact that if mild the pilot does not mention it and if severe he crashes. That vertigo we believe to be the cause of many of our unexplained military accidents.

Recently the effect of malocclusion on eustachian tube function has been proven highly important in cases of functional deafness, dizziness, and pain symptoms about the head. Dr. James Costen, of Washington University, reports many cases in this classification. In a paper entitled, "A Syndrome of Ear and Sinus Symptoms Dependent Upon Disturbed Function of the Temporomandibular Joint", he says, "The problem of temporomandibular-joint function and occlusion being a major dental issue, it appears almost entirely in the dental literature." The following group of symptoms may be observed frequently in patients with endentulous mouths and a marked overbite. The syndrome is classic for lesions of the sinuses or ears, yet overbite disturbances of the joint are so easily overlooked as etiological factors that it becomes a source of error in analyzing cases in otolaryngology. Conditions that have been given most attention in medical literature are anterior dislocations, fracture of the neck of the mandible, and ankylosis of the joint after chronic irritation or infection. These have received prolific comment, and appropriate surgical treatment has been carefully worked out. Each of these symptoms may be ascribed to some evident disturbance in anatomic function of the joint, its ligaments and muscular attachments. The ear symptoms depend upon actual involvement of the eustachian tube and tympanic structures. The sinus symptoms are more apparent than real. The actual source of this group of complaints was confirmed by the marked improvement which followed correction of the overbite, renewal of molar support to take pressure off the condyle, and establishment of proper articulation of the condyle within the fossa.

The ear symptoms observed were—

Impaired hearing, continuously, or with intervals of improvement.

Stopping or stuffy sensation in ears, marked about mealtime.

Tinnitus, usually low buzz in type; less often a snapping noise while chewing.

Pain, dull type, within and about ears.

Dizziness, mild; again, attacks of prostrating severity, definitely relieved by inflation of eustachian tubes.

Alleged sinus symptoms.

Headache, severe and constant, localized to vertex and occiput, and behind the ears—typical site of posterior sinus pain, but increasing toward the end of the day (atypical sinus history, and suggestive eye headache). Burning sensation in throat, tongue, and side of nose. The diagnosis of this condition is established by—

1. The lack of molar teeth, or badly fitting dental plates, permitting overbite. To this can now be added overbite of natural teeth.

2. Mild catarrhal deafness, improved at once by inflation of eustachian tubes.

3. Dizzy spells, relieved by inflation of tubes.

4. Tenderness to palpation of mandibular joints.

5. Marked comfort to patient from interposing a flat object between jaws.

6. Presence of the typical headache after sinus or eye involvement has been corrected; presence of the typical headache when sinuses or eyes are found to be negative.

This is quoted as a help in diagnosing ear conditions prevalent among flyers. In the diagnosis of eustachian-tube disturbance there are some mouth conditions to be added to those given above. To list all mouth conditions which should cause suspicion some repetition will be necessary.

1. Badly fitting dental plates, permitting overclosure of the mandible.

2. Lack of molar teeth either bilateral or unilateral.

3. Natural teeth wherein there is a wide overclosure.

4. Teeth which are short from the gingival to the occlusal surface.

5. Mouths wherein the curve of spee is lacking.

6. Posterior bridgework within improper occlusion.

Any of these conditions may cause changes in the temporomandibular joint, which functions in direct relation to the occlusal plane of the teeth. Changes in this joint are one of the exciting factors in any of the eustachian-tube symptoms. With the rapid change in atmospheric pressure caused by rapid loss of altitude the eustachian tube has been burdened more than ever before. It now becomes one of the prime factors in both comfort and safety in air travel. Comfort for the passenger by relieving pain within the ears. Safety for the pilot in preventing dizziness during rapid pressure changes. That the reader may realize the amount of pressure to be compensated for we will take an accurate record of an actual flight. Starting from the surface of 571 feet above sea level, we have an atmospheric pressure of 140.6 millimeters. This is equal to 14.4 pounds per square inch. At 17,928 feet we find that the pressure has dropped to 380.6 millimeters or 7.46 pounds per square inch. This is a decrease of 7 pounds per square inch. Since the rate of ascent is usually comparatively slow and providing there is any eustachian-tube function there will be no difficulty with this loss of pressure. However, the descent is usually made much faster and as the pressure increases the eustachian tubes must open adequately to compensate for this change. It can be readily seen that a change of 7 pounds per square inch on the eardrum could be very painful and hemorrhages in the ear are not an uncommon thing in military flying. Many cases have been reported wherein eardrums have been ruptured during diving tactics.

There have been numerous cases in diving wherein the pilot instead of pulling out of his dive in the usual manner has started the ship spinning and crashed. In the past these accidents have been explained by assuming that the controls have jammed, faulty construction of the airplane, or something coming loose in the cockpit striking the pilot or fouling his controls. One of the country's most famous test pilots, in an article published after his death, made this statement, "I have those kind of ears which will not adjust themselves to changes in atmospheric pressure."

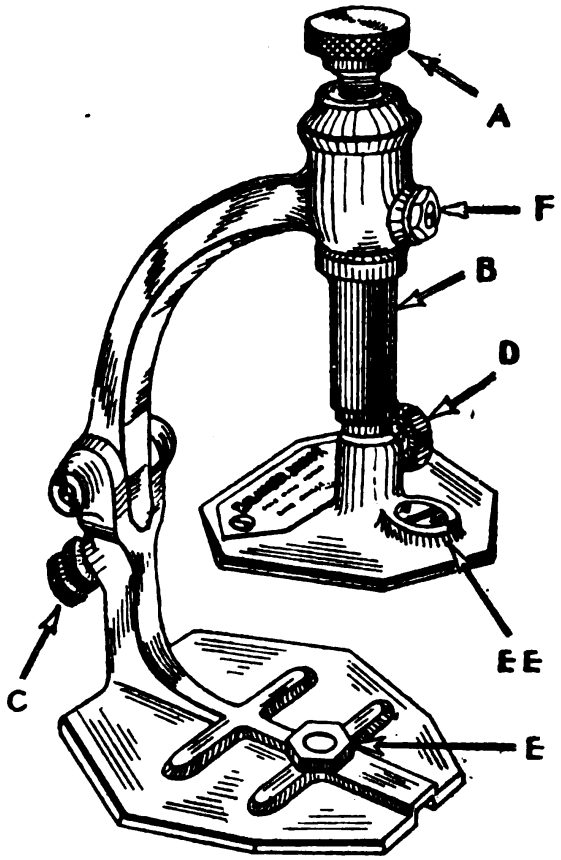
His death was attributed to the windshield on the cowling coming loose and striking him in the forehead with sufficient force to render him unconscious. Perhaps this explanation seems a little farfetched, since it is possible for the pilot's head to have struck the windshield and dislodged it at the time of the crash. It is common knowledge however, that things do come loose and that the excessive speed of the airplane frequently drives the object with tremendous force and if the pilot is struck might readily render him unconscious. So we are not disqualifying this possibility altogether. However, the writer feels that dizziness rather than unconsciousness was the cause of this crash. Much has been said regarding pain and dizziness in military flying. Many cases have been observed wherein descent was made at the rate of 300 foot per minutes or less, as is the Department of Commerce regulation for air lines.

TREATMENT

The treatment of these cases is identical but with variations in the amount of opening depending upon the type of case to be corrected. Successful completion of these cases is dependent upon careful diagnosis and complete understanding of what is to be done before actual work is started. To do this, three things are important: First, radiographs; second, accurate impressions; and third, a good diagnostic instrument.

If questionable teeth are found they should be removed. Any deep-pocket formation should be treated surgically and the mouth put in the best possible condition to receive the restorative work. When surgery is necessary the mouth should be allowed to heal before taking impressions. Impressions should be taken in plaster, solvite, dentocoll, or some other plastic material which will give accurate reproductions. These casts are then mounted on the instrument to plan a course of procedure. The Hagman (fig. 1) balancer is the instrument to be used here as I have found it an easy and efficient instrument with which to work. To mount the case lock the instrument in normal position with screw A. Lubricate bases of upper and lower supports to facilitate removal of casts after

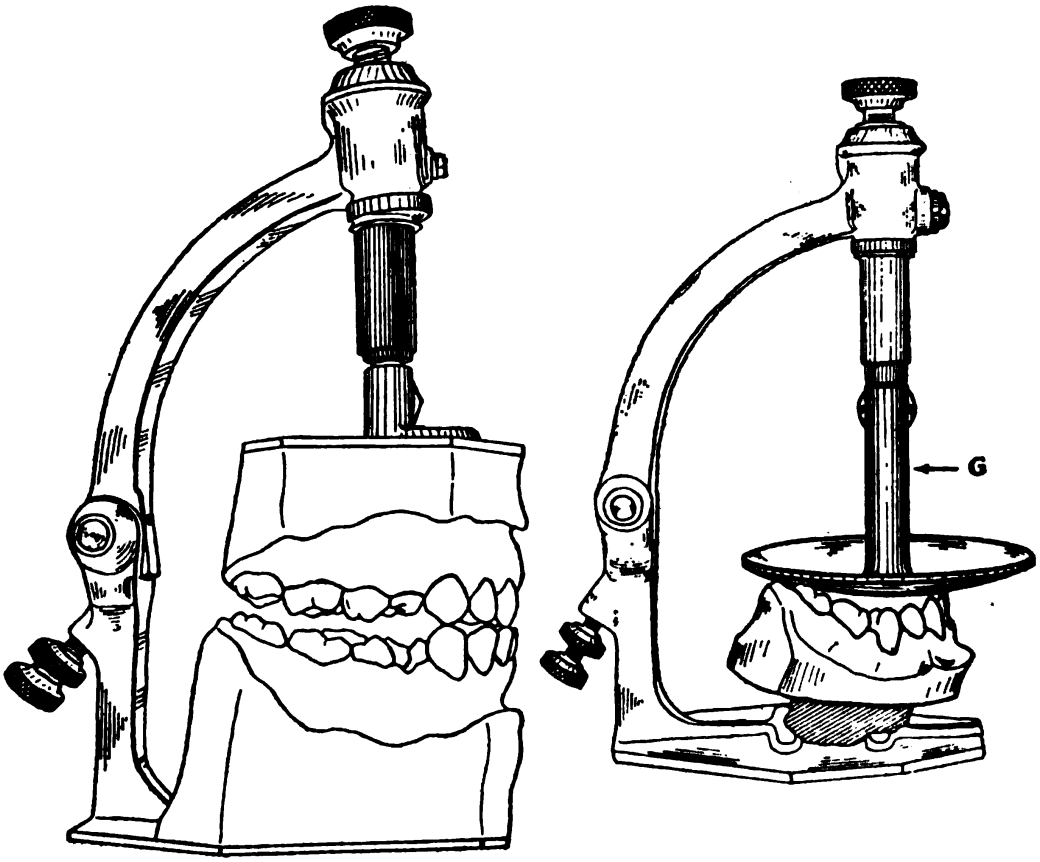
mounting. Place moulding clay or mouldine on lower base and position occlusal surfaces midway between upper and lower bases. Remove upper cast and model support and attach curved mounting plates G (fig. 2). This plate represents a segment of an arc with a 4-inch radius. The surface of this plate is 4-inches from the point of rotation.



Part A. Positioning or lock screw for holding models in any desired relation.
Part B. Automatic spring for returning upper model to central relation.
Part C. Jack screw for opening or closing the bite.
Part D. Set screw for upper model support. When loosened, allows horizontal rotational movement for grinding or milling to lessen interlocking cusps.
Part E. Retaining nut which retains the model on the base.
Part EE. Retaining screws which when removed from the base enable the model to be removed.
(Part E will remain in the model. With extra nuts (Part E) other cases may be mounted.)
Part F. A screw for adjustment to prevent any horizontal lateral movement.

Place lower cast against the mounting plate and position so that the center of the pendulum bisects the center of the curve of spee of the teeth of the cast. This should place the greatest number of teeth possible in occlusal contact with the mounting plate. When this is done attach lower cast to the base with soft plaster being

careful to engage the retaining screw. When plaster has set remove mounting plate and replace the upper model support. Set upper cast in occlusion with lower and add soft plaster to the top of the cast until it engages upper retention screw. After casts are mounted open the instrument sufficiently to allow the upper cast to swing on the pendulum freely. Then while swinging the upper cast, allow the instrument to close until the first teeth to come in contact are just touching. This may be any tooth, any in the arch, and the casts may be in any relation when this contact is made.



When this point is reached tighten the locknut on the set screw at the back of the instrument and allow the upper cast to fall into central relation with the instrument. In this relation the upper cast is probably too far forward and the cusps of the bicuspids and molars are striking end to end rather than interidgitating. If this is the case move the cast distally until the cusps of the upper teeth would fall into proper relation if the instrument was closed, and tighten screw A on the instrument to hold the cast in position while the tie-in is made (fig. III). The tie-in is made either of modeling compound or low-fusing metal. To use compound soften a small amount and place on the incisal of the upper and lower centrals and allow to cool. Before chilling the compound, check the casts

to make certain they are in the proper position mesiodistally. When compound is hard unscrew the upper cast from the instrument. This will allow the casts to be removed from the tie-in without breaking the teeth. The tie-in is then removed and trimmed with a sharp knife until only the upper centrals and the lower centrals and laterals are covered. The labial portion is then cut away just to the incisal or to that point where the teeth may be seen resting properly in the impression when placed on the instrument. It is advisable to vulcanize this tie-in as it is fragile when trimmed small enough to use in the mouth. The tie-in is then taken to the mouth and wax bites are taken with tie-in in position. The upper cast is then removed from the instrument and remounted to the new bites.

When remounting the upper cast loosen the instrument and allow clearance to completely close with the pendulum in a vertical position. Then add soft plaster to the top of the cast and engage retention screw. The casts are now in a working position and ready to make the skids. At this point the upper cast is removed and the occlusal guide replaced on the instrument. Tinfoil (0.003) is then burnished over the lower posteriors. This is covered with 28-gage casting wax, and inlay wax is added to the occlusal surface; the occlusal guide is closed on the soft wax giving a smooth surface. Once this surface is obtained the lower cast may be removed for convenience in carving. The buccal and lingual are carved down and grooved at the proximal spaces. The thickness of these surfaces should be as thin as it is practical to handle. These sides extend gingivally only to the bell of the teeth they cover. The occlusal carvings are then to be made. These must be more or less flat as the tops of the cusps must conform to the arc which was made by the occlusal guide. With the carvings complete attach spee and remove from cast. The tinfoil is then stripped and the wax invested and cast.

When the lower skids are cast and polished, they are placed back on the cast and the upper cast remounted. With the lower skids in place some of the upper teeth may interfere with closing the instrument. If this occurs cut these teeth at the point of interference until there is just clearance. Try the tie-in on the casts to make sure they are in proper and tinfoil the upper posterior teeth. The procedure of waxing is the same except the lower skids act as a guide from which to carve. When the upper skids are cast and polished, cut holes where the cusps were cut off. This allows the cusps of the teeth in the mouth to protrude when the skids are tried in. If there is any interference when seating the skids in the mouth, grind until they go into place easily and are completely seated upon the teeth. Where the cusps protrude through the skids

grind the teeth off flush. This will eliminate any interference during the excursion of the mandible. The skids are then cemented and patient allowed to wear them for several days. This gives the dentist an opportunity to see his result without cutting into good teeth and a means of correcting any mistake which may have been made in the mounting of the casts. Assuming the amount of opening is correct and the patient is comfortable we are ready to build the permanent castings.

As a starting point we prefer the upper arch. One skid is removed and the teeth on that side prepared. After impressions are taken, a bite is taken in soft wax with the tie-in in place. This, when poured, will act as a guide as to the thickness of wax necessary on the occlusal. When pouring this bite, place the dies in the wax and pour one side at a time allowing the plaster to extend over the ends. When the plaster has set cut a T-shaped groove in each end and apply separating medium. Then when the opposite side is poured, a fixed relation is established. Wax patterns are then carved to try in the mouth and check with the tie-in. When check has been made, cast with hard gold. The next step is the opposing lower and the same procedure is followed. After completion of one side remove the upper skid on the other side and follow through in the same manner.

SUMMARY

The symptoms of stopping and pain in the ears, temporary deafness, and dizziness are common to sudden loss of altitude in flight or in landing.

Observations in six cases are presented to show that an important factor in production of these symptoms is malocclusion with compression of the eustachian tubes.

Eustachian tube effects from this source are suggested as the explanation of certain air accidents, for which no mechanical cause could be found.

The technique of repositioning of the mandible is given in detail.

Increase in vertical dimension of the jaws and correction of malocclusion in six cases have entirely relieved the various eustachian effects for a period of 3 years.

CLINICAL NOTES

MEDIAN LOBE PROSTATIC HYPERPLASIA WITH PROSTATIC CALCULI—REMOVAL BY TRANSURETHRAL RESECTION

By JOHN F. LUTEN, Lieutenant, Medical Corps, United States Navy

Prostatic calculi are not uncommon.

They are often associated with benign hyperplasis, rarely with malignancy and usually with chronic inflammation with increase of fibrous tissue. Calculi in the seminal vesicles are rare. More common are seen calcareous deposits of tuberculosis or other chronic inflammations. Calculi in the prostate and vesicles are not urinary calculi. They arise by depositions from the secretion present in the ducts, just as urinary calculi arise from the urine. The laws of their formation are undoubtedly the same. They are usually composed of cholestrin. They are, as a rule, small, round, multiple, and dark colored.

Transurethral resection with the McCarthy resectoscope as a rule is not recommended for the removal of prostatic calculi. Perineal prostatectomy or perineal prostato-lithotomy is the operation of choice. The supra pubic method is not recommended. A case is reported below in which a hyperplastic median lobe was resected and calculi removed at the same time successfully.

CASE REPORT

R. R., chief boatswain's mate retired, married, white, male, age 66 years, was admitted to the United States Naval Hospital, San Diego, Calif., June 11, 1935, with a diagnosis of chronic prostatitis. His chief complaint on admission was difficulty of urination, dribbling, and intense burning on urination. There was a nocturia of two to three times but no dysuria or hematuria. The urine was cloudy and had a foul odor. His symptoms had been present since February 1931 and at that time he was told that he had stones in his prostate. According to the patient's own statement his bladder was "cut open." However, his symptoms were not relieved and have persisted since that time. He had been receiving prostatic massages and bladder irrigations for several months prior to admission to this hospital. He states that he could not empty his bladder completely and had a residual urine varying from a few drops to 200 cc.

He denied venereal infection. In July 1928, he had a fish bone removed from his intestinal tract, and at the same time his appendix was removed. He is troubled with constipation and takes mild cathartics. Smokes many cigars but uses very little alcohol.

Examination reveals a somewhat elderly white male, slightly obese, fairly well nourished, well developed, height 66 inches and weighing 175 pounds. The temperature and respiration were normal. The pulse was weak in volume and somewhat irregular. Blood pressure, systolic 160, diastolic 100. All teeth extracted. The prostate was slightly symetrically enlarged and uniformly indurated. There was marked crepitation on palpation. The sulci were clear and the anterior notch was well defined. The vesicles were not palpable. Secretion loaded with white blood counts. Urine was loaded with leucocytes and a few erythrocytes, otherwise negative; Kahn negative; white blood, red blood, and differential blood counts were normal. Urea nitrogen 12. Residual urine 80 cc. Pthalein: 62 percent of dye was eliminated during the first hour, and 26 percent of the dye was excreted during the second hour. Electrocardiogram showed serious myocardial damage.

Plain K. U. B.—Shows the kidney shadows in normal position. Psoas shadow well outlined both sides. No X-ray evidence of opaque stone in either kidney or ureteral area. There is a nest of radio-opaque shadows over the prostatic area which probably represent prostatic calculi. There is a marked hypertrophic arthritis involving the lumbar spine with complete bridging of the twelfth dorsal and first, second, and third lumbar vertebra right side (fig. 1).

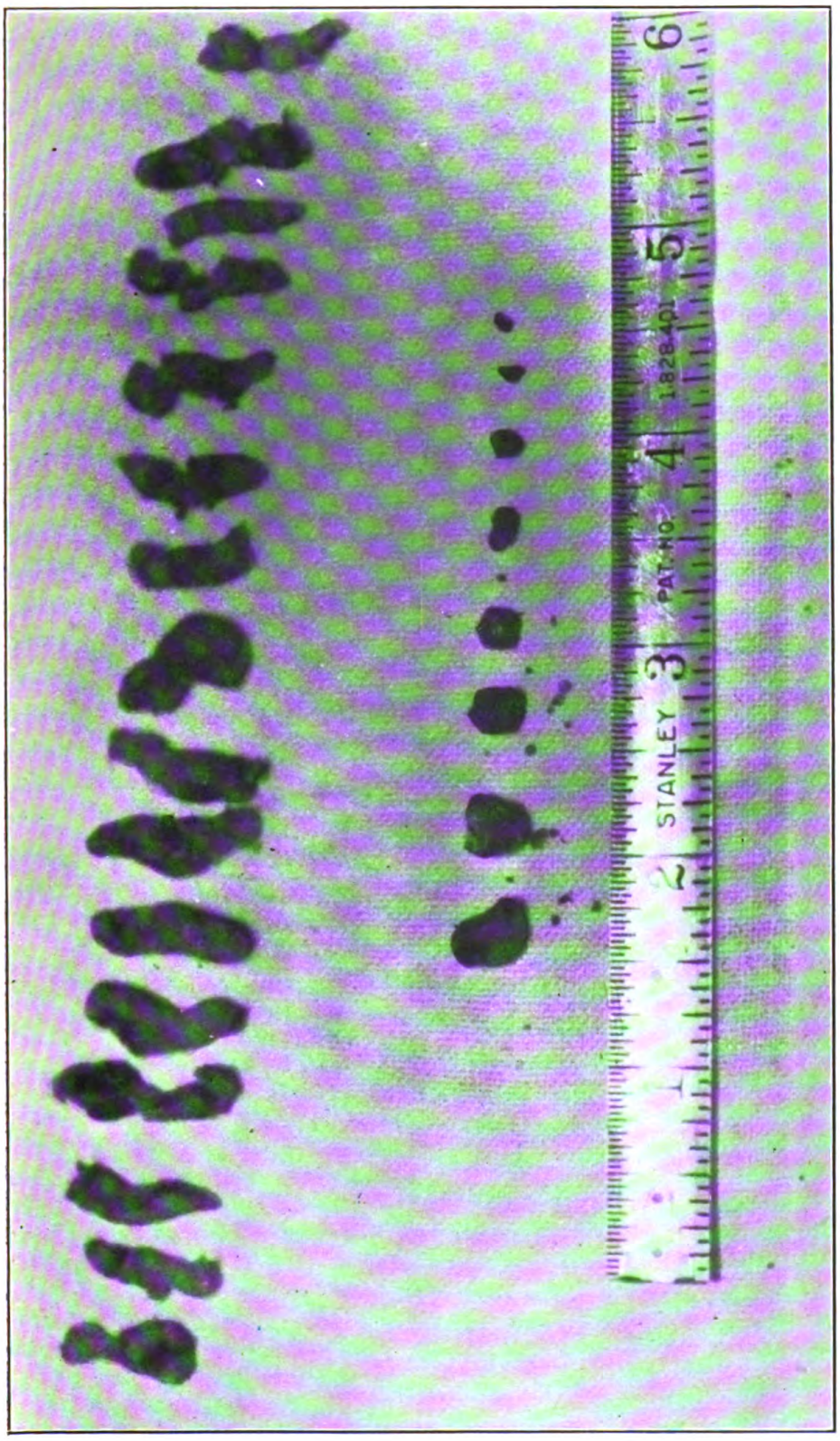
Cystoscopic examination revealed marked trabeculations with numerous deep cellules. Residual urine 50 cc. Bladder capacity 525 cc. The bladder urine was cloudy, foul smelling, and contained numerous shreds and flakes. The bladder mucosa and trigone were chronically inflamed. The ureteral orifices could not be seen. There was mild posterior lateral notching with encroachment of the median lobe on the vesicular neck. There was mild inflammatory edema about the vesicular neck. The verumontanum was edematous and inflamed. Intravenous pyelograms revealed mild bilateral hydronephrosis slightly more marked on right. X-ray taken with radio-opaque catheter in bladder, and a small amount of 3-percent sodium iodide injected showed the catheter in close proximity to the nest of stones in the prostate previously reported in the K. U. B. (fig. 2).

Bilateral vasectomy was done and an indwelling two-way catheter with continuous irrigation was begun. Short-wave radiotherpy was given daily to the prostate. As the stones gave the appearance of a mass of small calculci rather than large concretions, due to the apparently close proximity to the urethra. and as the patient was considered a poor operative risk due to serious cardiac damage, transurethral resection was decided upon.

Transurethral resection with the Stern-McCarthy resectoscope was done June 20, 1935, under caudal anaesthesia, 30 cc procaine, 2 percent with adrenalin being used. Seven (7) grams of tissue and eight (8) calculi varying in size from a millet seed to a buckshot were removed (fig. 3). There was no bleeding and the patient made an uneventful convalescence, being discharged from the hospital July 9, 1935. He was instructed to return for ambulatory treatment (fig. 4). K. U. B. following the operation shows all stones have been completely removed. There was a slight leakage when the bladder was full which persisted for 1 month. For 6 months following the operation there has been no residual urine. The patient voids freely and there is a complete absence of symptoms. His constipation has been relieved.

SUMMARY

Under ordinary circumstances transurethral resection would not be considered feasible for removal of prostatic calculi, the perineal



TISSUE AND STONES REMOVED AT OPERATION.

route being the operation of choice. However, the case presented has proved to be an exception to the rule and transurethral resection was selected as the method of treatment. Although somewhat early to draw definite conclusions it appears that this procedure has fully justified its selection and proven eminently successful in this selected case.

The writer wishes to acknowledge the very helpful cooperation and assistance of Dr. R. P. Parsons, Chief of the Urological Service, in the studies of these cases, and valuable help rendered by the X-ray and laboratory departments.

MENINGOCOCCUS MENINGITIS: CASE REPORT

By C. J. HOLEMAN, Captain, Medical Corps, and J. W. KIMBROUGH, Lieutenant, Medical Corps, United States Navy

It is not the purpose of this paper to go into the details of symptomatology and diagnosis which can be found in any textbook of medicine.

Some authorities feel that the disease occurs chiefly in carriers who have, for some reason, suffered a lowering of their resistance.

In the Public Health Bulletin on "The Control of Communicable Diseases", Reprint No. 1,697, it is stated that incubation period is 2 to 10 days, commonly 7. It is further stated that "in rare instances the period may be longer when a carrier develops the disease."

The following case is of interest due to the length of time between the last possible contact ashore and the development of the disease. As the patient had been under restriction for a considerable period his movements could be accurately checked. Prior to this one case, and subsequently, no other cases occurred.

I. G. G. Seaman, second class. U. S. S. *Texas* (at sea). April 22, 1929.

Diagnosis.—Cerebro spinal fever (#802), not misconduct.

Chief complaint.—Headache, general malaise, vomiting.

Present illness.—Patient says he began feeling bad last night from no apparent cause. Under confinement in brig and has not been ashore since March 4, 1929 (in Panama). Onset with nausea and vomiting accompanied by rather severe headache. Says he is nervous, feels badly all over, and thinks he is catching cold. Neck slightly stiff and sore.

Past history.—Syphilis in October 1928.

Examination.—Patient does not seem to be very ill. Temperature 101. Reflexes normal, neck slightly stiff. Kernigs and Brudzinskis signs not elicited.

Treatment.—To bed, saline purgation, A. P. C. caps., gr X, three times a day.

April 24, 1929.—Temperature still elevated and symptoms more marked. At 10 a. m. spinal puncture revealed very cloudy fluid under normal pressure, smears from which showed occasional extra cellular and intracellular gram negative diplococci. Cell count 6,814. Polymorphonuclear predominant type of

cell. 8½ cc fluid removed and an equal amount of antimeningococcic serum introduced. At 2 p. m. (same date) 12 cc very cloudly spinal fluid removed under definitely increased pressure, and an equal amount of antimeningococci serum introduced. Strict isolation.

April 24, 1929.—Blood Kahn test four plus.

April 25, 1929.—Temperature normal. General condition improved. Spinal puncture revealed cloudy fluid under normal pressure. 7 cc fluid withdrawn and an equal amount of anti meningococcic serum introduced.

April 25, 1929.—Transferred to U. S. S. *Relief* for further observation and treatment.

U. S. S. *Relief.*

Diagnosis.—Cerebrospinal fever (no. 802), not misconduct.

Admitted 7 p. m.

Chief complaint.—Severe headache.

Present illness.—See above entry. Patient too ill for reliable history. Patient became ill, according to entry on April 21, 1929, while in brig. On April 24, 1929, symptoms were suggestive of meningitis when a spinal puncture was done and diagnosis of cerebrospinal fever was made. Two injections of very small amount serum were given, being unable to obtain very much spinal fluid. Numerous meningococci in smear.

Physical examination.—Well developed and nourished white single male, 28 years of age. Temperature 99.8; pulse 112; respiration 28 at time of admission. Face flushed. Eyes prominent. Conjunctivae injected. Neck stiff. Kernig positive. High leucocytosis. Patient delirious, but at times talks rationally. Requires constant watch since he has a tendency to get out of bed.

8:00 p. m.—Spinal puncture. Fluid very cloudy and under increased pressure. 25 cc removed and 30 cc serum given intraspinally.

Spinal fluid-smear and culture.

Smear.—Occasional gram negative diplococci; many pus cells.

Culture.—Negative.

April 26, 1929—9 a. m. spinal puncture. Fluid thick with pus, 20 cc removed. Not much pressure. 30 cc serum given.

11:30 a. m.—Patient more delirious. Critical telegram sent to next of kin.

9:00 p. m.—Spinal puncture. Fluid purulent. Very little pressure. 30 cc serum given. During night pulse, respiration, and fever mounted.

Cell count and culture of spinal fluid.—Cell count, 10,220. Culture, negative.

April 27, 1929.—Died at 4:50 a. m.

Autopsy report.—Time of death, 4:50 a. m., April 27, 1929. Time of autopsy, 9 a. m., April 27, 1929.

(A) *General appearance.*—Well developed and nourished white male, 28 years of age. Moderate rigor mortis and post-mortem discoloration.

(B) *Cranium.*—Brain deeply congested and soft. Large amount purulent spinal fluid. Plastic purulent exudate over base of brain and medulla. Large amount of purulent spinal fluid in lateral ventricles with small amount of plastic yellow exudate in right ventricle similar to that observed at base of brain.

(C) *Thorax.*—Heart and lungs normal on inspection and palpation with exception of an old right pleurisy which resulted in numerous tenacious adhesions uniting visceral and parietal pleurae.

(D) *Abdomen.*—No pathology noted on inspection and palpation except for some adhesions between omentum and gall bladder.

(E) *Diagnosis.*—1. Cerebrospinal fever. 2. Chronic fibrinous pleurisy, right

(F) *Cause of death.*—1. Cerebrospinal fever.

TWO CASE REPORTS: I. TREATMENT OF EROSION OF SKIN OF ABDOMEN DUE TO FECAL FISTULA. II. FEEDING BY PROCTOCLYSIS OF FOOD OBTAINED BY SUCTION FROM AN INTESTINAL FISTULA

By FREDERICK G. FOX, Lieutenant Commander, Medical Corps, United States Naval Reserve

I. A soldier at Fort Wayne station hospital (G. E. C.) that had had an appendectomy and induced enterostomy, which resulted in a fecal fistula, through the jejunum, developed a skin erosion, due to the collection of digesting food over the wound. The dressings had to be changed often and were wet and irritating. The skin was always washed and dried, and different preparations were applied, but the condition became worse. Vaseline gauze was tried, also talcum, but the condition persisted, and the patient began to fear the dressing, as it was painful.

Through the suggestion of a colleague, who saw an article in the Journal of the American Medical Association, a 10-percent aqueous solution of tannic acid was prepared, and after cleansing the skin, it was applied by spraying with an atomizer. A heat lamp was used to dry the solution, and after several applications a black layer of tannic acid soon covered the eroded skin.

After about 5 days it began to peel, and when it was all removed the skin appeared normal and nonirritating. The skin was again sprayed to protect it from other food that collected. In this particular case, a problem of feeding by proctoclysis presented, and an electric suction machine was thereafter used to collect the food appearing on the abdominal surface. This also helped to keep the skin from irritation.

II. On December 4, 1934, a soldier (G. E. C.), age 21, was admitted to the hospital at Fort Wayne, with symptoms of abdominal distress. He was a heavy drinker. There was only moderate tenderness at McBurney's point, and no vomiting or rigidity. He was slightly nauseated. His blood pressure was 128–45; weight 145 pounds; temperature 98.6°; white cell count 9,600 at 10 a. m. The urine was negative. At 2 p. m. the white cell count was 11,300. An enema was given; also morphine sulphate one-fourth grain, and he was prepared for an appendectomy.

The appendix was very difficult to find, and manipulation of the intestines caused them to become rapidly distended. After further exploration the appendix was finally found deep in the right upper quadrant. It was impossible to bring the appendix up into the wound for enough to place a purse-string suture. The appendix was ligated, and a circular suture was tied over a crushed stump. It was acutely inflamed. The exposed intestines were kept warm. The wound was closed in the usual manner.

On December 5 the patient presented symptoms of acute dilatation of the stomach with vomiting. Morphine was given without relief. Pituitrin was given, without effect. A gastric lavage of normal saline solution was given, but the vomiting continued. A soapsuds enema was given with small results, and no relief. Glucose and saline solution were given intravenously. On December 6 the abdomen became distended. Nothing had been given by mouth since the operation, and the patient was not improved. He was still vomiting. On December 7 the patient was very restless and was straining abdominal muscles, trying to relieve the distended abdomen. During the night an intestinal hernia presented through the lower section of the incision, due to the separation of the wound. The patient was again prepared for operation. The gut was with difficulty returned to the abdominal cavity, and an enterostomy was performed with a catheter sutured into the beginning of the small gut. The wound was closed, and a continuous suction lavage apparatus was connected with the stomach, by a catheter passed through the nose. A small amount of fluid escaped through the enterostomy catheter for about 2 days, when it failed to be of any further use. The vomiting was finally checked. No bowel movements had occurred since entering the hospital. Intravenous glucose was given daily. On December 9 food was given by mouth and retained, except a certain amount that was returned by the lavage suction apparatus. The patient was fed water, broth, milk, and eggnogs. The intravenous saline and glucose had to be discontinued because the patient became very restless through fear of the procedure, and because of clotting of the veins at the site of puncture. The vessels were small. Fluids were forced by mouth, but much was lost through the lavage suction apparatus, and the patient showed signs of dehydration. On December 10 food began to escape around the catheter sutured into the small intestine. On December 12 the catheter became loose and had to be removed. The abdominal wound began to separate, and the gut again worked into the wound. Sloughing of tissue began, and an agglutinated mass of intestines formed the base of the abdominal wound. On December 16 the lavage suction apparatus was discontinued. On December 17 the patient had three bowel movements. On December 18 the patient seemed more restful. Food that was eaten presented immediately in the wound and became fecal under the dressings. This produced erosion of the skin surrounding the wound. Saline by proctoclysis was given and retained. On December 20 a semisolid clay-colored stool was passed. No further bowel movements were passed, and there apparently was an obstruction in the agglutinated intestines. The skin of the abdomen had become more eroded, and it was painful to change the dressings.

On December 26 it was discovered that many dressings had been used in treating the patient, and it was feared there would be a shortage. An idea then presented, and the partially digested food coming through the fistula was collected with an electric suction machine of the type used in collecting mucus and blood, while doing a tonsillectomy under general anaesthesia. The food collected consisted of milk, water, orange juice, soft-boiled eggs, soda crackers, cream of wheat, rice, and mashed potatoes. The food was thrown away until it was decided to try giving it by proctoclysis. The food was strained to exclude large particles that might clog in the tubing. It was placed into a glass flask, tapered to the bottom for connection of rubber tubing. The flask was attached to a floor stand and elevated to about 4 feet above the patient's head. Rubber tubing from the flask was connected to a rectal tube inserted into the colon. A stopper was connected and the food was given slowly. The food mixture in the flask can be kept warm by floating a stoppered glass bottle of hot water in the mixture. About 1,000 cc of food mixture can be slowly given in about a half hour. If the colon is irritable, the feeding can be extended over 1 hour's time.

The next morning after only one such feeding, of about 1,000 cc, the patient was more contented and had a better color. He appeared entirely relaxed. The collecting of the food was accomplished by placing a relay of convalescent patients near the patient, who threw on the electric switch to start the electric suction machine whenever food collected in the wound and applied a suction instrument, covered with rubber tubing on the end, into the wound. The tip of the instrument containing small holes was disconnected.

After 2 or 3 days of rectal feeding, the patient will have a normal bowel movement, if the colonic musculature is active. Should the patient become distended and restless, an enema may be given.

This method of feeding was continued until the patient developed a hypostatic pneumonia and died on January 15, 1935.

CONCLUSIONS

1. Feeding by proctoclysis of food obtained by means of the electric suction machine that had passed through the stomach and upper part of the small intestine was very successful in this case, but needs further experimentation.

2. Sufficient water can be given and absorbed by proctoclysis, and that simpler and easier than any other method, except by mouth. It should be given with the rectal feeding. There is report of a case where too much water was given, resulting in the death of the patient. Water Intoxication, Journal of the American Medical Association, May 4, 1935.

3. There is no simple method of knowing how much food is absorbed by proctoclysis, except that the patient holds or gains weight.

4. The patient becomes cooperative if he feels restful and receives food and water.

5. Cases of fecal fistula of the jejunum, without intestinal obstruction, can be more successfully treated by feeding predigested food that has passed through a stomach by this method.

6. Since most of the food passes through the jejunal fistula soon after eating the patient passes a restful night, with dry instead of wet dressings covering the wound.

7. There is no odor of feces around the patient when using the electric suction machine for cleansing the wound of food, and no erosion of the skin occurs.

8. This patient was kept alive by proctoclysis feeding from December 28, 1934, when it was first given, until January 15, 1935, a period of 18 days, when he died of hypostatic pneumonia. Had proctoclysis feeding been started on December 18, when the first food appeared in the wound, the patient might have lived to undergo a third operation for repair of the intestinal obstruction.

9. Glucose should not be added to the food mixture, as it causes fermentation of the contents.

NEW DEVICES

A NEW TYPE SLING FOR A THOMAS SPLINT

By H. M. Webber, Lieutenant Commander, Medical Corps, United States Navy

There are several objectionable features to the usual method of preparing a sling for suspension of a limb in a Thomas splint;

(*a*) Difficulty of placing the usual fabric on the splint.

(*b*) Need of frequent adjustment to prevent undue pressure at various points.

(*c*) Untidy appearance of the usual suspension.

(*d*) Frequent obstruction in X-ray work caused by the usual clips.

For these reasons the described piece of apparatus was designed. A moderately heavy piece of canvas was cut so as to be the general shape of the space between the lateral rods of the Thomas splint, coming up to the ring and extending almost to the distal end of the splint. It is wise to lap over and sew the cut edges just enough to give it a finished appearance and prevent unraveling. Then, about 3 inches apart, brass grommets are placed on the long sides which will be opposite the lateral rods. A piece of light line is tied firmly, where one of the rods joins the splint ring (point A or B, fig. 1) and is carried around the splint ring; then threaded alternately through each grommet and around the lateral rod of the splint. At the distal end of the splint the line is simply looped over it (point C, fig. 1) and continued back through the grommets and over the rod of the other side and secured to the point, where the rod joins the splint ring, again passing the line around the ring. Maintaining these fixed points of suspension at the distal end and at each point, where the lateral rods join the splint ring, is necessary to prevent shortening of the sling, because each lateral point is changeable depending upon the pressure placed upon it. The sling is pulled up quite taut, before it is finally fixed by the last tie at the ring. It will be found now that when the leg is placed in the splint the prominences of the extremity will make their own adjusted cradle and it has been my experience that it is immediately comfortable. It can be as well used, with the hinged splint, in fact, seems best adapted to this use, because it fits so well the bend of the knee. A thin layer of sheet wadding or soft material may be laid over the sling but is not necessary. There may be cases, where at the point of fracture there may be a tendency to sag. This has

not been my experience, however. A simple narrow strap would be sufficient to maintain the desired angulation, placed under the sling.

To me this sling offers the following advantages:

(*a*) It is easily attached to the splint.

(*b*) Needs relatively no adjustment, being immediately comfortable, fitting automatically the contour of the limb.

(*c*) Is neat in appearance.

(*d*) Offers little obstruction to the X-ray.

(*e*) When once made can be scrubbed and used as a piece of permanent equipment, a feature especially desirable it would seem in the military services, where complete equipment, which can be easily stored and transported, ready for use, is needed.

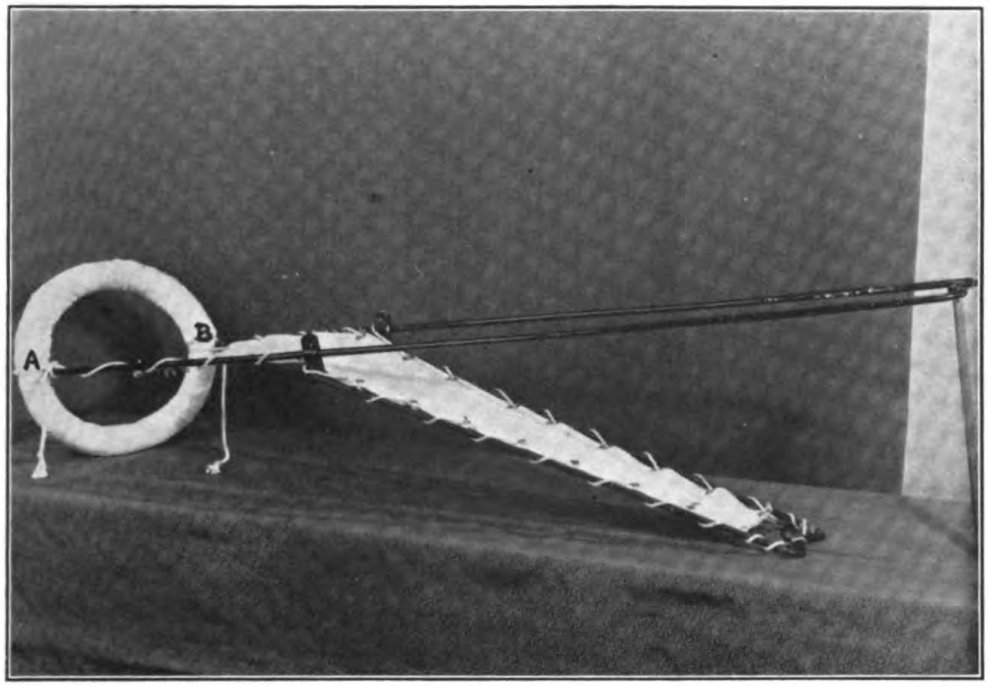


FIGURE 1.

Note the fact that the rope is passed around the ring at *A* and *B* before being tied to prevent lateral movement up on the rod; also that the distal end is looped over the bend in the splint at *C* for the same reason. Note also that the sling is quite taut on the splint.

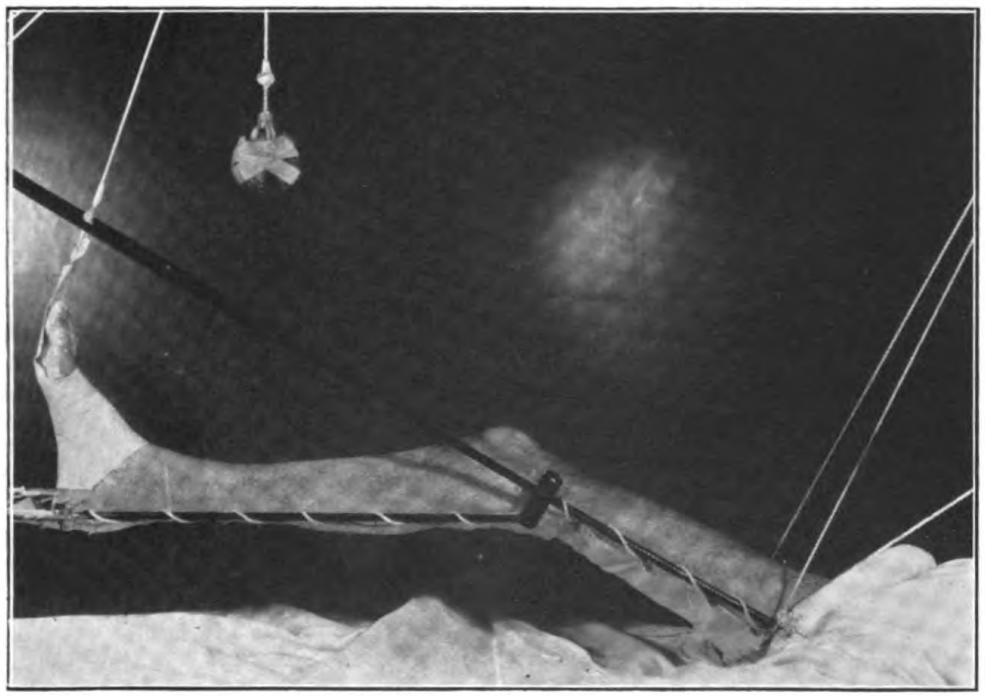


FIGURE 2.

Note the manner in which the prominence of the heel and calf have made their own well fitting cradle; also neat appearance of the suspension and snug even fit about the knee.

NAVAL RESERVE

MEDICAL CORPS

OFFICERS OF THE MEDICAL CORPS, UNITED STATES NAVAL RESERVE, SELECTED FOR PROMOTION TO CAPTAIN AND COMMANDER

A selection board which convened at the Navy Department, Washington, D. C., April 1 to April 11, 1936, selected for promotion to captain, Medical Corps, United States Naval Reserve, Commander P. B. Brockway and Commander L. R. G. Crandon. Lt. Comdr. James J. Hogan and Lt. Comdr. Albert Soiland were selected for promotion to commander.

PROMOTIONS OF MEDICAL OFFICERS, UNITED STATES NAVAL RESERVE, FOR THE FIRST AND SECOND QUARTERS OF 1936

Name	From—	To—	Appointed
James, Gordon Maxwell	Lieutenant (junior grade) MC-V (G)	Lieutenant MC-V (G)	Jan. 15, 1936
Williams, James Peery	do	do	Jan. 17, 1936
Duff, Alexander McGill	do	do	Apr. 9, 1936

DENTAL CORPS

PROMOTIONS

Name	From—	To—
Levin, Harry L	Lieutenant (junior grade) DC-V (G), U. S. N. R.	Lieutenant, DC-V (G), U. S. N. R.
Wargelin, Arno I	do	Do.

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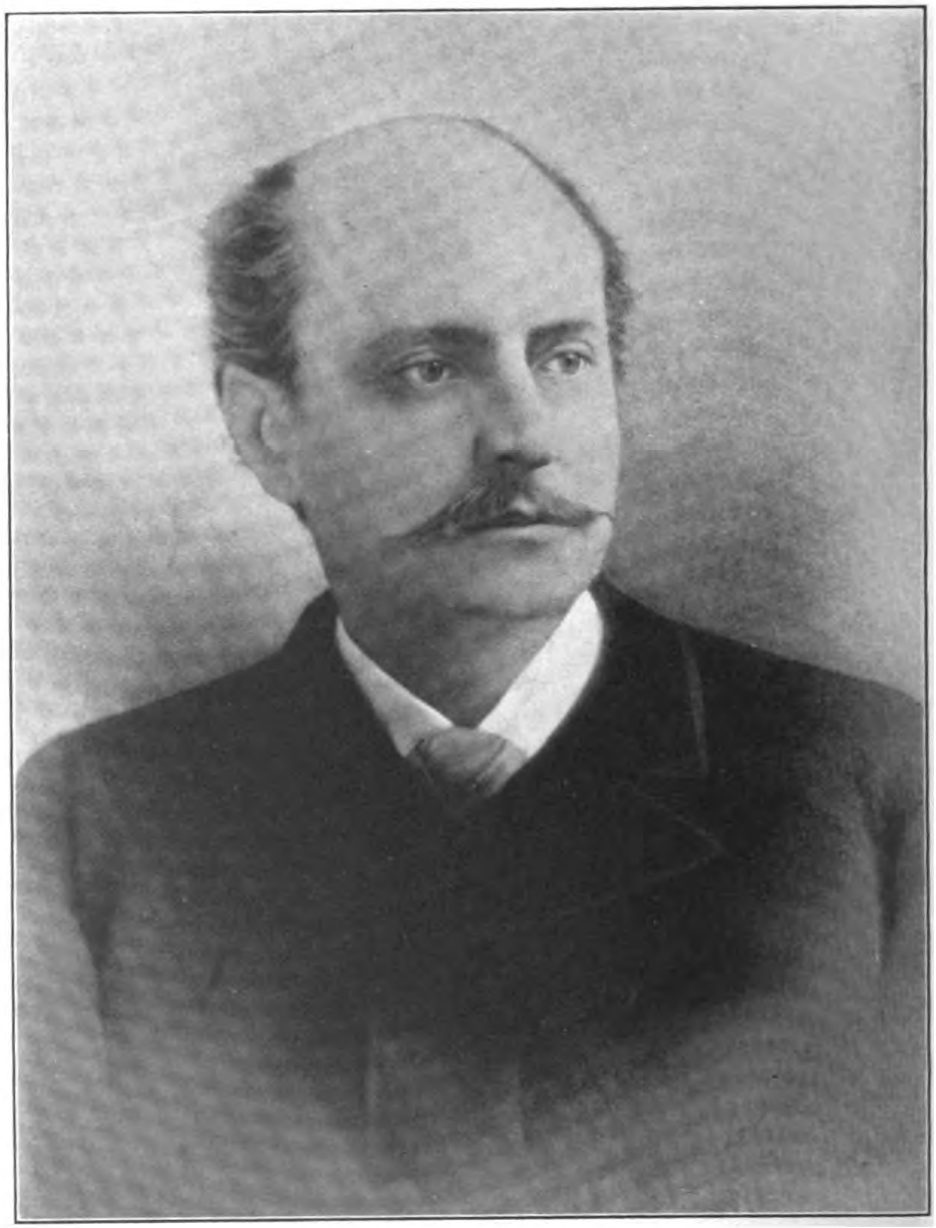

PHILIP SKINNER WALES.
Surgeon General, U. S. Navy, 1879–83.

NOTES AND COMMENTS

PHILIP SKINNER WALES, SURGEON GENERAL, UNITED STATES NAVY. 1879–83

The eleventh Chief of the Bureau of Medicine and Surgery and the seventh to hold office as Surgeon General of the Navy was born in Maryland on February 27, 1834, and appointed from that State as an assistant surgeon on August 7, 1856. His first service was in the Asiatic, or as it was then called, the East India Squadron, on the steam frigate *Mississippi*. He returned at the beginning of the Civil War and was on the *Water Witch* during the early years of the war. Except for a short tour at the naval hospital, Norfolk, in 1863, he was at sea the remainder of that conflict on the steamer *Fort Jackson* in the south Atlantic and the west Gulf blockade. He was commissioned surgeon on October 12, 1861, and medical inspector on June 30, 1873. He was employed on special duty, probably in connection with the preparation of a medical history of the Navy during the Civil War from 1874 to 1878. He was appointed Chief of Bureau by President Rutherford B. Hayes on August 20, 1879, and completed his term of office on January 27, 1884. His Assistant Chief of Bureau during the whole period was Surgeon A. Hudson.

Dr. Wales' death occurred on September 15, 1906, at Paris, France, where he had lived since 1900.

An event of great importance to the Medical Corps in his administration, was the founding of the Museum of Naval Hygiene in Washington which, when united later by Surgeon General Tryon with the old naval laboratory and department of instruction at Brooklyn, were the nucleus of the present naval medical school for the postgraduate instruction of young naval medical officers. Surgeon General Wales founded the museum in 1883 and placed in charge of it Medical Director J. M. Browne, afterward Surgeon General and one of the most able and active officers in the corps. Tryon, when Surgeon General in 1894, moved the museum to the old naval operating buildings, where in 1902 Admiral Rixey, then just made Surgeon General, organized and established the naval medical school, the only postgraduate school of naval medicine in the western hemisphere and the most important library and repository of naval medicine in the New World.

Some interesting comparative statistics of the Navy and the Medical Corps in 1880, when Dr. Wales was Surgeon General, and at present, are given below. Statistical data are for the calendar year where diseases and injuries are concerned, otherwise for the fiscal year.

	1880	1934
Deaths per thousand	11.1	2.73.
Leading cause of death	Pneumonia	Motor-vehicle accidents.
Second most important cause of death	Tuberculosis	Drowning.
Admissions for venereal diseases per thousand	81.64	90.28.
Admissions for alcoholism per thousand	17.3	1.43.
Percentage of recruits rejected	31	34.
Total complement of the Navy and Marine Corps.	10,235	109,383.
Medical officers	171	832.

Of course, the methods of collecting and compiling statistics then and now are not strictly identical so the comparison is only approximate in character. Still it is likely that the comparisons are not without significance. It would appear from them that the venereal rate has altered very little, alcoholism has declined, and the death rate from all causes is much less.

During the latter years of his administration Dr. Wales discovered that the Chief Clerk of the Bureau of Medicine and Surgery, with the assistance of a confederate in the auditor's office, had been for years embezzling large sums of money. He obtained the signature of Dr. Wales (and probably those of several previous Surgeons General) by placing blank requisitions among copies to be signed and by simply turning up the bottoms of the vouchers for the Surgeon General to sign. He thus secured blank signed requisitions. Although there was no question of the integrity of the Surgeon General himself, who was indeed partly responsible for discovery of the frauds and testified before the grand jury that indicted the culprits, he was tried by general court martial on charges of neglect of duty and culpable inefficiency in the performance of duty, with 64 specifications. The trial was a celebrated one and caused an enormous amount of comment and controversy, as it was felt by many that Dr. Wales was the victim of circumstance and had been dealt with unjustly.

The court met on March 9, 1885. There were several things in the trial which may excuse the mixing of a little naval legal history with the medical history. The court itself consisted of the full 13 members allowed by law. The record, consisting of three large volumes, is probably the first proceedings of a court martial in our Navy to be typewritten. It was also one of the first in which oral questions and oral arguments were permitted. The question of the jurisdiction of the court was raised by the defense and was settled by the Supreme Court. Dr. Wales was found guilty and sentenced to receive

furlough pay and to retain the number in his grade that he then held for 5 years. In approving the findings, July 31, 1885, the Secretary of the Navy remarks that:

> No dishonest or corrupt act or motive involving any breach of personal integrity on the part of Medical Director Philip S. Wales as Chief of the Bureau of Medicine and Surgery was alleged against him nor is there any evidence in the record which would have justified such a charge.

In other words he was the victim of the corrupt acts of subordinates when responsible for the supervision of their conduct. In effect it constituted an exoneration of any intentional breach of trust or honesty.

SOME QUALIFICATIONS FOR FELLOWSHIPS IN THE AMERICAN COLLEGE OF PHYSICIANS AND AMERICAN COLLEGE OF SURGEONS

The applications of candidates for fellowships in the American College of Physicians and the American College of Surgeons, together with case reports submitted, are presented for study and recommendation of the postgraduate board. In passing upon the eligibility of candidates for these fellowships, the postgraduate board is guided by policies formulated by both of the colleges, and by the Bureau of Medicine and Surgery. For the information of medical officers who wish to become fellows of either college, some of these policies are here stated.

Candidates for fellowship in the American College of Surgeons must be men actively engaged in the practice of surgery, and their professional attainments in surgery must be such as would qualify them for admittance to fellowship from civil practice.

In respect to the American College of Physicians, a candidate must be an internist, pathologist, or clinical laboratory specialist. In both colleges high professional attainment is the first requisite. Fellowship in either college may be considered as a reward to men of outstanding professional abilities and reputations. Medical officers who are very young, who have not had sufficient experience in their specialties, or who have not been selected for promotion when due for promotion, cannot therefore be considered as officially meeting the standards set.

The board, in examining the case histories submitted by candidates for fellowship in the American College of Surgeons, has noted frequently the inclusion of a large number of cases which can only be considered as minor surgery. In general, the cases submitted should be limited almost entirely to major operative procedures. Circumcisions, removal of small skin tumors, amputations of fingers, operations for bunions, and similar minor surgical cases should not be included in such case reports.

In a number of instances the postgraduate board has recommended that action in regard to candidates be deferred so that opportunity for more experience can be obtained. Such candidates may be recommended for fellowships at a later date when their qualifications are thought to be such that they may meet the eligibility standards.

THE TREATMENT OF SCABIES

Attention to common diseases and more lowly therapeutics is often of importance to the practitioner who may gain more reputation by the successful cure of an irritating skin condition such as scabies than from the treatment of rarer but more serious complaints.

There are several features in the treatment of scabies which it may be of value to emphasize. These are:

1. That much milder parasiticides are sufficient to destroy the itch mite than are usually employed.
2. That a bath at the beginning of treatment is unnecessary and may be harmful by increasing the irritating effects of the parasiticide used.
3. That boiling the underclothes and bed linen is essential or a recurrence is certain.
4. A repetition of the treatment after a short interval (3 or 4 days to a week) to mop up stragglers and "hide-outs" among the parasites is necessary.

The first of these four points is of particular interest where men with tender skins, and women or children are the patients. There are many parasiticides: Sulphur, betanapthal, tar, balsam of Peru, pyrethrum, storax, and many others. Practically, sulphur is the one usually used and perhaps the most efficient. It is, however, irritating to most skins if used in considerable strength or over any long period. An effective prescription not likely to be irritating is as follows:

℞

		Gms. or Cc.
Sulphur precipitatii		
Balsam Peru	aa	2
Petrolatii		
Ung. Aquae Rosa	aa	15
M. et Ft. Ung.		

Sig.—Grease lightly the body from the neck down at night. Warm bath with plenty of soap in morning. Boil bed linen and underclothes. Repeat in 5 days.

In children the following is excellent. For children of 2 to 6 years or when sulphur is particularly irritating.

℞		Gms. or Cc.
Balsam Peru		3
Ung. Aquae Rosa	ad	30
M. et Ft. Ung.		
Sig.—Apply to affected areas.		

For infants or children up to 2 or 3 years of age:

℞		Gms. or Cc.
Styrax		
Ol Olivae	aa	30
M.		
Sig.—Apply to affected areas.		

In the use of sulphur ointments stronger than that given, the following rules should be observed:

For adults: 1 drachm (4 grams) of precipitated sulphur to the ounce of base.

Ten to twelve years: 40 grains (2.5 grams) of precipitated sulphur to the ounce of base.

Three to 10 years: 15 to 30 grains (1 to 2 grams) of precipitated sulphur to the ounce of base.

The base should be rather thin. Petrolatum, petrolatum and lanolin equal parts, or cold cream. In warm weather or in the Tropics these bases may be almost too thin for use except the one with lanolin. Lanolin may be used to stiffen any of these bases.

The use of an ointment base to hold the parasiticide in place seems almost a necessity in order to secure effective action, but ointments are so "messy" that many dermatologists have devised other treatments. Ointments have another advantage, however, due to the fact that the base is itself inimical to small animal parasites. In the case of the acarus they clog the entrance to the burrows and the respiratory apparatus of the insect and tend to produce death in that way. An ointment of petrolatum and lanolin applied freely to scabies lesions between the fingers will bring about great improvement due to these effects alone. One of the best and most effective of the nonointment treatments is that of Sherwell. This con-

sists essentially in the dusting of the bed sheets with one-half to 1 drachm (2 to 4 grams) of flowers of sulphur. The patient goes to bed stripped of all clothing. A bath is taken with castile soap and warm water the next evening, and 2 days later, if there is no irritation of the skin, the treatment is repeated. A little sublimed sulphur may be rubbed into the skin where heavy infestation occurs, as in the webs of the fingers. Although this method was first used by the writer with considerable skepticism it has nevertheless proved to be very efficacious. In neglected and long-standing cases where pustulation and crusting has occurred the patient should be hospitalized and Wilkinson's ointment is very frequently employed. This is:

℞

		Gms or Cc
Sulphur sublimati		
Ol rusci	āā	15
Cretae prep		4
Saponis mollis		
Adipis	āā	30

M et ft. ung.

Recently Sweitzer and Tedder, of the University of Minnesota, have used a pyrethrum ointment in 618 cases. They used an ointment of 0.75 percent pyrethrum. One hundred grams of this ointment represented 83 grams of pyrethrum flowers. The following were the directions given the patient:

It is essential that the treatment should be followed exactly as set forth to obtain relief. Omission, or shortening of the time given, of either of the parts of the treatment, will accordingly delay relief.

Remove all clothing and change bed clothes. All should be laundered before further use.

First night:

1. Soak in a tub of hot water for 20 minutes.
2. Stand beside bath and soap yourself from head to toes, using any good soap, liquid preferred.
3. Re-enter bath, rinse off lather, and dry with a rough towel.
4. Apply ointment over entire body from neck to feet.

Second night:

Apply ointment over entire body.

Third night:

Repeat first night's bath and application of ointment.

Report to clinic on next regular day.

In most cases it is necessary to continue the treatment for a period of from 5 to 7 days, and in a few obstinate cases the use of the ointment was required for a longer time on resistant lesions.

Of the 618 cases, they found it necessary to continue the treatment into the second week in 88. They were very favorably impressed with the treatment, emphasizing the cleanliness, pleasant odor, and freedom from irritation with this method.

The toxicity of the various drugs used should be kept in mind. In general, sulphur is the least toxic for, although it will cause skin irritation if applied in too strong a concentration or over too long a period, constitutional effects are practically non existent. Absorption of balsam of Peru may set up a nephritis. A number of reports of allergy to pyrethrum have been reported and must be thought of in using this drug. Poisoning from absorption may also occur.

KONIMETER AVAILABLE FOR LOAN

The United States Naval Medical School possesses a konimeter, which is available for loan whenever dust surveys are contemplated.

The konimeter is a convenient instrument for investigating the dust content in workshops, streets, etc. By means of this instrument, for example, the observance of the regulations of public authorities can be facilitated, and liability to certain occupational diseases ascertained.

To use the instrument, a measured quantity of air is sucked by a pump through a jet past a circular glass plate, mounted to rotate. By the impingement of the fine jet of air upon the moistened plate the dust content of the air is abstracted. The area of the glass plate on which the dust has been deposited is then brought into the field of view of the microscope, and the nature and number of dust particles are determined. The attachment of a dark ground condenser often is of value in counting the dust particles.

The air pump is provided with a dust filter of sintered glass, the purpose of which is to retain very coarse dust particles with which measurement is not concerned. A squared micrometer is provided in the eyepiece of the microscope, for measuring the size of the dust particles. The size of the dangerous dust particles which engender silicosis is from 0.1 to 10 microns.

LYMPHOGRANULOMA INGUINALE IN THE NAVY NOMENCLATURE OF DISEASES

By Circular Letter No. 611, the Bureau of Medicine and Surgery made known to the Medical Department the decision of the Judge Advocate General of the Navy, approved by the Secretary of the Navy, February 27, 1936, holding lymphogranuloma inguinale to be of venereal origin. The same letter directs that this diagnostic

title be placed in its proper alphabetical sequence in class XII of Navy Nomenclature of Diseases and Injuries, as number 1212. This raises the sum of known venereal diseases to four, officially recognized in the nomenclature. The differentiation of all of these diseases is comparatively recent. It was not until 1838 that Ricord definitely showed that gonorrhea and syphilis were two separate clinical entities. In 1879 the causative relationship between the gonococcus and the gonorrhea was established by Neisser. Ten years later Ducrey discovered the bacillus of chancroid. In 1905 Schaudinn and Hoffman discovered the causative organism of syphilis In 1927 the Frei test was demonstrated as specific for the clinical syndrome of lymphogranuloma inguinale, subject to the limitations common to other such tests. Numerous workers were engaged upon the problem and, by 1933, when Stannus published his book on the subject, the establishment of the disease as a definite clinical entity and of venereal origin may be said to have been completed.

AVIATION MEDICINE

The increase of the air arm for both armies and navies and the great development of civil aviation gives to aviation medicine a new importance. Its position as a definite specialty of medicine is becoming more secure.

The need of the Medical Corps of the Navy for more flight surgeons was expressed in the July 1936 issue of the United States Naval Medical Bulletin. It may be said that probably as many as 25 new flight surgeons will be necessary during the next 3 years.

It is felt that the increasing importance and recognition of aviation and aviation medicine makes it a highly desirable specialty for young medical officers to enter. It must be pointed out that among other considerations a certain number of flight orders to flight surgeons have recently been authorized so that they will have the benefit of increased compensation when employed on duties involving a certain amount of actual flying.

ANNUAL MEETING OF THE ASSOCIATION OF MILITARY SURGEONS

The forty-fourth annual convention of the Association of Military Surgeons of the United States will be held October 29, 30, and 31, at Detroit, Mich. The Book-Cadillac Hotel will be the headquarters. An excellent program of military medical subjects has been arranged, and it is believed that all military medical men will enjoy and profit by the sessions. Col. Burt R. Shurly of the Medical

Reserve of the Army, is chairman of the convention committee, with Mrs. Shurly chairman of the ladies' committee. Lt. Comdr. J. E. Malcomson, Medical Corps, United States Navy, represents the Medical Corps of the Navy on the committee. A large attendance is anticipated from the Medical Corps and Dental Corps of the Army and Navy, the United States Public Health Service, Reserves, National Guard, and Veterans' Administration. Maj. Gen. Reynolds, the Surgeon General of the Army, expects to attend. As at past meetings, there will be a number of delegates from foreign countries. Mexico, Colombia, the Dominican Republic, Peru, Nicaragua, Canada, and Spain, are expected to send military medical representatives to the meeting from their armies, navies, air forces, militias, and reserves.

Of particular interest to all members of the association from the Navy is the fact that the Surgeon General of the Navy, Rear Admiral P. S. Rossiter, Medical Corps, now the first vice president of the association, will become the president in succession to Medical Director Charles M. Griffith, of the Veterans' Administration. Admiral Rossiter will be president of the Association of Military Surgeons of the United States during the period from October 1936 to the next meeting in 1937.

There has been a considerable addition to the membership from the Medical Corps of the Navy and Naval Reserve during the past year, and with the Surgeon General of the Navy as president of the association, it is expected that many new members will be received during 1936 and 1937. The association is in a very flourishing condition and with active chapters in a number of the larger cities.

AMERICAN BOARD OF OTOLARYNGOLOGY

The American Board of Otolaryngology has ruled that after January 1, 1937, all applicants for certification must pay the registration fee for the board before which they appear. Since this ruling affects men in Government service, both in the Army and in the Navy, the information is relayed.

THE FIRST INTERNATIONAL CONFERENCE ON FEVER THERAPY

The first international conference on fever therapy, originally scheduled for the end of September 1936, has been postponed because of numerous requests, to permit more time for the preparation of material. The new dates set for this conference are March 30 to April 2, 1937. The sessions will be held at the College of Physicians and Surgeons, Columbia University, New York City.

The advances in the treatment of gonorrhea, syphilis, and other diseases by pyretotherapy are of great significance. Invitations on behalf of the conference will be issued by the State Department of the United States to ministries of public health of other countries. The Medical Departments of the Army, the Navy, and the Public Health Service of the United States of America will be represented, as will also the New York City Departments of Health and of Hospitals.

Further information regarding the conference may be obtained from the General Secretary, Dr. William Bierman, 471 Park Avenue, New York City, United States of America.

BOOK NOTICES

Publishers submitting books for review are requested to address them as follows:

The Editor,
UNITED STATES NAVAL MEDICAL BULLETIN,
Bureau of Medicine and Surgery, Navy Department,
Washington, D. C.
(For review.)

SYPHILIS SIVE MORBUS HUMANUS. A RATIONALIZATION OF YAWS, SO-CALLED. FOR SCIENTISTS AND LAYMEN INTERESTED IN THE DAMAGE TO MAN FROM VENERAL DISEASES, *by Charles S. Butler, A. B., M. D., LL. D., Rear Admiral, Medical Corps, United States Navy. Member American Academy of Tropical Medicine, Fellow New York Academy of Medicine. President (1935–36) New York Society of Tropical Medicine.* The Science Press Printing Co., Lancaster, Pa. $3.00

Admiral Butler has long been known for his interest in the question of the unity of yaws and syphilis. For years he has been a decided protagonist of the unity of these conditions and has strongly advanced the view that yaws is merely syphilis modified by race and tropical conditions. In other words, he believes that the distinction between Treponema pallidum and Treponema pertenue is like that between tweedledee and tweedledum. Few men are better qualified than Admiral Butler to discuss the subject of syphilis and yaws. Years of duty in Haiti, the Virgin Islands, and in other parts of the Tropics have given him an unparalleled opportunity to observe these conditions. A specialist in tropical medicine, intensely interested in the subject, he has been a student of it for more than 30 years. The views of an earnest student with such large opportunities for clinical observation cannot but receive the most serious consideration from medical men.

Admiral Butler's book is not devoted entirely to the yaws and syphilis problem. As he says in the introduction, there are three things which the book aims to do. First, to give popular knowledge regarding the venereal diseases; second, to expose the fallacy of the theory that syphilis originated in the New World; third, to show the unity of so-called yaws and syphilis. One should not fail to read both the preface and introduction. In the first chapter of The Story of Syphilis is an extremely entertaining account of the history of that disease. Of great interest are the careful studies of the descrip-

tions of what were probably the initial lesions of syphilis given by Celsus and the Roman epigrammatist Martial. The quotations from Martial, given on page 14, are alone worth the price of the book. Of particular interest, too, is chapter 7, Argument, in which, by a series of questions and answers, the whole subject is reviewed. Another feature of the book is a glossary of the subject. The volume contains 137 pages and a number of splendid illustrations. It is handsomely bound and the paper and printing above any criticism. The last page of the book is as interesting as the first. The reviewer has recommended that the reader not fail to read the preface. He should also look at the back flyleaf, upon which is a picture of treponemas on a dark field surrounded by a phrase from Kipling's Recessional, "Lest We Forget."

MEDICAL MYCOLOGY—FUNGOUS DISEASES OF MEN AND OTHER MAMMALS, *by Carroll William Dodge, Ph. D., Mycologist, Missouri Botanical Gardens; Professor, Henry Shaw School of Botany, Washington University, St. Louis.* Illustrated. The C. V. Mosby Co., St. Louis, Mo., 1935. Price $10.

A volume which has recently appeared of which the preface is dated August 1, 1935, called Medical Mycology, by Dr. Carroll William Dodge, Ph. D., of Washington University, St. Louis, Mo., is one that has been long needed in American medicine.

Few diseases are of greater importance than those produced by molds. We meet such diseases in the human being all the way from the cradle to the grave. These diseases are of great importance not only on account of their primary inconvenience to the human being but also on account of their complicating affect upon other diseases. American medical science has for a century neglected this important field and for this reason Dr. Dodge's Medical Mycology is a credit to American science. Many of us, particularly those interested in tropical medicine, have felt that our National Institute of Health should have a department of medical mycology under the supervision of a competent mycologist. When you consider the fact that mycotic skin diseases alone are about as prevalent as tuberculosis; when you consider that wherever 100 men are gathered together perhaps 50 of them will show some evidence of recent or old mycotic affection, the importance of these diseases to the public health will at once be apparent.

This volume will undoubtedly supply a need and do it adequately. The systematic part of the book leaves nothing to be desired. The references are complete after each chapter. If there is any criticism of the book that might suggest itself to me it would be that Dr. Dodge had not included a chapter on the treatment of mycotic diseases. This I believe is customary with foreign books on mycology, such as Brompt and others. Dr. Dodge may have considered, however, that

books on treatment would handle this matter adequately. The references also would take care of this feature.

C. S. BUTLER.

PARENTERAL THERAPY, *by W. F. Dutton, M. D., formerly Medical Director, Polyclinic and Medico-Chirurgical Hospitals, Graduate School of Medicine, University of Pennsylvania, and G. B. Lake, M. D., Editor of Clinical Medicine and Surgery.* 376 pages, 90 halftones and line engravings. Charles C. Thomas, publisher, Springfield, Ill., and Baltimore, Md., 1936. $7.50.

Considering the extensive extra oral administration of drugs, it is remarkable that a book of this character has not appeared before as one has really been needed. Here are the answers to all the questions that arise about drug administration by other routes than the gastro-intestinal tract. The work covers all fields of parenteral medication dividing it, like all Gaul, into three parts: (1) Without penetration of the tissues by a needle (mucous membrane and skin absorption); (2) penetration of the skin by a needle; (3) through inhalation, or by injection of the lungs. There is a valuable therapeutic index, pharmacologic notes, and an appendix containing an index of manufacturers and their products.

ALLERGY OF THE NOSE AND PARANASAL SINUSES, *by French K. Hansel, M. D., M. S., Assistant Professor of Clinical Otolaryngology, Washington University School of Medicine.* 820 pages, 58 text illustrations, and 3 color plates. C. V. Mosby Co., St. Louis. 1936. $10.00.

"A Monograph on the Subject of Allergy as Related to Otolaryngology" is the secondary title Dr. Hansel has given his book, and it describes it well. It rather understates the scope however, for really it is almost a complete treatise on this subject, certainly the largest and most comprehensive book on the subject of allergy in English. It is a most valuable work for the physician dealing with allergy in all its clinical manifestations. Asthma, hay fever, urticaria, eczema, angioneurotic edema, and migraine are given the most careful consideration as regards diagnosis and treatment. Diets and menus are not merely outlined but printed in full. Indeed the sections devoted to treatment are particularly well written and would be of the utmost practical value to the general practitioner as well as to the specialist.

PEDIATRIC NURSING, by *John Zahorsky, M. D., Professor of Pediatrics, St. Louis University School of Medicine.* 568 pages, 144 illustrations in the text and 7 color plates. C. V. Mosby Co., St. Louis. 1936. $3.00.

This is a nursing manual so complete, up to date, and scientific as to be almost a practical manual of pediatrics.

ROENTGENOGRAPHIC TECHNIQUE. A MANUAL FOR PHYSICIANS, STUDENTS, AND TECHNICIANS, by *Darmon Artelle Rhinehart, A. M., M. D., F. A. C. R., Professor of Roentgenology and Applied Anatomy, University of Arkansas.* 431 pages, 183 engravings. Second edition. Lea & Febiger, Philadelphia. 1936. $5.50.

This is one of the best books on the subject of X-ray technique, giving as it does the scientific background and reasons for each procedure. The summaries of exposure factors for various parts of the body are most valuable. His chapter on miscellaneous instructions dealing with such subjects as the size of films, use of the diaphragm, number of exposures, identification of films, timing, stereoscopy, and terminology is very useful. At the end of the general instructions regarding technique each part of the body is taken up in considerable detail and typical positions for exposure shown. The illustrations and diagrams are excellent and the book is handsomely printed and bound.

DENTAL FORMULARY, by *Herman Prinz, A. M., D. D. S., M. D., Sc. D., Professor of Materia Medica and Therapeutics; The Thomas W. Evans Museum and Dental Institute School of Dentistry, University of Pennsylvania.* 366 pages. Fifth edition. Lea & Febiger, Philadelphia. 1936. $3.50.

This is a completely revised edition of this well-known dental formulary, a feature of which is that each recipe is basic and can be modified to suit individual cases.

EXAMINATION OF THE PATIENT AND SYMPTOMATIC DIAGNOSIS, by *John Watts Murray, M. D., Quincy, Mo.* Second edition. 1,219 pages. 274 illustrations. The C. V. Mosby Co., St. Louis. 1936. Price $10.00.

This is the second edition of a work originally published in 1926. That volume aimed to help in history taking and diagnosis by presenting "a large number of old-established facts in condensed and helpful form." The objective of the revised edition is to "deal mainly with the earliest stages of disease, prior to the occurrence of structural damage and at a time when treatment is most effective."

Section 1 is titled History-taking; section 2 encompasses diseases of single organs or systems of the body. In style of presentation the question-answer method is alternated with some discussions of disease entities and symptom complexes. In general the style is reminiscent of the State board quiz compends.

THE DIVISION OF PREVENTIVE MEDICINE

C. S. STEPHENSON, Commander, Medical Corps, United States Navy, in charge

TOXIC EFFECTS OF ARSENICAL COMPOUNDS AS ADMINISTERED IN THE UNITED STATES NAVY IN 1935 WITH SPECIAL REFERENCE TO ARSENICAL DERMATITIS

By S. S. COOK, Commander, Medical Corps, United States Navy, and E. H. WINGO, Chief Pharmacist's Mate, United States Navy

For the past 11 years medical officers of the Navy have been required to submit monthly reports to the Bureau of Medicine and Surgery of the number of doses of arsenicals administered and the reactions therefrom. This information, including that for 1934, has been compiled and published in the United States Naval Medical Bulletins of September 1925, January 1927, January 1929, July 1930, October 1931, October 1932, April 1933, October 1933, October 1934, January 1935, October 1935, and January 1936.

In table 1 are shown the number of doses of each arsenical administered in the year 1935, the reactions which occurred, and similar data for the 11-year period 1925–35. It is noted that in 1935 there was 1 reaction to 1,209 doses and 1 death to 64,726 doses. For the 11-year period 1925–35 there was 1 reaction to 1,292 doses and 1 death to 28,108 doses.

TABLE 1.—*Arsenicals, U. S. Navy, 1935 and 1925–35—type of drug, reactions, and ratio of doses to reactions*

	Number of doses administered	Reactions				Ratio of reactions to doses 1 to—	Ratio of deaths to doses 1 to—
		Mild	Severe	Fatal	Total		
Year 1935:							
Acetarsone	0	0	0	0	0	0	0
Arsphenamine	3,574	2	0	0	2	1,787	0
Bismarsen	634	0	0	0	0	0	0
Mapharsen	573	0	0	0	0	0	0
Neoarsphenamine	113,686	73	27	2	102	1,114	56,843
Silver arsphenamine	274	0	0	0	0	0	0
Sulpharsphenamine	5,315	1	1	0	2	2,657	0
Tryparsamide	5,397	1	0	0	1	5,397	0
Total	129,453	77	28	2	107	1,209	64,726
11-year period 1925-35:							
Acetarsone [1]	805	0	0	0	0	0	0
Arsphenamine	37,101	27	13	1	41	904	37,101
Bismarsen [2]	950	0	0	0	0	0	0
Mapharsen [3]	573	0	0	0	0	0	0
Neoarsphenamine	994,176	498	248	38	784	1,268	26,162
Silver arsphenamine [4]	501	0	1	0	1	501	0
Sulpharsphenamine	22,512	14	6	0	20	1,125	0
Tryparsamide	39,602	2	0	0	2	19,801	0
Total	1,096,220	541	268	39	848	1,292	28,108

[1] First administered during the year 1932.
[2] First administered during the year 1929.
[3] First administered during the year 1935.
[4] First administered during the year 1931.

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TABLE 2.—*Arsenical Reactions, U. S. Navy, 1935*

Classification	Cases	Deaths
Vasomotor phenomena	46	0
Arsenical dermatitis	40	0
Blood dyscrasias	9	1
Gastrointestinal	6	0
Liver damage (jaundice)	3	0
Acute renal damage	1	0
Vascular damage (probable adrenal hemorrhage)	1	1
Optic neuritis	1	0
Total	107	2

From table 2 it may be seen that the most frequent reactions are those that are classified as vasomotor phenomena and as arsenical dermatitis. Dermatitis in some form was observed in 37.38 percent of the cases. In 1934 dermatitis in some form was observed in 33.07 percent of the cases.

TABLE 3.—*Proportion of reactions of various types, 1929–35*

Classification	Number of reactions	Percent of total reactions
Vasomotor phenomena	284	46.41
Arsenical dermatitis	201	32.85
Blood dyscrasias	28	4.58
Table reactions [1]	26	4.25
Liver damage	24	3.92
Reactions of minor importance	17	2.78
Jarisch-Herxheimer	12	1.96
Gastrointestinal	9	1.47
Hemorrhagic encephalitis	4	.65
Arsenical neuritis	2	.33
Acute renal damage	1	.16
Border-line, hemorrhagic encephalitis	1	.16
Liver damage (doubtful reaction)	1	.16
Vascular damage (probable adrenal hemorrhage)	1	.16
Optic neuritis	1	.16
Total	612	100.00

[1] First classified during the year 1933.

In this article will appear a brief summary of the clinical history of each of the 40 cases of arsenical dermatitis.

ARSENICAL DERMATITIS

The 40 cases of arsenical dermatitis reported in 1935 were classified as 22 mild and 18 severe reactions. The type of lesion was exfoliative in 16 instances, urticarial in 10, macular in 8, erythematous in 5, and maculopapular in 1.

MILD REACTIONS

The 22 mild reactions occurred after the following number of injections:

Number of injections	Number of reactions	Number of injections	Number of reactions
2	4	27	1
3	3	38	1
4	1	46	1
6	2	47	1
7	1	52	1
8	1	58	1
13	1	79	1
21	1		
23	1	Total reactions	22

The interval between injection and appearance of symptoms varied from immediately after the injection to 3 days.

The length of time required for recovery varied from 42 minutes to 43 days.

A brief history of each case is cited.

Arsphenamine.—(1—1935) This patient experienced two mild reactions; the first was a gastrointestinal reaction which will be described in a later bulletin. The patient was given a diagnosis of syphilis on January 24, 1934, because of general adenopathy, constant sore throat, and a three-plus Kahn blood reaction. He received two courses of neoarsphenamine, for a total of 9.9 grams, and 38 injections of bismuth salicylate during the year 1934.

On January 10, 1935, the third course of arsenical treatment began with a 0.3 gram injection of neoarsphenamine. A 0.3 gram injection of arsphenamine was administered on January 17, followed by 0.4 gram injections on January 24 and 31. About 3 hours after the last injection, and after eating a sandwich, the patient became nauseated and vomited. He developed a mild gastrointestinal reaction with recovery in 4 hours.

Arsenical treatment was continued and he received 0.4 gram injections of arsphenamine on February 7, 14, and 21. Two and one-half hours after the last injection he complained of his skin burning, and five hours after the injection a light pink macular rash appeared over the extensor surface of arms and thighs, with a faint erythema over the abdomen. He received 1 gram of sodium thiosulphate intravenously. One hour later he became nauseated and vomited.

The following day the rash began to fade with recovery in 4 days.

Neoarsphenamine.—(2—1935) This patient experienced two mild arsenical dermatitis reactions in 1934 (cases 91 and 92—1934, U. S. Naval Medical Bulletin, October 1935). After the second reaction on January 15, 1934, arsenical compounds were discontinued. He received 16 injections of bismuth from January 22 to December 6, 1934.

On January 22, 1935, the fifth course of arsenical treatment began with 0.3 gram of neoarsphenamine, followed by 0.4 gram on January

29. Immediately after the last injection he became nauseated and vomited. Facies and conjunctivae were injected and pulse rapid. A generalized urticarial rash soon appeared. Red blood count, 5,360,000; white blood count, 10,600; hemoglobin 90 percent; band forms 51; segmented 41; lymphocytes 2; monocytes 6; blood platelets 155,500. He received 10 minims of adrenalin, followed in 5 minutes by 1 gram of sodium thiosulphate intravenously. The rash and injection of facies and conjunctivae subsided in 2 hours. Nausea persisted and he complained of slight headache. A second injection of sodium thiosulphate, 1 gram intravenously, was given.

The patient returned to duty the following day.

(3—1935) A patient who was infected in December 1934 began treatment on January 4, 1935, with a 0.3 gram injection of neoarsphenamine followed by a 0.6 gram injection on January 11. Ten hours after the last injection he was admitted to the sick list with chills, headache, and general malaise. Temperature 101° F., pulse 96, and respirations 18. This condition was regarded as acute catarrhal fever, as there were no definite indications of an arsenical reaction and because he had been receiving treatment for a cold for 4 days previous to admission.

On January 14, 3 days after the last injection of neoarsphenamine, he vomited and complained of general malaise and vertigo. Temperature 102° F., pulse 100, and respirations 20. The following day a fine maculopapular rash appeared over the entire body and extremities. Red-blood count, 4,400,000; white-blood count, 3,750; hemoglobin, 80 percent; band forms, 13; segmented, 43; lymphocytes, 22; eosinophiles, 10; monocytes, 12. He received 0.5 gram of sodium thiosulphate intravenously on January 15 and 0.8 gram intravenously on January 16. The following day the rash began to fade. Recovery in 10 days.

(4—1935) A patient was given a diagnosis of syphilis on November 12, 1934, because of the presence of *Treponema pallidum* in the ulcer on the right lateral coronary sulcus of his penis and a 4-plus Kahn blood test.

Arsenical treatment began on November 13, 1934, with a 0.3 gram injection of neoarsphenamine, followed by 0.4 gram injections on November 20, 27, December 4, 11, 18, and 25. As concurrent treatment he was given 7 intramuscular injections of bismuth salicylate and potassium iodide three times daily, the dosage varying from 10 to 50 grains.

On January 1, 1935, seven days after the last injection of neoarsphenamine, the patient complained of general malaise, pain in joints, and itching of the body. He was receiving treatment for a cold at this time and his condition was not considered to be an arsenical reaction.

Arsenical treatment was continued and on January 3 he received a 0.3 gram injection of neoarsphenamine, followed in 30 minutes by nausea and vomiting. The following day he complained of itching and loss of sleep. Roughening of the skin was noted but no definite rash appeared. He received 1 gram of sodium thiosulphate intravenously on January 5, 8, and 11.

The skin surfaces were very dry and a slight exfoliation was noted in the inguinal region.

Recovery in 9 days.

(5—1935) The source of infection in this case is unknown. The patient was given a diagnosis of syphilis on April 12, 1934, while under treatment in a naval hospital, because of repeated 4-plus Kahn blood tests.

From April 12 to November 21, 1934, he received 18 injections of neoarsphenamine (total amount not stated) and 16 injections of bismosol. On February 5, 1935, the third course of arsenical treatment began with a 0.3 gram injection of neoarsphenamine, followed by 0.4 gram injections on February 12, 19, and 26 and 0.3 gram on March 5. The last injection was diluted in 5 cubic centimeters of distilled water and injected in 1½ minutes. Immediately after the injection the patient developed nausea, his skin became suffused and itchy red blotches appeared on his face and body. He received 10 minims of epinephrine hydrochloride hypodermically. The urticarial rash disappeared with recovery in 42 minutes.

(6—1935) A patient developed a lesion on the coronal sulcus of his penis on March 22, 1935. A darkfield examination was positive for Treponema pallidum. Treatment began the same day with a 0.3 gram injection of neoarsphenamine. On March 30 the second injection of neoarsphenamine, 0.4 gram, was diluted in 10 cubic centimeters of sterile distilled water and injected in 3 minutes. Twelve hours later the patient was admitted to the sick list with chills, vomiting, temperature 101° F., and a generalized maculopapular rash over the entire body.

An erythematous rash developed with slight edema of the face and itching of the hands and feet.

Recovery in 6 days.

(7—1935) This patient was given a diagnosis of syphilis in July 1925 because of a positive blood reaction. From August 13 to October 8, 1925, he received 9 injections of neoarsphenamine (total amount not stated) and 30 mercury inunctions.

On January 7, 1929, he developed a lesion on his penis after exposure on December 28, 1928. A Kahn blood test was 4-plus.

From January 21, 1929, to April 30, 1934, he received 52 injections of neoarsphenamine, a total of 31.8 grams, and 14 injections of an arsenical compound (name and amount not stated). As concurrent

treatment he received 17 intramuscular injections of mercury, 25 injections of bismuth, and an unstated amount of mercury inunctions and mixed treatment.

On February 25, 1935, the tenth course of arsenical treatment began with a 0.3 gram injection of neoarsphenamine, followed by 0.45 gram injections on February 12, 19, and 26. Two days after the last injection the patient reported with edema of the mucous membranes of the mouth, lips, and uvula; swollen hands; and an urticarial rash over the flexor surfaces of arms and on the abdomen. There was no complaint other than itching. He received 10 minims of epinephrine hydrochloride by hypodermic injection followed by 1 gram of sodium thiosulphate intravenously. Recovery in 5 days.

(8—1935) The source of infection in this case is unknown. The patient had a history of a small boil on his penis after exposure in late 1931. He was given a diagnosis of syphilis in November 1933, because of a mild degree of general adenopathy and repeated 4-plus Kahn blood tests.

From November 11, 1933, to January 22, 1934, he received 9 injections of an arsenical compound (name and amount not stated) and 9 injections of bismuth and from November 4, 1934, to January 11, 1935, he received 8 injections of bismuth.

On February 25, 1935, the second course of arsenical treatment began with a 0.3-gram injection of neoarsphenamine, followed by 0.45 gram on February 1, and 0.5 gram on February 8, 14, 26, and March 9. Two days after the last injection he noted a rash on his arms which he thought was heat rash and did not report this condition. On March 14, 5 days after the last injection, he reported with a mild superficial dermatitis on forearms and abdomen. He received 1 gram of sodium thiosulphate intravenously on March 16, 2 injections of 1 gram each on March 17 and 18, and 1 gram on March 19. The patient had no complaint except itching and was not admitted to the sick list. The macular rash began to fade after 5 days and had completely disappeared in 25 days.

(9, 10—1935) Two reactions occurred in the same individual. A patient presented himself for treatment in October 1927, complaining of a rash on his chest and general malaise. Examination revealed a macular rash over his chest, enlargement of right inguinal glands, neurasthenic symptoms, a 4-plus Kahn blood test, and a history of a small venereal sore on his penis about 6 months before.

From October 13, 1927, to December 26, 1932, he received 5 courses, a total of 45 injections of salvarsan (total amount not stated), 24 injections of mercury, and 17 injections of bismuth. From November 20 to December 18, he received 6 injections of bismuth.

The sixth course of arsenical treatment began on April 23, 1935, with a 0.3-gram injection of neoarsphenamine. A few minutes later the patient had a measly flush on the abdomen, small of his back,

and medial aspect of thighs. The rash soon disappeared. He recovered in 2 days.

On April 30 he was given a 0.1 gram injection of neoarsphenamine and 5 hours later there was a recurrence of the urticarial rash over abdomen, chest, back, and thighs. He complained of itching, headache, chills, and muscular cramps in his legs. Temperature, 99.6° F.; pulse, 93; respirations, 20; blood pressure, 140/80; red-blood count, 4,270,000; white-blood count, 3,800; hemoglobin 80 percent; band forms, 38; segmented, 51; lymphocytes, 5; eosinophiles, 1; and monocytes, 5. He received 1 gram of sodium thiosulphate intravenously. The following day the rash had practically disappeared and the patient's only complaint was itching.

On May 5 the rash reappeared on chest, abdomen, and back but faded the following day. Recovery in 8 days.

(11—1935) A patient who was exposed on March 28, 1935, developed a typical chancre on the prepuce of his penis and inguinal adenopathy on April 8, 1935. Darfield examination was positive for Treponema pallidum on April 11, and a Kahn blood test was 2-plus on April 15.

Arsenical treatment began on April 11 with a 0.3 gram injection of neoarsphenamine, followed by 0.4-gram injections on April 16 and 19. Eight and one-half hours after the last injection he had chills. The following day he complained of general malaise and sore throat. Examination revealed a reddened pharnyx, a scarlatiniform rash over his chest, abdomen, back, and extremities, edema of eyelids, and injection of conjunctivae. He received 1 gram of sodium thiosulphate intravenously.

On April 21, 2 days after the last injection of neoarsphenamine, the erythematous rash began to fade. Recovery in 4 days.

(12—1935) The source of infection in this case is unknown. The patient was admitted to a naval hospital on March 2, 1932, with a diagnosis of dysentery. A blood test was 4-plus and a spinal fluid examination was positive.

From March 23, 1932, to November 24, 1933, he received 44 injections of neoarsphenamine, 16 injections of mercury, and 65 injections of bismuth. From May 15 to August 3, 1934, he received 12 injections of tryparsamide. During the months of October, November, and December he received malaria treatments.

On March 28, 1935, he was given a 0.3-gram injection of neoarsphenamine, the first injection of the seventh course of arsenical treatment. He became nauseated and vomited 5 minutes after the injection and 2 days later had generalized itching. He did not report his condition until the following week. A patch test made on April 4 was negative. Stroking of the skin produced no reaction.

He was given the second injection of 0.3 gram of neoarsphenamine on April 5. On April 11 he reported that itching had begun following a bath on April 6, and lasted 3 or 4 days. By this time a generalized urticarial rash appeared. He received 1 gram of sodium thiosulphate intravenously. The rash promptly faded but a faint icterus of conjuntivae was noted. He remained under observation until May 10, 1935, 43 days after onset of symptoms.

(13—1935) This patient (supernumerary female), who was probably infected in October 1935 by her husband, experienced 2 reactions during her first course of arsenical treatment. The first was mild and the second was a severe arsenical dermatitis which is described under case (28–1935).

She was given a diagnosis of syphilis because of a chancre on the cervix, sore throat, a papulosquamous eruption over the face, body, and extremities, general adenopathy, and a 4-plus Kahn blood test.

Treatment began on January 7, 1935, with a 0.2-gram intramuscular injection of thio-bismol. On January 9 she received a 0.3-gram injection of neoarsphenamine, followed by 0.45 gram on January 12. Three days later she was awakened from sleep by a severe itching of legs and hands. She did not report this condition.

On the morning of January 16, four days after the last injection of neoarsphenamine, the patient reported to the hospital with numerous scattered broad and flat papules having a slight umbilicated appearance as if some foreign body had penetrated the skin of the legs and hands. Each papule was surrounded by a pale halo. The lesions were inflamed in appearance and very pruritic. The possibility of a toxic reaction to neoarsphenamine was entertained and she was given a 0.2 gram intramuscular injection of thio-bismol.

January 17: Patch test shows no redness, papules, or vesicles. The skin lesions have spread to involve the thighs and a few lesions are present on the body.

January 18: The skin eruption is disappearing rapidly. Patch test negative. Recovered in 4 days after onset of symptoms.

(14–1935) This patient developed multiple herpetiform lesions of the glans penis and the foreskin after exposure on January 19, 1935. Repeated Kahn blood tests were 4 plus.

From February 26 to April 9, 1935, he received 6 injections of neoarsphenamine, a total of 2.35 grams, and 4 injections of bismuth salicylate. The last injection of 0.3 gram of neoarsphenamine was diluted in 10 cubic centimeters of distilled water and injected in 2 minutes. Five minutes after the injection the patient became nauseated, vomited, and complained of severe headache. A few minutes later a very definite urticarial skin eruption appeared.

Temperature, 102° F. Received 10 cubic centimeters of adrenalin subcutaneously.

Eight hours later the skin condition and temperature subsided with recovery in 24 hours.

(15–1935) This patient was given a diagnosis of syphilis on September 17, 1934, because of general lymphadenopathy, a 4-plus Kahn blood test, and history of a penile lesion on August 4, 1934.

From September 17, 1934, to January 7, 1935, he received 9 injections of neoarsphenamine, a total of 3.825 grams, and 4 injections of bismuth.

On March 31, 1935, he received a 0.225-gram injection of neoarsphenamine followed by 0.45 gram on April 6. Immediately after the last injection the patient complained of general malaise, headache, and a dull backache. Twenty-four hours later a macular rash appeared over the chest, abdomen, back, and flanks.

The rash promptly faded. Recovery in 2 days.

(16–1935) A patient was given a diagnosis of syphilis on June 12, 1933, because of generalized glandular adenopathy, 4-plus Kahn blood test, and history of a penile lesion on November 15, 1930.

From July 19, 1933, to September 1, 1934, he received 31 injections of neoarsphenamine, a total of 18.0 grams. As concurrent treatment he was given a total of 4.9 grams of bismuth salicylate.

On April 10, 1935, the patient began his fourth course of arsenical treatment. Neoarsphenamine was administered as follows: April 10, 0.3 gram; April 18, 0.45 gram; April 24, May 1, 8, and 15, 0.6 gram; and May 22, 0.3 gram. As concurrent treatment he was given 19 injections of thio-bismol, a total of 3.8 grams.

Immediately after the last injection of neoarsphenamine the patient complained of nausea and a warm glow over the entire body. Examination revealed marked puffiness of the face, especially the eyes, which were practically closed. The skin became purplish red with urticarial wheals over the face, body, and extremities. He received 7 minims of adrenalin intravenously, followed by 1 gram of sodium thiosulphate intravenously. The rash and all symptoms promptly disappeared and the patient was returned to duty the following day.

(17—1935) This patient reported to the sick bay on July 6, 1935, complaining of general malaise and a rash on his body and extremities. Examination revealed: Temperature 101° F., rash of secondary syphilis, enlarged lymphatic glands, and an almost completely healed lesion on his penis. A Kahn blood test was 4-plus.

Arsenical treatment began on July 6 with a 0.3-gram injection of neoarsphenamine, followed by 0.3 gram on July 13. Shortly after the last injection he complained of symptoms suggesting a cold in

his head. He was continued on the sick list and received routine cold treatment. Temperature 100° F.

July 16: Temperature remains over 100° F. He complains of a pain in his right shoulder. Adenopathy has increased and the rash is accentuated. Dickens' test was normal following neoarsphenamine.

July 20: Temperature normal. Urine normal. He received 0.3 gram of neoarsphenamine intravenously. Eight hours later he developed a temperature and an urticarial rash over his abdomen and buttocks. He received one-half cubic centimenter of epinephrine hydrochloride solution 1–1,000.

Two days later the rash began to fade with recovery in 7 days.

(18—1935) This patient developed a macular rash, lymphadenopathy and a right inguinal bubo on July 28, 1935. History of exposure in April 1935, but no history or scar of primary lesions was evident. A Kahn blood test was 4-plus.

On August 2, 5, 8, 11, and 15 he received 1 cubic centimeter intramuscular injections of bismosol. On August 6 he received a 0.3-gram injection of neoarsphenamine; on August 13, 0.5 gram; and on August 19, 0.7 gram. About 1 hour after the last injection the patient felt hot and developed pains in the calves of his legs. Three hours after the injection a macular erythema of the face, neck, and chest appeared. He received 5 cubic centimeters of adrenalin hypodermically, after which he vomited. One hour later he received 0.75 gram of sodium thiosulphate intravenously.

The following day the rash began to fade and he recovered in 2 days.

(19–1935) A patient was given a diagnosis of syphilis on September 20, 1935, because of a positive darkfield examination of a penile lesion and a 3-plus Kahn blood test. Arsenical treatment began immediately with a 0.3-gram injection of neoarsphenamine, followed by 0.45 gram on September 23, and 0.6 gram on September 26 and October 1. Two hours after the last injection the patient developed a headache with flushing of the face and a generalized macular rash. He received immediately 15 minims of epinephrine solution 1–1,000 subcutaneously. The symptoms and rash disappeared within 24 hours.

(20, 21—1935) This patient experienced three arsenical dermatitis reactions during the first course of arsenical treatment. The first two were mild and the third severe which is described under case (35—1935).

The patient developed an indurated ulcer on the mucous membrane of the foreskin of his penis on September 16, 1935. Repeated darkfield examinations were positive for *Treponema pallidum*, and a Kahn blood test was 4-plus.

From September 16 to October 8, 1935, he received six injections of neoarsphenamine and three injections of bismuth salicylate. Twenty-four hours after the last injection of neoarsphenamine he developed a slight temperature followed by a generalized macular rash. The symptoms and rash disappeared the following day. Recovery in 2 days.

On October 15 he received a 0.6-gram injection of neoarsphenamine. Within 24 hours the patient again developed a slight temperature and a generalized macular rash. The rash promptly disappeared and he recovered in 2 days.

Arsenical treatment was continued and the patient received 0.45 gram injections on October 22 and 29. Two days after the last injection he developed a severe exfoliative dermatitis. Recovery in 26 days.

(22—1935) A patient who was exposed on October 22, 1935, developed a chancre on his penis, macular rash of secondary syphilis, enlarged lymphatic glands, and a positive blood reaction on November 6, 1935.

Treatment between November 8 and December 1, 1935, consisted of 10 minims of potassium iodide three times daily.

On November 20 and December 3 he received 0.3-gram injections of neoarsphenamine, and on November 26 an injection of bismosol. Five hours after the injection on December 3 he developed a severe headache, swelling of the tongue, and generalized aching. Temperature, 101° F.; pulse, 112; respirations, 20; conjunctivae moderately injected and suffused; and a generalized urticarial rash. He received three-eighths grain of ephedrine hydrochloride by mouth, followed by 1 gram of sodium thiosulphate intravenously.

All symptoms including the rash disappeared within 11 hours.

SEVERE REACTIONS

The 18 severe reactions occurred after the following number of injections:

Number of injections	Number of reactions	Number of injections	Number of reactions
1	1	9	1
3	2	11	1
4	1	15	1
5	1	16	1
6	2	18	1
7	5		
8	1	Total reactions	18

The interval between the injection and appearance of symptoms varied from 1½ hours to 5 days.

Of the 18 severe reactions, recovery occurred as follows:

Interval between onset and recovery	Number of cases
0 to 15 days	3
16 to 30 days	3
1 to 2 months	3
2 to 3 months	6
3 to 4 months	0
4 to 5 months	2
5 to 6 months	1
Total cases	18

(23—1935) A patient who was infected on December 1, 1934, developed two ulcers on the shaft of his penis and general adenopathy. A darkfield examination of the ulcers was positive for *Treponema pallidum* on December 26, and repeated Kahn blood tests were 4-plus.

From December 26, 1934, to January 26, 1935, he received seven injections of neoarsphenamine and three injections of thiobismuth.

On January 29, 2 days after the last injection of neoarsphenamine, he noticed a generalized skin eruption, but did not report this condition until the following morning, at which time he had a generalized light red macular eruption involving the trunk, head, and extremities, with slight edema of the face.

The patient was admitted to a naval hospital on January 29, 1935, with a generalized rash, most prominent over the trunk, especially the back, with moderate itching. No signs or symptoms other than the rash. He received 1 gram of sodium thiosuphate intravenously on January 29, 30, and 31.

February 6: The rash has almost completely faded. The patient feels well. One gram of sodium thiosulphate intravenously.

February 10: A new type of skin eruption, vesicular in character with marked itching, appeared on his hands and forearms. His temperature is normal and there are no constitutional symptoms.

February 11: The entire body is covered with a severe acute vesicular dematitis, especially severe on the upper extremities. Temperature, 99.2° F.; pulse, 80; respirations, 18.

February 12: Not much change in his condition. Temperature, 100.5° F.; pulse, 85. Slight headache. Considerable weeping of lesions on the wrists and shoulders. White blood count, 12,950; band forms, 4; segmented, 50; eosinophiles, 1; lymphocytes, 20; monocytes, 1.

February 15: Considerable serum is discharging from the lesions on the wrists and shoulders. The arms and forearms are moderately involved, but the rash on the rest of the body is mild, with no vesicles and no weeping. The face, mouth, and throat are not involved. Temperature, 101° F.; pulse, 82. White-blood count, 14,050;

juveniles, 1; band forms, 5; segmented, 40; eosinophiles, 40; basophiles, 1; lymphocytes, 10; monocytes, 3.

February 19: The patient shows marked improvement. The skin is scaling and being shed rapidly. He is perspiring freely. White blood count, 13,000; juveniles, 6; band forms, 10; segmented, 50; eosinophiles, 14; basophiles, 1; lymphocytes, 16; monocytes, 3.

February 26: The patient continues to improve. The skin is clear except for slight scaling of the hands and wrists. White blood count, 6,000; juveniles, 3; band forms, 8; segmented, 40; eosinophiles, 19; basophiles, 1; lymphocytes, 20; monocytes, 3; Turk's cell, 1; myelocyte, 1.

March 5: The skin is normal. White blood count normal, eosinophiles have disappeared. The patient is gaining in weight and strength.. Recovery in 78 days.

(24—1935) A patient who denies having a primary lesion was given a diagnosis of syphilis on October 6, 1926, because of an inguinal bubo and a 4-plus Wassermann blood test. He received five injections of neoarsphenamine and five injections of mercury from October 7 to November 9. During this course of treatment he experienced a severe exfoliative dermatitis reaction, and neoarsphenamine was discontinued.

From March 4, 1932, to March 7, 1935, he received 58 injections of mercury and 25 injections of bismosol.

Notwithstanding the fact that the patient had been under treatment since 1926 with mercury and bismuth, he had maintained a persistently positive Kahn blood reaction. After due consideration and study, it was decided to give the patient a small trial dose of neoarsphenamine. A 0.3-gram injection was administered intravenously on April 16, 1935, and 18 hours later he reported with a diffuse redness of the skin and a temperature of 104.5° F. He was given 1 gram of sodium thiosulphate intravenously, 48 hours after which the redness began to fade. Moderate puffiness under the eyes and some swelling of the lips and ears remained. Red-blood count, 4,630,000; white-blood count, 18,050; band forms, 17; segmented, 78; lymphocytes, 3; eosinophiles, 2. Urine: Appearance, dark amber; reaction, strongly acid; specific gravity 1.023; albumin, sugar and microscopic negative. Received proctoclysis with Fischer's solution from 6 p. m. until 11 p. m., with 1 hour rest at 8 p. m.

April 19: Rash is fading and patient feels well, but in view of the rather severe skin manifestations and high temperature, 103.2° F., he is transferred to a naval hospital.

Patient admitted to hospital with desquamative, exfoliative dermatitis affecting the entire body, but especially marked over the face. Temperature, 102° F.; pulse, 88; resiprations, 22. The con-

dition gradually improved, and he was discharged to duty after 61 sick days.

(25—1935) This patient was given a diagnosis of syphilis on November 26, 1934, because of positive darkfield examination of a chancre on the dorsal surface of the prepuce and inguinal adenopathy. Repeated Kahn blood tests were negative.

Arsenical treatment began on November 29, with a 0.3-gram injection of neoarsphenamine, followed by a 0.4-gram on December 4, 0.6-gram on December 11 and 18, and 0.8-gram on January 8, 1935. A few hours after the last injection the patient was in a whaleboat with a landing party and became immersed and chilled. Nine hours after the injection he reported to the sick bay complaining of malaise, sweating, and a flushing sensation; temperature, 101° F.; pulse, 126; respirations, 20. He received 1 gram of sodium thiosulphate intravenously. It was believed that he had suffered chilling from exposure, and that symptoms were aggravated by neoarsphenamine. Uneventful recovery in 48 hours.

On January 11, 3 days after the injection, the patient noticed a rash on the ulnar surface of his right arm, and numbness of forearms and hands, but did not report his condition until January 21, by which time the rash had developed into raspberry colored erythemato-vesicular eruption involving the arms, chest, abdomen, and back. The patient had no complaint other than the rash which itched considerably. He received 1 gram of sodium thiosulphate intravenously, and 2 grams by mouth daily for 10 days.

February 8: The skin eruption is progressively worse, involving the trunk, neck, arms, hands, thighs, and back. The patient has no complaint other than moderate itching.

February 11: The patient shows some improvement but in view of probable long convalescence he is transferred to a naval hospital. Admitted to hospital with a desquamative dermatosis over trunk, neck, arms, thighs, and back, especially marked over the back. He complains of moderate itching and some limitation of motion of ankle and wrist joints.

February 17: Marked improvement of rash on chest and back.

February 20: He started a course of sodium thiosulphate, five intravenous injections of 1 gram each at 24-hour intervals. His condition gradually improved and desquamation continued until March 19, 1935, 67 days after onset of symptoms.

(26—1935) This patient for whom a severe arsenical dermatitis was reported in 1933 (case 62–1933, U. S. Naval Medical Bulletin, October 1934), at which time arsenical treatment was discontinued, received 64 intramuscular injections of bismosol between the dates of April 21, 1933, and March 12, 1935.

On April 30, 1935, he was given a 0.3-gram injection of neoarsphenamine, the first injection of the third course of arsenical treatment. The following day he vomited and developed a temperature and edema of the legs. He received 1 gram of sodium thiosulphate intravenously.

May 2: Temperature, 101° F.; pulse, 92, respirations, 18. White-blood count, 18,100; band forms, 12; segmented, 56; lymphocytes, 20; eosinophiles, 10; monocytes, 2.

The rash developed into a desquamative erythematous dermatitis. Recovery in 15 days.

(27—1935) This patient was admitted to the sick list with a sore on his penis on April 25, 1935, 12 days after exposure. Repeated darkfield examinations were negative for *Treponema pallidum* and Kahn blood tests were negative. On May 4, 1935, he developed a violent generalized erythema 6 hours after he was given a 0.3-gram provocative injection of neoarsphenamine. The erythema was still present on May 11 but the patient was returned to duty under treatment.

On May 14 the patient was admitted to a naval hospital with acute exfoliative dermatitis involving the head, face, neck, chest, and arms. There are a few small ruptured vesicles on the face and chest. His face, ears, and neck are slightly swollen. He received sodium thiosulphate intravenously.

May 21: The dermatitis involves the entire body. The face and neck are greatly swollen. Red-blood count, 5,600,000; white-blood count, 26,200; hemoglobin, 95 percent; segmented, 24; eosinophiles, 63; lymphocytes, 13. Urine: Positive trace of albumin, many white blood cells, much epithelium, few hyaline casts, and occasional granular casts.

May 23: Slight improvement. The patient is receiving colloid baths twice daily. White-blood count, 15,200; segmented, 45; eosinophiles, 23; lymphocytes, 29; monocytes, 3.

May 25: The dermatitis has definitely begun to subside. Swelling of face and ears decreasing. His condition gradually improved and desquamation continued until July 7, 1935. Recovery in 58 days.

(28—1935) This patient (supernumerary female) experienced the second attack of arsenical dermatitis during the first course of treatment. The first, a mild reaction, is described under case 13—1935.

The patient was given a diagnosis of syphilis because of a lesion suggesting a chancre on the cervix of the uterus, sore throat, papulosquamous eruption over the face, body, and extremities, general adenopathy, and a 4-plus Kahn blood test.

Treatment began on January 7, 1935, with a 0.2 gram intramuscular injection of thio-bismol. She received a 0.3-gram injection of neoarsphenamine on January 9 and a 0.4-gram injection on January

12. Three days later she developed a mild arsenical dermatitis and recovered in 4 days.

Treatment was continued with a 0.2-gram intramuscular injection of thio-bismol on January 16. On January 19 she received a 0.3-gram injection of neoarsphenamine and 1½ hours later complained of a severe headache, severe aching of all muscles, bones, joints, and post-orbital pains. The skin showed a diffuse, blotchy erythema over the entire body and extremities with moderate itching. She had a chill lasting one-half hour, followed by a temperature of 103° F. and pulse 120.

January 20: The patient had a fair night. Some aching of bones and joints, and headache continued. The rash has practically disappeared. Temperature, 100.2° F.; pulse, 120; respirations 25.

January 21: The patient is feeling better. Complaining of some aching of legs and hips. No dermatitis, icterus, liver damage, or headache. Temperature, 99° F.; pulse, 100; respirations 18.

January 23: Her condition is gradually improving. There is peeling of the superficial epidermis over the entire body following baths.

Blood picture

Date	White-blood count	Juveniles	Band forms	Segmented	Eosinophiles	Monocytes	Basophiles	Lymphocytes
Jan. 9, 1935	7,100		9	43	5	12		31
Jan. 16, 1935	7,200		9	50	3	11		27
Jan. 19, 1935	7,600	1	18	72		2		7
Jan. 20, 1935	7,800		8	46	9	6	1	30
Jan. 21, 1935	4,150	1	9	33	16	3		38
Jan. 22, 1935	4,750		9	29	16	7		39
Jan. 23, 1935	5,100		4	31	13	9		43
Jan. 24, 1935	4,400	2	4	31	12			51
Jan. 26, 1935	7,200		4	44	8	9	2	34
Jan. 28, 1935	11,850			37	11	6	4	42
Jan. 29, 1935	10,650		3	47	7	8	5	30

Treatment.—Fischer's solution by proctoclysis, 1,000 cubic centimeters in 3 days, January 19, 20, and 21. One gram of sodium thiosulphate intravenously at 3 p. m. and 4:30 p. m., January 19. Forced fluids and fruit juices. Codeine for pain. One dram of sodium bicarbonate every 4 hours. Lime water and olive oil to skin and careful nursing.

Recovery in 12 days.

(29—1935) A patient who was exposed on December 23, 1934, was given a diagnosis of syphilis on February 13, 1935, because of a typical chancre on his penis, a 4-plus Kahn blood reaction, general adenopathy, and a macular rash.

He began treatment on February 11, 1935, and received 8 injections of neoarsphenamine, a total of 4.4 grams, and 1 injection of bismuth between that date and March 27, 1935. On March 28, one day after

the last injection of neoarsphenamine, he complained of itching. Examination showed the beginning of a fine papular variety of dermatitis on the flexor surface of his arms. Close questioning of the patient revealed that he remembered a slight itching following the sixth injection of neoarsphenamine but noticed no rash at that time. The dermatitis developed slowly with periods of improvement but became generalized. He received 5 grains of sodium thiosulphate three times daily by mouth.

April 13: The dermatitis is much more active. The patient shows a papulo-vesicular rash over his entire body, with some weeping and considerable desquamation. Mild constitutional symptoms.

April 15: The patient was admitted to a naval hospital, his entire body covered with a erythematous macular rash. Chains of glands enlarged. The mucous membrane of the eyes, ears, nose, and throat are injected. Blood pressure 110/62.

April 20: The patient complains of inability to rest due to itching. The rash on the face and elbows is becoming eczematous.

April 30: Symptoms are less severe. The patient gets considerable relief from colloid baths.

May 10: The patient feels well. The rash is clearing but he has severe callouses on the soles of his feet and the palms of his hands.

Recovery in 64 days.

(30—1935) On December 3, 1932, patient developed a firm indurated area along the pendulous urethra with generalized lymphadenopathy. No history of a chancre other than the intraurethral lesion. Repeated Kahn blood tests were positive.

From December 3, 1932, to January 6, 1933, he received 4 injections of an arsenical compound (name and amount not stated), 3 injections of bismuth, and 10 minims of potassium iodide 3 times daily. From February 8 to July 5, 1933, he received 16 injections of bismuth salicylate.

From March 5 to April 16, 1935, he received seven injections of neoarsphenamine (3.3 grams) as the second course of arsenical treatment. Three hours and forty minutes after the last injection he complained of severe itching over the entire body. Examination revealed a brick red rash over the entire body and head and considerable edema of the eyes, ankles, and feet.

The condition gradually developed into a severe desquamative exfoliative dermatitis.

Recovery in 63 days.

Treatment.—He received magnesium sulphate 1 dram every 15 minutes for 10 doses on April 17, and sodium thiosulphate 1 gram intravenously, each morning and evening, on April 17, 18, 19, 20, 21,

and 22. Warm sodium bicarbonate baths followed by boric acid ointment.

(31—1935) A patient who was infected in April 1935 was given a diagnosis of syphilis on May 16, 1935, after repeated darkfield examinations of a sore on his penis were positive for *Treponema pallidum*. Arsenical treatment began on May 16 with a 0.4-gram injection of neoarsphenamine, followed by 0.6-gram injections on May 18 and 21, a total of 1.6 grams or an average of 266 milligrams per day. The patient states that 2 days after the second injection of neoarsphenamine he noticed a fine papular rash on the inner side of his arms and legs.

On May 24, 3 days after the last injection, the patient complained of severe headache and aching of limbs and body and stated that the headache began 10 hours after injection. Temperature 104° F.; pulse, 110. No skin rash or shock. Urine negative. Symptomatic treatment.

May 25: A marked scarlatinform rash appeared involving the face, body, and limbs. Temperature, 101.2° F.; pulse, 90; respirations, 20; and a faint erythematous rash over the entire body. He complains of slight headache, nausea, and vomiting. The eyes and pharynx are injected. Acne on back and chest. Red-blood count, 4,540,000; white-blood count, 6,800; hemoglobin, 90 percent; juveniles, 8; band forms, 35; segmented, 37; lymphocytes, 20; monocytes, 5.

His condition gradually improved, and the rash began to fade on May 27, but his eyes continued to have some injection until recovery on June 10, 1935, 17 days after onset of symptoms.

(32—1935) This patient, in whom a severe arsenical dermatitis was reported in 1933 (Case 53—1933, U. S. Naval Medical Bulletin, October 1934), was reinfected after exposure on June 1, 1935. One month after exposure he developed three indurated ulcers in the coronal sulcus of his penis and bilateral inguinal adenopathy. Repeated darkfield examinations of the ulcer were positive for *Treponema pallidum*.

After the severe reaction in 1933 arsenical treatment was discontinued. The patient received ten injections of bismuth salicylate between the dates of May 9 and July 14, 1933.

On April 17, 1934, he reported to the sick bay complaining of a skin rash, which was later diagnosed prickly heat. To duty after 15 sick days. On January 24, 1935, he was admitted to the sick list with diagnosis eczema. To duty after 36 sick days.

On July 12, 1935, he was given a 0.3-gram injection of neoarsphenamine, first injection of the second course of arsenical treatment. Eight hours later he developed a generalized exfoliative dermatitis which terminated in desquamation and recovery in 14 days.

Treatment.—One-gram intravenous injections of sodium thiosulphate were administered on July 15, 16, 17, 19, 21, 23, and 26. Colloid baths and olive oil on July 19.

(33—1935) A patient who was exposed on April 12, 1935, developed a sore on his penis which healed rapidly leaving an indurated nodule on the foreskin. The nodule disappeared under treatment. The following month he reported to the sick bay with a faint macular rash, left inguinal bubo, and generalized adenopathy. A Kahn blood test was 4-plus.

From May 29 to July 31, 1935, he received 10 injections of neoarsphenamine, a total of 5.4 grams, and 10 injections of bismuth as concurrent treatment.

The second course of arsenical treatment began on August 22, 1935, with a 0.3-gram injection of neoarsphenamine, followed by 0.45 gram on September 4, and 0.6 gram on September 12, 19, 26, and October 3. The last injection of 0.6 gram was diluted in 12 cubic centimeters of distilled water and injected in 2 minutes. Six hours later he developed a headache and general malaise which he did not report at that time. On October 6, 3 days after the last injection he noticed itching but did not report this condition until the following morning at which time an examination revealed a generalized macular eruption over the entire body and slight thickening of the skin, especially of the scrotum.

The patient developed a severe weeping exfoliative dermatitis which terminated in desquamation and recovery in 37 days.

Treatment.—One gram intravenous injections of sodium thiosulphate were administered daily for 4 days; sodium phosphate, 3 grams daily; sodium bicarbonate, grains 15, three times daily for 3 days; and bicarbonate of soda baths twice daily and calamine lotion as indicated.

(34—1935) A patient who developed an indurated chancre on his penis after exposure on February 16, 1935, was given a diagnosis of syphilis on March 11 because of positive darkfield examination.

Arsenical treatment began on March 13 with a 0.2 gram injection of neoarsphenamine, followed by 0.3 gram on March 16, 0.6 gram on March 20, 23, 30, and April 6, a total of 2.9 grams or an average of 120 milligrams per day. Several hours after the last injection the patient complained of being hot, then chilly, and later developed a headache. The following day he noticed a fine red rash over his body, most pronounced over the lower extremities, with considerable itching. He also noticed his feet were sore. The rash soon disappeared and the patient felt well for the remainder of the week.

On April 13 he received a 0.3-gram injection of neoarsphenamine diluted in 20 cubic centimeters of distilled water and injected very slowly. Several hours later he had a chill, general malaise, and headache, followed by a generalized rash with severe itching. He received 1 gram of sodium thiosulphate intravenously.

The skin condition increased in intensity and developed into a severe exfoliative dermatitis with marked edema of the eyelids. Recovery in 62 days.

(35—1935) This patient experienced the third arsenical dematitis during his first course of arsenical treatment. The first two were mild reactions (cases 20, 21—1935). He was given a diagnosis of syphilis on September 16, 1935, because of positive darkfield examination of an indurated ulcer on his penis and a 4 plus Kahn blood test.

From September 16 to October 29, 1935, he received 9 injections of neoarsphenamine, a total of 3.9 grams, and 7 injections of bismuth salicylate as concurrent treatment. The mild reactions occurred after the sixth and seventh injections of neoarsphenamine, with recovery in 2 days after the injection.

Two days after the last injection of neoarsphenamine on October 29, he noticed his skin was "blotchy" and the following day began to peel as though from sunburn. There was no apparent dermatitis until November 2, at which time an examination revealed considerable desquamation of the skin surface over the entire body.

November 4: Red-blood count 4,420,000; white-blood count, 9,000; hemoglobin, 82 percent; band forms, 1; segmented, 53; lymphocytes, 22; monocytes, 6; basophiles, 1; eosinophiles, 17; blood platelets, 250,000. He received sodium thiosulphate 1 gram intravenously.

November 5: Generalized exfoliation of the entire body. There is no inflammation of the underlying skin and no vesicles, fissuring, weeping, or excoriations. No tremor or ataxia. Sense of touch diminished on hands and feet. Red blood count, 4,450,000; white blood count, 7,600; hemoglobin, 80 percent; segmented, 48; lymphocytes, 33; monocytes, 4; eosinophiles, 15; icterus index, 9.

November 26: The skin appears normal except for fine branny scaling of the face and hands. Sense of touch in hands normal. Red-blood count, 4,500,000; white-blood count, 10,000; hemoglobin, 80 percent; lymphocytes, 41; eosinophiles, 2; monocytes, 2.

Recovery in 26 days.

(36—1935) A patient who was infected on January 2, 1932, was given a diagnosis of syphilis on February 25 because of enlarged inguinal glands and a positive darkfield examination of a sore on his penis.

From February 25 to March 31 he received 6 injections of neoarsphenamine. Seven days after the last injection he developed a

severe arsenical dermatitis, from which he recovered in 57 days. Arsenical treatment discontinued.

From April 12, 1932, to November 5, 1935, he received 15 cubic centimeters of bismuth salicylate and 56 cubic centimeters of bismosol.

On November 13, 1935, he received a 0.05-gram injection of neoarsphenamine, the first injection of the second course of arsenical treatment. Fourteen hours after the injection, which was diluted in 20 cubic centimeters of distilled water and injected in 2 minutes, he complained of headache and nausea. Red-blood count, 3,405,000; white-blood count, 3,250; hemoglobin (Tallqvist), 70 percent; neutrophiles, 54; lymphocytes, 12; transitionals, 3; eosinophiles, 3; band forms, 26. He received a 1-gram intravenous injection of sodium thiosulphate.

November 18: The patient has a generalized scarlatiniform rash with slight edema of the eyelids. Temperature 101° F.; pulse, 98; respirations, 20. Red-blood count, 3,970,000; white-blood count, 4,600; neutrophiles, 54; lymphocytes, 14; monocytes, 3; transitionals, 7; eosinophiles, 6; band forms, 16.

The patient developed a generalized desquamative exfoliative dermatitis. Recovery in 17 days.

(37—1935) A patient who was exposed on June 10, 1935, developed a lesion on the prepuce of his penis which was positive for *Treponema pallidum* on June 25, 1935. A Kahn blood test was negative on June 25 but was 3-plus on August 5.

From July 1 to September 27, 1935, he received 11 injections of neoarsphenamine, a total of 4.5 grams, and 8 injections of bismosol, and 5 injections of bismuth salicylate as concurrent treatment.

On September 30, 3 days after the last injection of neoarsphenamine, an exfoliative dermatitis appeared involving the scalp, chest, abdomen, and extremities. The rash is moderately severe but associated with healing syphilitic pustules and staphylococcus infection of these lesions and of old scabies lesions. He received 1 gram of sodium thiosulphate intravenously on September 30 and October 1.

October 3: The patient's condition is gradually improving. He received 1 gram intravenous injections of sodium thiosulphate on October 3, 5, and 6.

October 31: Liver extract and force feeding begun and continued until recovery.

November 4: Subcutaneous abscesses behind the lobes of both ears requiring drainage.

November 11: The skin has thickened and begun to scale. The face and scalp are now the worst areas, having a tendency to formation of small pustules under the skin.

November 16: Large abscesses on the calf of the left leg and in the left submaxillary area, requiring drainage.

November 18: Icterus index, 5. Red-blood count, 4,100,000; white-blood count, 24,500; hemoglobin, 85 percent; lymphocytes, 16; eosinophiles, 7; mature, 71; bands, 6. Autenrieth's test negative. Blood culture negative in 48 hours.

November 26: Autenrieth's test negative. Red-blood count, 4,190,000; white-blood count, 21,500; hemoglobin, 85 percent; lymphocytes 24; eosinophiles, 2; mature, 66; bands, 8; icterus index, 4.

December 11: The patient is much improved, and is up and about. Some scattered subcutaneous abscesses persist about the neck and face.

January 6: General condition much improved. Red-blood count, 4,720,000; white-blood count, 15,050: lymphocytes, 25; eosinophiles, 4; mature, 46; bands, 22; monocytes, 3; icterus index, 6.

January 27: Red-blood count, 4,850,000; white-blood count, 9,900; hemoglobin, 90 percent; lymphocytes, 27; eosinophiles, 10; mature, 55; bands, 4; monocytes, 4.

Treatment.—Daily cleaning of the skin with soap and water. Careful daily cleaning of the furuncles and proper drainage. Recovery in 127 days.

(38—1935) This patient developed a chancre on his penis and mild lymphadenopathy after exposure on July 28, 1935. A Kahn blood test was 3-plus on September 24.

Arsenical treatment began on October 15 with a 0.3 gram injection of neoarsphenamine, followed by 0.45 gram injections on October 22, 29, and November 5. The patient states that 5 days after the last injection he noticed a mild discoloration of his forearms which he did not report. Neoarsphenamine was continued and he received 0.4 gram injections on November 12 and 19, after which the dermatitis became more generalized. Examination showed a generalized erythematous rash of the face and extremities, slight lymph gland enlargement, no constitutional symptoms.

He received sodium thiosulphate intravenously, 5 injections of 1 gram each, at 24-hour intervals.

The rash began to fade on December 4, 1935, and desquamation ceased on January 7, 1936. Recovery in 68 days.

(39—1935) The source of infection in this case is unknown. The patient was given a diagnosis of syphilis on August 16, 1935, because of repeated 4-plus Kahn blood tests.

Arsenical treatment began on August 26 with a 0.3-gram injection of neoarsphenamine, followed by 0.6-gram injections on September 5, 10, 17, 24, October 1, and 7. As concurrent treatment he was given 6 injections, 1 cubic centimeter each, of bismuth salicylate.

Twelve hours after the last injection of neoarshpenamine he noticed a generalized flush accompanied by itching. He reported to the dispensary the following morning by which time the rash had developed into a widely distributed discrete punctate maculo-papular eruption.

October 11: The eruption persists unchanged except for slight excoriation due to scratching.

October 15: The rash has grown progressively worse and the patient states that itching prevents sleep. There is a typical early arsenical dermatitis present over the arms, shoulders, trunk, legs, and the tips of the ears. Slight generalized adenopathy.

October 17: Dermatitis progressing. Temperature, 99.8° F.; red-blood count, 4,380,000; white-blood count, 7,450; hemoglobin, 90 percent; bands, 8; segmented, 56; lymphocytes, 20; monocytes, 8; eosinophiles, 8.

October 20: Exfoliation beginning. Temperature 101.2° F. Red-blood count, 4,330,000; white-blood count, 10,050; hemoglobin 80 percent; bands, 2; segmented, 68; lymphocytes, 20; monocytes, 4; eosinophiles, 6.

October 23: His condition is unchanged. Exfoliation progressing. Edema of the face and eyelids present. Red-blood count, 3,770,000; white-blood count, 12,900; hemoglobin, 85 percent; bands, 10; segmented, 18; lymphocytes, 17; monocytes, 2; eosinophiles, 50; basophiles, 3.

October 30: There is exfoliation of the skin along lines of cleavage of skin, especially the palms and neck. He has early morning chills of short duration. No other symptoms. Red-blood count, 3,770,000; white-blood count, 20,200; hemoglobin, 80 percent; bands, 4; segmented, 23; lymphocytes, 6; monocytes, 7; eosinopiles, 60.

November 13: The skin is clearing slowly. Edema of the lower eyelids persists. Early morning chills continue. Red-blood count, 4,990,000; white-blood count, 12,450; hemoglobin, 85 percent; bands, 14; segmented, 34; lymphocytes, 13; eosinophiles, 33; monocytes, 5; basophiles, 1.

November 18: Edema of the eyelids increasing. The skin is weeping and oozing a light-colored serum. The patient's mental state is somewhat disturbed. Marked exfoliation of the skin over entire body.

December 9: Periodic oozing of light-colored serum from skin of the limbs and ear lobes. Chills less frequent.

December 23: The patient has developed a deep abscess in lower third, inner aspect of the right thigh. Incised and drained with evacuation of 50 cubic centimeters of pus. Smear for organisms showed Gram positive diplococci.

The patient's condition gradually improved and exfoliation continued until March 7, 1936. Recovery in 172 days.

(40—1935) This patient was exposed on October 28, 1935, and 8 days later developed 4 indurated ulcers on his penis.

On November 14 he was admitted to a naval hospital with diagnosis chancroid, penis, by which time two of the ulcers had practically healed and showed definite white indurated elevated margins. A darkfield examination of the serum from these margins showed *Treponema pallidum.*

Arsenical treatment began on November 19 with a 0.35-gram injection of neorsphenamine, followed by 0.7-gram injections on November 22, 26, December 3, 10, 17, and 24. Five injections of bismuth salicylate were given as concurrent treatment.

On December 29, five days after the last injection of neoarsphenamine, he noted a rash around his ankles and on the back of his neck. The following morning the rash had spread to involve the abdomen and arms. No other symptoms or complaint.

On December 31 he reported for the eighth injection of neoarsphenamine, at which time examination showed a delayed scarlatiniform rash with pin-point vesicles, involving the face, trunk, and arms, most marked over back and chest. There is slight edema of the eyelids and the throat is slightly reddened.

January 1: The rash is somewhat accentuated, especially over the face, ears, and neck, where a slight edema is present. A patch test shows increased vesiculation and small purpuric spots where neoarsphenamine solution was in contact. White-blood count, 7,100; neutrophiles mature, 55, immature, 2; eosinophiles, 12; basophiles, 1; lymphocytes, 28; mononuclear transitional, 2. He received 1 gram of sodium thiosulphate intravenously. Temperature 99° F.

January 2: The rash is about the same. He received 1 gram of sodium thiosulphate intravenously.

January 3: The patient's face is considerably swollen and his eyes are nearly shut. Temperature remains over 100° F.

January 4: The rash is deepening in severity, and the vesicles about the face appear to be infected. White-blood count, 10,250; neutrophiles mature, 74, immature, 12; lymphocytes, 12; eosinophiles, 2.

The rash about the face is taking on the character of exfoliative dermatitis, and there is some vesiculation and oozing of the external surface of the arms.

January 6: His face is soaked. Superficial layers of epidermis softened and removed and oil treatment applied.

January 7: Edema of the face is subsiding. The redness has increased in the rash on his back and chest. Redness and sore-

ness of his throat is severe. A temperature ranging from 100° F. to 103° F. has been noted for the past week.

January 8: Edema of the face and skin is subsiding, though considerable redness is still present. The fingertips show many small purpuric spots. The phase of chemical reaction is considered passing and persistence of fever is probably due to secondary bacterial dermatitis.

January 9: The patient shows no improvement. His temperature ranges from 100° to 102.5° F. His ears are treated daily. Blood: Erythrocytes, 3,200,000; hemoglobin, 78 percent; neutrophiles mature, 62, immature, 24; lymphocytes, 10; eosinophiles, 2; mononuclear-transitional, 2.

January 11: The skin is desquamating in large peels. The patient is kept comfortable by daily applications of petrolatum to entire skin area.

January 15: There is a fresh crop of small superficial pustules in the pores of his face and considerable induration of superficial cervical glands. His hands are very tender. The soreness in his throat is subsiding. Morning temperature is 103° F.

January 19: Several small incisions in softening superficial lymph glands of his neck, which probably became infected from the ears and skin of the face. The external ear is considerably swollen and crusted. The throat is hyperemic but no swelling or ulcers have appeared. The patient is placed on the serious list.

January 21: The soft suppurating glands in the anterior cervical chains and left axilla are opened daily and drained.

January 23: The patient is feeling much better. The swelling in his face is subsiding. Ultraviolet light applications to the skin of his ears.

January 25: The patient is improving slowly. His temperature is slightly above 100° F. Opening and drainage and suppurating glands through small incisions.

January 27: The patient feels very well, except for occasional itching. The swelling of his ears and external canals are subsiding considerably. Less drainage in opened abscesses.

January 31: The patient is improving slowly. He complains of considerable itching at night. He is removed from the serious list.

The patient gradually improved. The hair on his scalp and eyelashes grew out and his skin returned to normal 137 days after the onset of symptoms.

Treatment.—He received two 1-gram intravenous injections of sodium thiosulphate on January 3, February 5, 6, and 7 and 1-gram injections on February 8, 10, 11, 12, 13, 14, 15, 17, 18, 19, 20, 21, 22, 24, 27, 28, 29, March 2, 3, 4, and 5. X-ray exposure to lower legs and

face for 2 minutes on February 5. Cold quartz orificial applicator 30 seconds one-fourth inch into ear canals, and body applicator 30 seconds 5 inches to external ears daily for 10 treatments.

SUMMARY

In 1935 naval medical officers administered 129,453 doses of arsenicals and reported the occurrence of 107 reactions therefrom. Of these reactions, more than one-third (40) were arsenical dermatitis; a ratio of 1 case of dermatitis to 3,233 doses.

Of interest in connection with the etiology of arsenical dermatitis is the number of instances in which premonitory signs were noted. These signs are repeated below and serve to indicate the necessity for careful examination and questioning of each patient before administering an arsenical.

Case 1.—A mild gastrointestinal reaction developed after 24 injections of an arsenical compound. The 27th injection, given 21 days later, caused macular dermatitis.

Case 4.—This patient complained of general malaise and itching after the seventh injection. Nine days later exfoliative dermatitis followed the eighth injection.

Cases 9 and 10.—This patient developed urticarial dermatitis 3 hours after the forty-sixth injection. The following injection, given 7 days later, caused recurrence of the urticaria.

Case 12.—Five minutes after receiving the fifty-seventh injection of an arsenical compound, he felt nauseated and vomited. Urticarial dermatitis followed the next injection which was administered 8 days later.

Cases 13 and 28.—Urticaria followed the second injection, and erythematous dermatitis followed the third injection administered 7 days later.

Case 17.—General malaise and fever followed the second injection and urticarial dermatitis developed after the third injection administered seven days later.

Cases 20, 21, and 35.—Unreported slight temperature followed each of the first six injections. The day after the sixth injection a macular rash appeared. Seven days later an injection caused recurrence of the macular dermatitis. Exfoliative dermatitis followed the ninth injection which was administered 14 days later.

Case 24.—Severe exfoliative dermatitis after fifth injection on October 30, 1926. Eight and one-half years later he received the sixth injection of an arsenical compound and again developed a severe exfoliative dermatitis.

Case 31.—Two days after the second injection a fine macular rash appeared, and 3 days later an injection caused erythematous dermatitis.

Case 32.—Exfoliative dermatitis in 1933 after seventh injection. Syphilitic reinfection in June 1935. Severe exfoliative dermatitis followed an injection given 2 years and 5 months later.

Case 34.—Chills, headache, and a light red rash over the entire body followed the sixth injection, and 7 days later the seventh injection caused exfoliative dermatitis.

Case 38.—Unreported mild discoloration on his forearms appeared 5 days after the fourth injection. An injection given 2 days later caused a generalized rash. Exfoliative dermatitis developed after the sixth injection, given 7 days later.

ABSTRACT FROM THE ANNUAL SANITARY REPORT OF THE U. S. S. "TENNESSEE" FOR THE YEAR 1935 [1]

The venereal problem.—There were 100 admissions for venereal disease during the year, classified as follows:

Gonococcus infection, urethra	78
Syphilis	15
Chancroidal infection	7
Total	100

The incidence of venereal disease aboard this vessel is gradually being reduced, as the following statement will show. During the year 1933 there were 175 admissions for venereal disease; 131 during 1934; and 100 during the year 1935. These figures are offered to show that some progress is being made, but the results in the campaign for the control of venereal disease on the U. S. S. *Tennessee* have been extremely discouraging and disappointing. Chemical prophylaxis will never control venereal infection, but I am firmly convinced that mechanical protection plus chemical protection will practically eliminate venereal disease from the Navy. However, the difficulty lies in getting cooperation in complying with these sensible, easy, and reasonable precautionary measures. It is felt that a certain percentage of men on this ship are strictly complying with my five fundamental principles in venereal prophylaxis, yet to be really successful you must get 100 percent cooperation. In any group of 1,100 healthy young men there will always be a certain minority who are reckless and foolhardy and will not comply with any system of prophylaxis. It is this class of men who swell the venereal lists. Nevertheless I am confident that the great majority of men throughout the Navy who are contracting venereal disease are a distinct credit and asset to the Service rather than a detriment. Therefore any regulation promulgated for the discharge of men simply because they contract venereal disease is not only unfair but also unwise and distinctly prejudicial to the best interests of the naval service. In order to obtain some definite, practical, and valuable data on this moot subject I carried out the following plan during the calendar year 1935. A special form was prepared in every case of admission for venereal disease. This form shows the name, rate, length of service, diagnosis, date of admission, age, habits, education, and prior venereal history. In addition, the division officer under whom the man was serving was required to submit pertinent remarks under the following captions, "Aptitude", "Desirability", "Char-

[1] Submitted by Commander L. D. Arbuckle, Medical Corps, U. S. Navy.

acter", and to further revaluate the merits of the man by checking him in one of the following groups, "Outstanding", "Above average", "Average", "Below average", "Definitely inferior." Many people assume that it is only the raw recruit who contracts venereal disease. The more seasoned enlisted man is supposed to take greater interest in his health and comply more strictly with well-recognized prophylactic methods. However the compilation of figures shows that for the 100 cases under consideration the average length of service was over 3 years—39 months to be exact. Now as to habits. It is a well-established fact that indulgence in alcohol is one of the most important contributory factors in the spread of venereal disease. In my campaign for the control of venereal disease I have appreciated this fact and have made "Avoid excessive indulgence in alcohol" my first fundamental principle. In checking up on the cases of venereal disease for the year it is noted that 95 percent admit indulgence in alcoholic beverages; five individuals stated that they never used alcohol in any form.

The next point of interest is the educational background of those contracting venereal disease. Many self-styled authorities lead one to believe that the man who exposes himself to venereal infection is an individual of little education and inferior mentality. The basic education of the 100 cases of venereal disease under consideration is as follows:

3 percent have had 2 years of college work.
35 percent are high-school graduates.
9 percent have completed 3 years of high-school work.
20 percent have completed 2 years of high-school work.
10 percent have completed the first year at high school.
3 percent have completed the seventh grade at grammar school.

Now it is further contended that the man with venereal disease is a general nuisance and his division officer would be glad to get rid of him as he is of inferior quality and incapable of development. Let us see what the division officers of the *Tennessee* have to say with reference to the 100 cases of venereal disease under consideration:

22 percent are classified as outstanding.
35 percent are classified as above average.
39 percent are classified as average.
4 percent are classified as below average.

Not a man out of the 100 is placed in the "Definitely inferior group." I believe that the above classification establishes conclusively that the men who are contracting venereal disease are certainly not detrimental to the naval service. It has been thought by many in the Navy that a man could possibly be excused for his first admission to the venereal list but that "repeaters" should be considered as "definitely inferior" and unfit for further retention in the naval service.

Let us examine this group of 100 men and see how many times each man has been admitted for venereal disease during his entire naval service.

	Percent
First admission	60
Second admission	27
Third admission	11
Fourth admission	2

It may be interesting also to know that the two men who show four admissions for venereal disease are placed by their division officer in the "Outstanding group." I believe that this tabulation tends to show rather convincingly that the retention, advancement, or discharge of a man in the Navy should be based on his aptitude and ability to perform the duties of his rating and not be influenced by his venereal history. To keep all officers and men advised of the prevalence of venereal disease among our personnel a memorandum addressed to all divisions is prepared on the first of each month showing the percentage and total number (names are not given) of men admitted for venereal disease in each division during the past month. This venereal story is read to all divisions by division officers and then posted on all bulletin boards. A copy is submitted to the executive officer and commanding officer for their information.

REPORT OF AN OUTBREAK OF SCARLET FEVER ON BOARD THE U. S. S. "PENNSYLVANIA"

There was a mild epidemic of scarlet fever in the ships visiting the Puget Sound Navy Yard during January, February, and the early part of March 1936.

The most extensive outbreak occurred on board the U. S. S. *Pennsylvania* between January 28 and March 8, when a total of 48 cases were admitted. The peak of admission was reached on February 10 when nine cases were admitted.

According to a report received from the U. S. S. *Pennsylvania* dated March 8, "The source of the original infection is not positive. However, the patient stated that he had been attending the yard moving-picture theater where men from various other activities congregated.

"Predisposing factors are numerous: (*a*) A large percent of nonimmune individuals; (*b*) a fertile field of numerous upper respiratory infections, such as catarrhal fever, tonsillitis, and pharyngitis; (*c*) ship's movement from a warm to a cold climate; and (*d*) unsatisfactory sanitary conditions of the ship due to overhaul.

"The mode of transmission has been difficult to determine, as no mess or division has produced a simultaneous number of infections.

On the other hand the admissions have come sporadically from different divisions, which indicate that transmission has been from man to man.

"Methods of control have been rigidly instituted: (*a*) Immediate isolation of suspects; (*b*) transfer of all strongly suspected individuals; (*c*) repeated inspections of divisions or messes where cases have occurred; (*d*) sterilization of mess gear and thorough cleansing of mess tables; and (*e*) Dick tests have been done on members of the crew under 30 years of age. Approximately 25 percent of men tested have been positive, varying from a faint to a strongly positive reaction. Dick positive mess cooks have been replaced by Dick negative individuals, and all other Dick positive food handlers have been inspected daily.

"The large number of other pharyngeal conditions of a minor nature has practically nullified the results to be expected from routine physical inspection. Only a few cases have been detected before the appearance of a fever or a rash. The results of the Dick test has been used satisfactorily in sick-bay management of the patients and suspects and is to be recommended for future use in epidemic control."

The following discussion regarding the prevalence of scarlet fever is abstracted from Public Health Reports of March 13 and April 10, 1936:

"For the country as a whole, 29,134 cases of scarlet fever were reported for the current 4-week period, January 26–February 22, which was the highest incidence for this period in recent years. The West North Central and the Mountain and Pacific regions, where the disease has been unusually prevalent, reported a decline from the preceding 4-week period, but the number of cases was 2.3 and 1.5 times, respectively, the figures for those regions for the corresponding period last year, and in each region the incidence was the highest in the 8 years for which data are available.

"For the 4 weeks ended March 21 there were 35,318 cases reported, which was an increase of approximately 10 percent over the figure for the corresponding period in 1935 and more than 40 percent over the number in 1934. The high incidence was still confined to the West North Central region, where the number of cases (5,902) was about 2.4 times that for the corresponding period last year, and to the Mountain and Pacific sections where the incidence (4,770 cases) was about 14 times that of last year."

AN OUTBREAK OF FOOD POISONING CAUSED BY CREAM PUFFS

An outbreak of food poisoning occurred on board the U. S. S. *Saratoga* on the afternoon and evening of May 12, 1936.

The following account is based on a report submitted by Commander W. J. C. Agnew, Medical Corps, United States Navy, senior medical officer of the ship.

Only men who partook of the noonday meal on May 12 were affected. The menu for this meal was beefsteak en casserole, boiled potatoes, Waldorf salad, fresh squash (boiled), bread and butter, and cream puffs, the suspected foods being beefsteak and cream puffs.

Of the 255 members of the crew who were mildly affected, 68 were placed on the Binnacle List for May 13 because of diarrhea and vomiting and returned to duty on May 14.

Investigation of the galley and the process of preparing this meal revealed the following facts:

"(*a*) *Beef:* Received from the U. S. S. *Bridge* on April 16, 1936; broken out of icebox during the afternoon of May 10, 1936, and allowed to thaw in butcher shop; cut into steaks during the morning of May 12, 1936, cooked shortly afterward and served for the noon meal the same day. When the meat was cut into steaks, it still had ice on the bone.

"(*b*) *Cream puffs:* Puffs baked the day before serving. Contents of cream puffs prepared during the afternoon of May 11, 1936, by cooking lemon preparation with powdered milk, eggs, corn starch, and flavoring extract. This mixture was brought to a boil and cooked about 15 minutes in a steam cooker. It was then removed from this container at 1800 (May 11) in a mixing bowl and placed in a dough-mixing trough in the bake shop. It was allowed to remain in this trough overnight and during the forenoon of May 12 was removed from this trough and placed in the cream-puff casings. The dough-mixing trough is galvanized iron, as are also the mixing bowl and the steam cooker. The galvanizing of all containers appears intact, except the mixing bowl, in which the mixture remained approximately 15 minutes, which shows area where the galvanizing has worn off.

"The first symptoms of this epidemic appeared from 2½ to 7 hours after the noon meal. The attack started with nausea, vomiting, pain in epigastrium, and severe diarrhea. Eight cases showed marked prostration but were entirely recovered within 48 hours. The treatment consisted of the administration of a large dose of sodium bicarbonate in a glass of water to induce vomiting, and followed later by castor oil and camphorated tincture of opium. In four cases morphine sulphate was administered for muscle cramps. If the castor oil was not retained, double Seidlitz powders were given when vomiting ceased.

"The medical officer is of the opinion that the cream-puff mixture was the cause of the epidemic and this was due to keeping it at the bake-shop temperature (approximately 95° F.) for 15 to 16 hours.

"Specimens of the vomitus from 3 cases, two specimens of cream puffs and two specimens of steak were sent to the U. S. S. *Relief* for bacteriological examination."

The following is the official report from the U. S. S. *Relief* upon the examination of specimens submitted by the U. S. S. *Saratoga:*

"Cultures were made from the following specimens submitted for bacteriological examination and the results recorded as follows:

(*a*) Stomach contents_____ Escherichia Communior (B. coli communior).
(*b*) Cream puffs_____ Do.
(*c*) Meat specimen_____ No growth.

"Guinea pigs fed with specimens of cream puffs and meat caused no symptoms of illness.

"*Conclusions.*—Food contaminated by Colon Bacilli and not by the Salmonella Enteritidis group."

Information and regulations governing the production of custard-filled bakery products in the State of New Jersey are abstracted herewith from Public Health News, June 1936, Department of Health, State of New Jersey:

"Reviewing records of food poisonings following the eating of bakery products containing custard, we find over two thousand persons affected, three fatally, all showing the same symptoms developing from 2 hours to 36 hours after partaking of these foods.

"Edwin O. Jordan and William Burrows say, 'The use of custard medium or the addition of starch to ordinary medium favors the production of the enterotoxic substance * * * certain bacteria that had never yielded enterotoxic filtrate in previous laboratory tests gave positive results after they had been transferred on starch medium.'

"Dr. Breed points out the increase in germ growth when sugar and milk is added to the medium.

"From custards unquestionably guilty of carrying the causative agent of illness, proven by common incidence in multiple cases in epidemics, bacteriological examination in every investigation has shown a very high count of some of the more common bacteria.

"We believe that toxins developed by a luxuriant growth of these organisms rather than a direct infection to be the active agent. It has been demonstrated by heroic volunteers that a culture with a heavy infection of these bacteria grown on a custard medium when eaten give the identical clinical picture of the field outbreaks.

"In two of the instances reported, careful investigators have traced the bacteria found in the victims and in the food product carrier to its point of origin; in one case a lesion on the neck of a worker and in another to rats which infested the bakery.

"In other epidemics displaying the identical symptoms following the eating of custard products, we find reports as follows:

"Examined for arsenic—arsenic suspected—resembles lead poisoning—chemical examination negative—no botulism—ptomaine suspected—no causative agent located. From this evidence both positive and negative, it appears conclusive that the cause of custard-borne illness is bacteria either by direct infection or by production of toxin, and that the proper angle of preventive regulation is the bacteriological.

"We have collected in sterile container at time of preparation, custard which, properly cared for, when delivered to the laboratory 19 hours later, showed but 100 colonies of bacteria per gram, after exposure to room atmosphere and temperature for 24 hours it showed 800,000 per gram, after 48 hours, 2,000,000. It was with these thoughts uppermost that our regulations governing the preparation, distribution, and sale of custard and cream-filled bakery products were formulated and adopted.

"*Regulation No. 1.*—All commercially prepared custard or cream-filled bakery products shall be made under clean conditions.

"*Regulation No. 2.*—Only pasteurized milk or cream shall be used in the preparation of custard and cream-filled bakery products.

"*Regulation No. 3.*—The temperature and time of heating the mix shall be as a minimum the equivalent of a temperature of 142° F. for a period of not less than 30 minutes.

"*Regulation No. 4.*—Upon completion of the cooking of the mix, it shall be immediately transferred into previously sterilized containers, properly covered, and chilled without delay to 50° F. or below and maintained at such temperature until used.

"Now we meet with arguments as to practicability—completely cooled custards are too stiff for use in machine for filling puffs and eclairs. The common practice has been to pour the cooked custard into shallow pans and allow to partially cool exposed to bakery atmosphere and possibly contamination. It is at this point probably that the sickness producing agent is most often introduced.

"One enterprising baker has attempted to meet this problem with a flat pan constructed with a water jacket; another uses a draft of air from an electric fan. Our suggestion is to put the custard in a covered container and almost completely immerse in cold water, running water where available, thus getting the custard to the proper temperature for filling the pastry in the shortest time, then fill, and refrigerate the finished product.

"*Regulation No. 5.*—The filling apparatus shall be of suitable impervious material and shall be cleaned and properly sterilized before each use, no cloth filling bags shall be used.

"It is planned to add to this regulation a definite requirement that filling machines be completely taken apart for sterilization. In a

great many cases the filling apparatus has not been taken apart daily and machines were in use that evidently had not been thoroughly cleaned for a long time, thus providing a very likely source of germ infection. There are no arguments against cleanliness, as for filling bags—inexpensive paper bags are available; these can be discarded after using.

"*Regulation No. 6.*—The hands of employees engaged in the preparation of custard and cream-filled bakery products shall not touch the custards or cream fillings. Observation confirms the need for this prohibition."

EPIDEMIC AMEBIC DYSENTERY—THE CHICAGO OUTBREAK OF 1933[1]

Because of the national interest of this epidemic, and because it presents many lessons in preventive medicine, this general summary and conclusions are reprinted here from the National Institute of Health Bulletin No. 166. Medical officers of the Navy may not have ready access to this health bulletin, and it is believed the reprinting in the Naval Medical Bulletin will make available to them some of the features of this epidemic which may be of particular value to them.

GENERAL SUMMARY AND CONCLUSIONS

1. *Epidemiology.*—An epidemic of amebic dysentery had its origin in Chicago during the summer and fall of 1933. It was the first recognized water-borne outbreak, and the only known extensive epidemic of this disease in a civilian population.

2. During the period of the epidemic, June 1 to December 31, 1933, there were approximately 8,500,000 out-of-town visitors to Chicago, with resulting unusual congestion of downtown hotels and public eating places.

3. Chiefly involved in the epidemic were two neighboring large downtown hotels, here designated "X" and "Z." They had in part a common water supply.

4. During the epidemic period approximately 160,000 persons had contact with these hotels. Slightly over one-half were registered guests, including approximately 300 who resided there more or less permanently. Some 2,300 were employees, and the remainder visited the hotels for meals, beverages, or both.

5. During the period June 1, 1933, to June 30, 1934, evidently incomplete reporting brought to light a total of 1,409 cases. Slightly more than two-thirds of those infected were out-of-town visitors to Chicago. Approximately 75 percent had had contact with one or

[1] Reprinted from National Institute of Health Bulletin No. 166 by permission of the Surgeon General, U. S. Public Health Service.

both of the hotels, but less than one-half of the infected Chicago residents reported such contacts.

6. Out-of-town residents who developed this infection following exposure in Chicago usually became ill after returning to their homes rather than in the city of exposure, thus making difficult the early recognition of the epidemic. Amebic dysentery so acquired was reported from over 400 cities scattered in 43 States, the Territory of Hawaii, and three Canadian Provinces.

7. There was a prolonged interval, averaging over 3 months, between exposure to infection and the report of illnesses to the Chicago Board of Health. To varying degrees this was due to: (*a*) An incubation period which, compared with other epidemic diseases, was somewhat prolonged; (*b*) the general lack of concern for diarrheal disorders and the consequent delay in seeking medical counsel; (*c*) difficulties in diagnosing a clinical entity with which the physicians were not generally familiar; (*d*) the early lack of suspicion, or evidence, that the widely scattered, sporadically appearing cases originated in Chicago.

8. In mid-August attention was first called to the possibility of amebic infections originating in one of the hotels by the simultaneous report of 2 cases involving registered guests at one of them. By the end of the month 7 others had been reported. In September 2 more, involving guests, 1 from each of the hotels, came to light. In addition, an intensive study of employees of one hotel revealed several (11 officially reported) infections in that group.

9. The first out-of-town infections were reported to the board of health after mid-October, but reports on almost all of this group resulted from the circularization then begun of those who had been registered guests at these hotels and the widespread publicity, early in November, relative to the epidemic.

10. There is reason to believe that the recognition of the existence of the epidemic would have been delayed even longer than it was, save for the unusual and effective means taken to discover and disclose the facts.

11. Only one focus was discovered which accounted for any considerable number of cases, namely the two hotels. The similarity of the epidemic period to that of A Century of Progress Exposition suggested that the epidemic was related to conditions at the exposition, but there was no evidence to support this view.

12. The general incidence of infection was higher in one of the hotels (X) than in the other (Z) and markedly so for the early weeks of the epidemic.

13. The infections were spread within the hotels from about June 1 to December 31, 1933, with few cases originating after early Novem-

ber. Except at the beginning and end of the epidemic period, infections were being spread almost continuously, but with marked variation in the risk of exposure at different times.

14. There was a particularly high incidence late in June, during the last half of August, and early in October. Of those exposed during certain of the peak periods as high as 5 percent acquired the disease. Relatively brief exposures in one convention group resulted in a case rate of not less than 8 percent.

15. In general, the more prolonged the exposures at the hotels the greater was the hazard of infection. Of the guests who lived at the hotels for 30 days or more, slightly over 10 percent became infected, while one-half to 1 percent of the other registered guests acquired the disease. Employees also were commonly infected.

16. The excessively high rate of clinical dysentery among those exposed to possible infestation stands in marked contrast to previous observations of this infection. We suspect that this was chiefly related to repeated exposures to massive doses of the infecting organisms. Possibly also a particularly pathogenic organism was concerned.

17. Ten percent of those apparently infected within the hotels and who reported concerning places patronized for meals gave no record of eating at either of the two hotels. Almost one-half of these specifically denied eating in the hotel in which they stayed. All these presumably drank water there.

18. Approximately 20 percent of the cases apparently originating in the hotels were multiple infections in the same family or group. Evidence strongly indicates that these were concurrently acquired.

19. There was but one reported instance in which it seemed highly probable that a secondary infection originated from an individual who acquired the disease in Chicago.

20. The age, occupation, and socio-economic status of the hotel guests infected corresponded with those of visitors to these two hotels.

21. The sex distribution of those infected at the hotels agreed closely with that of hotel guests and employees as a whole. No evidence of variations in sex susceptibility was detected.

22. The incidence of carriers was high among employees of the two hotels (37.8 percent and 47.4 percent).

23. The repeated examinations of employees indicated that infestations with *Endameba histolytica* were taking place during the period of the epidemic.

24. In one of the hotels the incidence of carriers was found to be much higher among employees who commonly worked on the upper floors which were supplied with water subject to contamination in the other hotel. In contrast there was a relatively low incidence of

carriers among those who ordinarily worked where the quality of the water was unquestioned.

25. Vigorous measures directed to the prevention of the spread of infection by carriers among food handlers were ineffective.

26. The opinion is held that the principal if not the sole means of spread of this epidemic was through water polluted within one of the hotels. A contributing factor was the progressively increasing number of carriers of *E. histolytica* among the employees and probably also among the guests who remained for more than the average stay of transients. Thus any pollution of the water with hotel sewage would become progressively more hazardous.

27. The existence of amebic dysentery in Chicago, not traced to these two hotels, is also recognized but neither the source or sources nor the modes of spread of these nonhotel infections have been satisfactorily determined.

1. *Engineering.*—The two hotels involved in the epidemic, while among the older ones in Chicago, were well patronized by reason of a long-established reputation. An unusual service relationship existed between these hotels in that one (hotel Z) was in part supplied with electricity, steam, and water from the other (hotel X).

2. The sewers in both hotels were overloaded to an unusual degree during the period of the epidemic. This was due to the high guest occupancy of rooms and also occasionally to excessive rainfall.

3. In providing modern conveniences, especially in connection with plumbing, drainage loads were placed on sewer lines in excess of their safe carrying capacities. This was especially true of one gravity sewer in the basement of hotel X, with which the two principal points of possible pollution of the water supply common to both hotels were associated.

4. The two major points of possible pollution which are considered to have resulted in water-borne infections in the two hotels were:

(a) Two cross-connections in hotel X which joined an overhead sewer to condenser-water discharge pipes. This water which had been first used for cooling purposes was distributed throughout hotel X and to the upper floors of hotel Z. The pollution of this would account for the observed parallelism of the incidence of infection in the two hotels.

(b) An old rotting wooden plug in an overhead sewer which permitted leakage into the cooled drinking-water tank below. This would account for infections among guests and patrons in hotel X only because this water system was limited to that hotel.

5. Both sources of pollution probably were operative intermittently during the period of the epidemic, the influencing factors causing

peaks of infection being (*a*) overloading of sewers, (*b*) restriction of sewer capacities due to flooding of the city sewers during heavy rainfall, (*c*) conditions surrounding the operation of the condensers, and (*d*) probably the use of the cross-connections for disposal of condenser water through the sewers.

6. The leak over the cooled drinking water tank probably was the principal cause for high infections among guests at hotel X on June 29 and 30, 1933, due to pressure in that hotel's sewer, accompanying the flooding of city sewers during and following a short period of excessive rainfall.

7. The infections attributed to the cross-connections appear to be related, especially in August and September, to the intermittent operation of one of the condensers in hotel X.

8. Flooding of a portion of the basement of hotel X occurred during an unusual storm in the early morning of July 2, 1933, from a break in two sewers under an ice-storage room. This temporarily created a potentially hazardous situation, but does not appear to have contributed to the spread of amebic infection; in fact, the relief of pressure in the sewers due to the rupture was a fortunate occurrence, since it probably prevented a repetition of the pollution of the cooled water which apparently occurred 3 days previously.

9. While other potential health hazards were found by detailed inspection at both hotels, they are not considered to have contributed to any considerable degree, if at all, to the epidemic.

Clinical.—1. The acuteness of onset and the gravity of symptoms in many cases and the shortness of course in fatal infections were striking.

2. The outbreak gave an unprecedented opportunity to study the incubation period. This proved to be much shorter than was usually believed. Most commonly it was about 2 weeks. In 59 percent it ranged from 1 to 4 weeks. Symptoms occasionally appeared within 1 week, and in a few cases only after 3 to 4 months.

3. Outstanding in this epidemic were the delays, uncertainties, and difficulties in diagnosis. Prior to the general knowledge of the occurrence of a widespread epidemic not more than about 20 percent of the illnesses were correctly diagnosed.

4. The absence of the characteristic diarrhea, and also the difficulty in some cases of finding *E. histolytica*, often proved misleading.

5. An unusual clinical observation in this series was the occurrence of fever in many cases. Even in uncomplicated infections it sometimes was relatively high.

6. An acute onset, unusual for amebic dysentery, which was often associated with abdominal pain and tenderness and fever, frequently led to an erroneous diagnosis of a surgical condition. The illness

was diagnosed as appendicitis in at least 3.6 percent of all cases, in 16.3 percent of all fatal cases, and 28.6 percent of the fatal cases in which erroneous diagnoses were reported. This was particularly serious since in 32 cases, according to reports received, the fatality rate following appendectomy was over 40 percent.

7. The usual complications of amebic dysentery were observed. Liver involvement was diagnosed in 6.1 percent, and liver abscess in 3.9 percent of all cases on which we have clinical data. The frequency of this complication afforded evidence in support of the nature of this epidemic.

8. The pathological findings in general agreed with those previously reported for this disease.

9. There were 98 deaths reported, a case fatality rate of 7 percent. Amebic dysentery was diagnosed in two-thirds of these at autopsy or in retrospect.

10. The most common erroneous diagnosis in fatal cases was malignancy, chiefly of the rectum.

11. The response to early and adequate antiamebic therapy was striking. In cases so handled prompt subsidence of symptoms and recovery were the rule.

Control.—1. Efforts were made to control the outbreak by the elimination of carriers of cysts of *E. histolytica* from among the food-handling staffs, but there is no evidence that these efforts were successful. The experience in this respect is not to be regarded as an indication either of the value or lack of value of the procedure under some other conditions.

2. The measures required to prevent the recurrence of such an epidemic are: (*a*) Effective supervision of the installation of plumbing in new buildings and of changes in old ones; (*b*) reasonably frequent inspections of the water and sewage systems of buildings, especially of the older ones; (*c*) particular attention to the elimination of hazardous cross-connections, through preventing their installation and through detecting and removing existing ones.

3. Institutions serving the public, particularly those providing residence, meals, or beverage, should be encouraged, aided, and required to provide adequately for the protection of the public health. Properly trained sanitarians should more commonly be included in the personnel of such organizations.

HEALTH OF THE NAVY

The following tables are summaries of morbidity rates per 1,000 for the first quarter of 1936 in comparison with rates for the corresponding quarter of the preceding 5 years:

ENTIRE NAVY

Year	All diseases	Injuries	Poisonings	All causes	Communicable diseases		Venereal diseases
					A	B	
1931	533	40	0.20	592	(1)	(1)	121
1932	428	37	.07	465	(1)	(1)	125
1933	319	35	.44	354	(1)	(1)	92
1934	405	55	.75	461	13	118	88
1935	428	62	.65	490	21	147	90
1936	335	42	.34	377	10	130	43
FORCES ASHORE							
1931	643	39	0.20	682	(1)	(1)	74
1932	407	37	.10	444	(1)	(1)	75
1933	319	35	.44	354	(1)	(1)	92
1934	537	61	.47	598	22	182	54
1935	505	66	1.14	572	41	191	50
1936	441	47	.65	488	12	188	29
FORCES AFLOAT							
1931	502	40	0.17	542	(1)	(1)	147
1932	440	36	.06	476	(1)	(1)	152
1933	292	35	.29	326	(1)	(1)	116
1934	342	53	.89	396	8	87	105
1935	386	60	.38	447	10	124	111
1936	285	40	.17	326	9	100	56

Common infectious diseases of the respiratory type.—A total of 6,650 cases of the common infections of the respiratory type was reported from the entire Navy, 5,431 of which were catarrhal fever.

There were 2,730 admissions for these diseases reported by all shore stations in the United States during the quarter, of which 818 were notified by the United States Naval Training Station, Norfolk, Va., 409 by the United States Naval Training Station, San Diego, Calif., 336 by the United States Naval Academy, Annapolis, Md. (midshipmen), 222 by the United States Naval Training Station, Newport, R. I., 132 by the United States Naval Air Station, Pensacola, Fla., and 125 by the United States Naval Training Station, Great Lakes, Ill. Acute catarrhal fever constituted 84 percent of these admissions.

The epidemic at the Naval Academy was mild in character. The majority of cases admitted at Norfolk were confined to recruits and were "strongly suggestive of influenza, particularly the aftermath of weakness noticeable at times." While not extremely severe in nature the cases admitted in January and February showed a tendency to be accompanied with higher temperatures and to convalesce less rapidly. A more severe type were admitted in March, requiring more transfers to hospital. The increased number of admissions at Great Lakes during the month of March was due to climatic conditions and the prevalence of these diseases in the surrounding territory.

A total of 315 cases of these diseases was reported by shore stations outside of the continental limits of the United States, as compared with 106 for the previous quarter. The greatest numbers of cases were reported as follows: Fourth Marines, Shanghai, China, 85; Fleet Air Base, Pearl Harbor, Territory of Hawaii, 58; Marine Detachment, American Embassy, Peiping, China, 54; and the Navy Yard, Pearl Harbor, Territory of Hawaii and the Fleet Air Base, Canal Zone, 27 each.

There were 3,605 admissions for respiratory diseases reported by all ships during the first quarter, catarrhal fever being responsible for 2,872. Ships reporting the largest numbers of cases were:

Ship	January	February	March	Total
Ranger	85	26	15	126
Lexington	76	22	17	115
Nevada	32	49	26	107
Saratoga	34	47	17	98
New Mexico	31	38	18	87
Arizona	16	51	18	85
California	46	14	18	78
Arkansas	40	12	20	72
Colorado	37	25	8	70

Cerebrospinal fever and meningitis, cerebrospinal, acute.—Thirty-one cases of cerebrospinal fever were reported during January, February, and March 1936, as follows:

Rate	Age	Place of original admission	Date of admission	Length of service (years)	Disposition
A. S.	20	Naval training station, San Diego, Calif.	Jan. 9, 1936	28 days	Continued.
A. S.	20	do	Jan. 20, 1936	1/12	(1).
A. S.	18	do	Feb. 10, 1936	1/12	Died Feb. 22, 1936.
A. S.	20	do	Feb. 12, 1936	1/12	(1).
Sea. 2	20	do	Feb. 23, 1936	4/12	(1).
A. S.	20	do	Feb. 25, 1936	1/12	(1).
A. S.	20	do	do	1/12	Died Feb. 27, 1936.
A. S.	19	do	Feb. 28, 1936	2/12	(1).
A. S.	19	do	Mar. 1, 1936	1/12	Duty June 9, 1936.
A. S.	21	do	do	3/12	(1).
A. S.	20	do	Mar. 2, 1936	1/12	Duty June 9, 1936.
A. S.	17	do	Mar. 7, 1936	1/12	(1).
A. S.	18	do	Mar. 9, 1936	2/12	(1).
Sea. 2	18	do	Mar. 28, 1936	5/12	(1).
MAtt. 3	19	Naval training station, Norfolk, Va.	Mar. 5, 1936	1/12	(1).
MAtt. 3	21	do	Mar. 6, 1936	2/12	(1).
MAtt. 3	19	do	Mar. 22, 1936	Less than 1 month.	(1).
A. S.	19	do	Mar. 30, 1936	1/12	Died Mar. 31, 1936.
A. S.	17	Naval training station, Newport, R. I.	Feb. 8, 1936	1/12	Died Feb. 12, 1936.
Sea. 1	21	Navy Yard, Washington, D. C.	Mar. 20, 1936	2-3/12	(1).
Pvt.	24	Headquarters, Twelfth naval district.	Mar. 29, 1936	3-6/12	Died Apr. 6, 1936.
F-1	26	Receiving station, Navy Yard, Philadelphia, Pa.	Mar. 6, 1936	7-9/12	(1).
F-3	20	U. S. S. Tennessee	Jan. 13, 1936	2-2/12	Died Jan. 27, 1936.
Sea. 2	21	U. S. S. Detroit	Jan. 23, 1936	1-7/12	Died Feb. 4, 1936.
Sea. 1	21	U. S. S. Portland	Jan. 27, 1936	3-7/12	Duty Mar. 20, 1936.
Sea. 1	25	do	Feb. 22, 1936	5-2/12	(1).
F-2	21	U. S. S. Wyoming	Feb. 6, 1936	1-7/12	Died Feb. 10, 1936.
F-2	24	U. S. S. Jacob Jones	Mar. 6, 1936	3-9/12	(1).
Sea. 2	19	U. S. S. Saratoga	Mar. 19, 1936	1	(1).
Sea. 2	19	U. S. S. Nevada	Mar. 25, 1936	6/12	(1).
SK-1	27	U. S. S. Astoria	do	8-1/12	(1).

[1] Not known at this time.

The medical officer of the Naval Powder Factory, Indian Head, Md., reported that a case of cerebrospinal fever developed in the Civilian Conservation Corps camp located within the limits of that reservation. Onset occurred the morning of March 26, 1936, and a tentative diagnosis was made on March 27. The patient was transferred immediately to Walter Reed Hospital, where confirmation of diagnosis was made the following day. Strict quarantine and other recognized protective measures were instituted and the county and State health authorities notified.

Meningitis, cerebrospinal, acute, was reported as the secondary cause of 2 deaths, coccidiosis being the primary cause in one instance and otitis, media, acute, in the other.

Mumps.—There appears to have been more than the expected incidence of mumps at the United States Naval Training Station, Norfolk, Va., during the quarter—2 in January, 27 in February, and 24 in March. Norfolk reports that "mumps appearing in two recruits definitely exposed in their homes initiated a small epidemic, although the disease is prevalent at this time in Norfolk and vicinity." The U. S. S. *Arizona* reported 56 cases, the first case appearing January 12 in a marine who reported aboard from the United States Marine Corps Base, San Diego, Calif., on December 20, 1935; United States Naval Academy (midshipmen), 30; United States Naval Training Station, Great Lakes, 25; U. S. S. *Mississippi*, 22; Marine Detachment, American Embassy, Peiping, 7; United States Naval Training Station, San Diego, 4; and the Fourth Marines, Shanghai, 3.

Scarlet fever.—Scarlet fever was responsible for 63 admissions during the quarter. A list of the ships and stations and the number of admissions recorded are shown in the following tabulation:

	January	February	March
U. S. S. Pennsylvania [1]	3	24	1
U. S. S. Arizona	4	5	4
U. S. S. Nevada	0	0	9
U. S. S. Maryland	0	0	1
U. S. S. Chicago	0	1	1
U. S. S. Louisville	1	0	0
U. S. S. Saratoga	1	0	0
U. S. S. Relief (duty personnel)	0	1	0
U. S. S. Relief (patient personnel)	0	1	0
Naval Ammunition Depot, Iona Island, N. Y.	0	0	1
Marine Barracks, Puget Sound, Wash.	0	1	0
U. S. S. Medusa	0	0	1
Puget Sound Naval Hospital, Bremerton, Wash. (staff)	0	1	0
U. S. S. Ranger	0	0	1
U. S. S. Zane	0	0	1

[1] 20 additional cases were transferred to hospital "Diagnosis undetermined (scarlet fever)" and diagnosis established after transfer.

Scarlet fever was recorded as the primary cause of 2 deaths. A seaman, second class, 19 years of age, with 1 $\frac{6}{12}$ years' service, attached to the U. S. S. *Chicago*, was admitted to the sick list on January 24, 1936, with catarrhal fever, acute, and transferred to the

U. S. S. *Relief* 4 days later. Scarlet fever developed on January 30, with associated membranous stomatitis, pharyngitis, laryngitis, and rhinitis. Scarlet-fever antitoxin administered. Involvement of nasopharyngeal, oral and laryngeal mucous membrane increased. Pulmonary edema and infarction developed February 5. Patient died on February 7. Autopsy findings: (1) Congestion of all viscera; (2) acute inflammation of bronchial tree; (3) pulmonary infarction, right lower lobe; (4) edema of lungs; (5) sinusitis, sphenoidal, purulent; and (6) thrombosis, lateral sinuses.

A seaman, second class, 22 years of age, with $17\frac{7}{12}$ years' service, attached to the U. S. S. *Pennsylvania*, was admitted to the sick list on January 27 with what appeared to be catarrhal fever. A rash developed and the patient was transferred on January 30 to the Puget Sound Naval Hospital, Bremerton, Wash., where diagnosis was changed to scarlet fever on February 3. A rapidly fulminating pneumonia developed on February 4, and the patient died 2 days later.

Chickenpox.—Sixteen cases of chickenpox were reported for the quarter as follows: In January, one each from the United States Naval Academy (midshipmen), United States Naval Academy (other than midshipmen), Submarine Base, New London, Conn., U. S. S. *Oklahoma*, U. S. S. *Relief* (patient personnel), U. S. S. *Colorado*, U. S. S. *Concord*, and the U. S. S. *Lexington;* in February, three from the marine detachment, American Embassy, Peiping, China, and two from the Fourth Marines, Shanghai, China; and in March, one each from the Naval Air Station, Pensacola, Fla., U. S. S. *Idaho*, and the U. S. S. *Gold Star.*

German measles.—Admissions for German measles at the United States Naval Training Station, San Diego, Calif., increased from 22 in January to 52 in March. "The prevalence of German measles in San Diego area accounts in a large measure for the increase among recruits who had been granted liberty after the detention period." Thirty-five admissions for this disease were recorded in March from personnel serving with the marine detachment, American Embassy, Peiping, China.

Malaria.—The United States Naval Training Station, Norfolk, Va., reported seven cases of malaria for the quarter—two in January, two in February, and three in March.

Typhoid fever.—A moderately severe case of typhoid fever with no complications was readmitted to the Naval Hospital, Mare Island, Calif., from the U. S. S. *Henderson* on January 27 under diagnosis deafness, bilateral (intercurrent to typhoid fever). The probable place of infection is unknown. The patient had not been on liberty since admission. The "health record shows typhoid and paratyphoid prophylaxis on March 17, 24, 31, 1933."

A corporal of the Marine Corps returned to the United States from China on January 24, via the U. S. S. *Henderson*, which sailed from Shanghai on December 14 and stopped at Hong Kong, Manila, Guam, and Honolulu. The patient and a shipmate ate in the "Mother's Lunch" in Honolulu on January 17. (The shipmate became sick soon afterwards and was put to bed in the ship's sick bay.) He left the west coast on February 5 and arrived in Chicago on February 8 with a "cold" and fever which had developed the previous day. He was admitted on February 10 to the Marine Hospital, Chicago, for treatment of a moderately severe case of typhoid fever. Two courses of straight typhoid vaccine had been completed, one on April 10, 1931, and the other on May 16, 1935.

Paratyphoid fever.—A moderately severe case of paratyphoid fever B with no complications was reported by the Fourth Marines, Shanghai, China. The probable place of infection was recorded as Shanghai. Two complete courses of typhoid prophylaxis had been administered—one in September 1923 and one in October 1933.

A hospital apprentice, first class, 25 years of age, with $4\frac{6}{12}$ years' service, was readmitted to the U. S. S. *Relief* from the U. S. S. *Louisville* on January 11, 1936, under diagnosis undertermined (glandular fever). The diagnosis was changed to paratyphoid fever A on January 20. The case was mild with no complications. Two courses of typhoid prophylaxis had been completed, one in November 1931 and one in September 1935.

Dengue.—The V. O. Squadron, 9 M, F. M. F., reported 19 cases and the Naval Station, Olongapo, P. I., 11 cases of dengue for the quarter. In reporting the occurrence of the 11 cases, the station medical officer at Olongapo comments as follows:

"The general health of the service personnel attached to the station has not been as good due to the occurrence of 11 mild cases of dengue fever among the marine personnel. Although nets were used and the barracks are screened their close approximation to the low areas of the town where dengue-carrying mosquitoes breed is considered responsible for this condition. Efforts have been made and are still being made with all available resources by the present commanding officer to eradicate these low areas. Where this is not possible, efforts are being made to have the tides flush such areas daily. In addition to these measures all available oil is placed in low areas where mosquito breeding places are found. Efforts are made to eradicate stagnant water in pans, bamboo stalks, and banana trees about dwellings."

Acute intestinal disorder.—An outbreak of an acute intestinal disorder occurred early in March at the Navy Yard, Philadelphia. As reported by the medical officer of the yard: "Late in February and

extending into early March numerous cases of an acute intestinal disorder were treated, all having practically the same characteristics—those of an intense irritant. The affection was largely among the officers attached to the Marine Basic School, although others living in the yard were affected. No true explanation of the cause has been found; but because the same symptoms appeared in many people within the city of Philadelphia, the water supply is believed to be responsible. It is possible that extra chlorine was injected into the water at city distributing reservoirs because of the excessive rains and melting snow on watersheds."

Jaundice, acute infective.—A mild epidemic of jaundice, acute infective (epidemic type), appeared on board the U. S. S. *Canopus* in February. As reported by the senior medical officer: "During the month of February, 18 cases of the above disease occurred on this ship. Blood serum of all patients was examined and no organisms were found. These cases resembled the epidemic type of jaundice. All were returned to duty and none were seriously ill. The average complement of the U. S. S. *Canopus* for the month of February was 410."

Injuries.—The medical officer of the United States Naval Torpedo Station, Newport, R. I., states in his March sanitary report that "There has been a progressive increase over a period of 4 months of cases of foreign body in the eye. With a view to reduction of these hazards, there will be closer supervision of the enforcement of the use of protective devices such as shields and goggles."

Summary of morbidity in the United States Navy for the quarter ended March 31, 1936

	Forces afloat		Forces ashore		Entire Navy	
Average strength	75,476		42,835		118,311	
	Admissions	Rate per 1,000	Admissions	Rate per 1,000	Admissions	Rate per 1,000
All causes	5,920	325.78	5,226	488.01	11,146	376.84
Disease only	5,185	285.34	4,721	440.85	9,906	334.91
Injuries	732	40.28	498	46.50	1,230	41.50
Poisonings	3	.17	7	.65	10	.34
Communicable diseases transmissible by oral and nasal discharges (class VIII):						
(A)	157	8.64	127	11.86	284	9.60
(B)	1,819	100.10	2,014	188.07	3,833	129.59
Venereal diseases	1,020	56.13	312	29.14	1,332	45.03

Deaths reported, entire Navy, during the quarter ended Mar. 31, 1936

Cause—Disease		Navy			Marine Corps		Nurse Corps	Total
Primary	Secondary or contributory	Officers	Midshipmen	Men	Officers	Men		
Average strength		9,610	1,972	89,105	1,225	16,049	350	118,311
Abscess:								
Brain	None			1				1
Pharynx	Hemorrhage, throat			1				1
Temporal bone	Septicemia				1			1
Appendicitis, acute	Edema, lung			1				1
Do	Obstruction, intestinal, from paralytic causes.			1				1
Do	Peritonitis, general, acute.			1		1		2
Do	Peritonitis, local, acute.			1				1
Carcinoma, intestines.	Peritonitis, general, acute.	1						1
Cerebrospinal fever	None			5		1		6
Do	Pneumonia, broncho-			1				1
Do	Pneumonia, lobar			1				1
Cholecystitis, chronic.	Peritonitis, general, acute.			1				1
Coccidiosis	Meningitis, cerebrospinal, acute.			1				1
Diverticulitis, descending and sigmoid, colon.	Peritonitis, general, acute.					1		1
Hemorrhage, cerebral.	None			2				2
Hyperthyroidism	Myocarditis, acute			1				1
Leukemia	None					1		1
Myeloma, multiple	None			1				1
Myocarditis, acute	Thrombosis, coronary.	1						1
Otitis, media, acute	Meningitis, cerebrospinal, acute.			1				1
Pneumonia:								
Broncho-	Septicemia			1				1
Lobar	None			7				7
Do	Arterial hypertension					1		1
Do	Pleurisy, serofibrinous			1				1
Do	Pleurisy, suppurative					1		1
Do	Septicemia		1					1
Do	Ulcer, duodenum			1				1
Rupture, nontraumatic, heart.	Myocarditis, chronic Thrombosis, coronary.			1				1
Scarlet fever	Pneumonia, lobar			1				1
Do	Thrombosis, lateral sinuses.			1				1
Sinusitis, sphenoidal	Abscess, lung					1		1
Syphilis	Poisoning, acute, neoarsphenamine.							
	Hemorrhagic encephalitis.			1				1
Thrombosis:								
Cavernous sinus	Septicemia			1				1
Coronary	None			2				2
Tuberculosis:								
General miliary	None			1				1
Do	Meningitis, cerebral			1				1
Pulmonary, chronic.	None			1				1
Ulcer, stomach	Peritonitis, general, acute.			1				1
Tumor:								
Benign, mixed, cerebellum, variety unknown.	None			1				1
Malignant, mixed, adenocarcinoma, flank.	None	1						1

Deaths reported, entire Navy, during the quarter ended Mar. 31, 1936—Cont.

Cause—Disease		Navy			Marine Corps		Nurse Corps	Total
Primary	Secondary or contributory	Officers	Midshipmen	Men	Officers	Men		
Tumor—Cont'd.								
Malignant, mixed, adenocarcinoma, testicle.	None	1						1
Malignant, mixed, carcinoma, multiple.	None	1						1
Valvular heart disease, combined lesions, aortic and mitral.	None			1				1
Total for disease		5	1	43	1	7		57
INJURIES AND POISONINGS								
Drowning	None	1		3		1		5
Fracture: Compound, skull.	None			1				1
Simple, skull	Pneumonia, lobar							
	Psychosis with infectious disease.					1		1
Simple, vertebra, cervical.	Intraspinal, injury			1				1
Heat exhaustion	None			1				1
Injuries, multiple, extreme.	None			5		1		6
Do	Pneumonia, broncho					1		1
Intracranial injury	None			1		1		2
Strangulation, neck	None					1		1
Do	Psychosis, unclassified				1			1
Wound, gunshot, head.	None			1				1
Do	Psychoneurosis, psychasthenia.	1						1
Poisoning, illuminating gas.	None			3				3
Poisoning, carbon monoxide, acute.	None			1				1
Poisoning, mercury, acute.	Nephritis, acute	1						1
Poisoning, strychnine sulphate, acute.	None			1				1
Total for injuries and poisonings.		3		18	1	6		28
Grand total		8	1	61	2	13		85
Annual death rate per 1,000:								
All causes		3.33	2.03	2.73	6.53	3.24		2.88
Diseases only		2.08	2.03	1.93	3.27	1.74		1.93
Drowning		.42		.13		.25		.17
Poisonings		.42		.22				.20
Other injuries		.42		.45	3.27	1.24		.57

ADMISSIONS FOR INJURIES AND POISONINGS, FIRST QUARTER, 1936

The following table, indicating the frequency of occurrence of accidental injuries and poisonings in the Navy during the first quarter, 1936, is based upon all form F cards covering admission in those months which have reached the Bureau:

	Admissions, January, February, and March 1936	Admission rate per 100,000, per annum	Admission rate per 100,000, year 1935
INJURIES			
Connected with work or drill	479	1,620	2,592
Occurring within command but not associated with work	343	1,160	1,709
Incurred on leave or liberty or while absent without leave	408	1,379	1,651
All injuries	1,230	4,159	5,952
POISONINGS			
Industrial poisoning	1	3	17
Occurring within command but not connected with work	6	20	43
Associated with leave, liberty, or absence without leave	3	10	19
Poisonings, all forms	10	34	79
Total injuries and poisonings	1,240	4,192	6,039

Percentage relationships

	Occurring within command				Occurring outside command—leave, liberty, or A. W. O. L.	
	Connected with the performance of work, drill, etc.		Not connected with work or prescribed duty			
	January, February, and March, 1936	Year 1935	January, February, and March, 1936	Year 1935	January, February, and March, 1936	Year 1935
Percent of all injuries	38.9	43.6	27.9	28.7	33.2	27.7
Percent of all poisonings	10.0	21.1	60.0	54.4	30.0	24.4
Percent of total admissions, injury and poisoning titles	38.7	43.3	28.1	29.0	33.2	27.7

NOTE.—Poisoning by a narcotic drug or by ethyl alcohol is recorded under the title "Drug addiction" or "Alcoholism", as the case may be. Such cases are not included in the above figures. There were no cases during the first quarter of 1936 worthy of notice from the standpoint of accident prevention.

STATISTICS RELATIVE TO MENTAL AND PHYSICAL QUALIFICATIONS OF RECRUITS

The following statistics were taken from monthly sanitary reports submitted by naval training stations:

January, February, and March 1936	United States naval training station			
	Norfolk, Va.	Newport, R. I.	Great Lakes, Ill.	San Diego, Calif.
Recruits received during the period	1,478	740	880	1,266
Recruits appearing before Board of Medical Survey	9	0	0	0
Recruits recommended for discharge from the service	9	0	0	0
Recruits discharged by reason of Medical Survey	8	0	0	0
Recruits held over pending further observation	0	0	0	0
Recruits transferred to the hospital for treatment, operation, or further observation for conditions existing prior to enlistment	29	42	12	15

The following table was prepared from reports of medical surveys in which disabilities or disease causing the surveys were noted existing prior to enlistment. With certain diseases, survey followed enlistment so rapidly that it would seem that many might have been eliminated in the recruiting office.

Cause of survey	Number of surveys	Cause of survey	Number of surveys
Abscess:		Gonococcus infection:	
Periapical	1	Urethra	5
Acquired, teeth	1	Epididymis	1
Adhesions, abdominal	1	Hypertrophy, bone, mesial surface, distal extremity, right femur	1
Astigmatism	1	Mental deficiency, moron	1
Bursitis, chronic, left knee	1	Metatarsalgia	1
Caries, teeth	3	Myopia	2
Cicatrix, skin	1	Myositis, chronic	1
Color blindness	1	Otitis, media, chronic	9
Constitutional psychopathic state:		Paroxysmal tachycardia	1
Emotional instability	2	Psychoneurosis:	
Inadequate personality	5	Hysteria	2
Curvature, spine, lordosis	1	Psychastenia	1
Cyst, maxillae, left side	1	Rheumatism, muscular	1
Deformity, acquired:		Sprain, sacro-iliac	1
Left ankle	1	Syphilis	6
Left hip and leg (Perthes' disease)	1	Trichophytosis	1
Dislocation:		Ulcer, duodenum	1
Articular cartilage, left knee	1	Union of fracture, faulty	1
Chronic, right knee	1	Valvular heart disease:	
Enuresis	8	Aortic stenosis	1
Epilepsy	3	Mitral insufficiency	2
Flat foot	9	Mitral stenosis	3
Fracture, simple, tibia and fibula	1		
Glycosuria	3		

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[(nc) = notes and comments]

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