VOLUME XXXI JANUARY, 1933 NUMBER 1

United States Naval Medical Bulletin

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

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

VOL. XXXI JANUARY, 1933 No. 1

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
LIEUTENANT COMMANDER JOHN HARPER
MEDICAL CORPS, U. S. NAVY, IN CHARGE

Compiled and published under the authority of Naval Appropriation Act for 1933, Approved June 30, 1932

UNITED STATES
GOVERNMENT PRINTING OFFICE
WASHINGTON : 1932

For sale by the Superintendent of Documents, Washington, D. C. - - - - - See page II for price

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 XII, No. 1, January, 1918.
Volume XIII, No. 1, January, 1919.
Volume XIII, No. 4, October, 1919.
Volume XVII, No. 3, September, 1922.
Volume XIX, No. 3, September, 1923.
Volume XX, No. 5, May, 1924.
Volume XXVII, No. 3–4, July–October, 1929.

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Subscription should be sent to Superintendent of Documents, Government Printing Office, Washington, D. C.

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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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C. E. RIGGS,
Surgeon General United States Navy.

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U. S. NAVAL MEDICAL BULLETIN

VOL. XXXI JANUARY, 1933 No. 1

SPECIAL ARTICLES

EXPIRATORY FORCE AS RELATED TO SUBMARINE ESCAPE TRAINING

By C. W. SHILLING, Lieutenant (Junior Grade), Medical Corps, United States Navy

INTRODUCTION

The object of the present paper is the determination of the greatest possible expiratory force of trainees in the course of training with the submarine escape apparatus. For the purpose of brevity the latter will be indicated by the term "lung" throughout this paper.

The literature of the subject of expiratory force by normal men will first be considered. Such standard textbooks of physiology as those of MacLeod (1), Starling (2), and Howell (3), report the expiratory force as from 75 to 100 mm of mercury, and the inspiratory force as slightly higher. Schneider (4), investigated the physical efficiency of 128 aviators in relation to low oxygen and measured the vital capacity and expiratory force with the following results:

	Average	Probable error
Vital capacity in liters	4.33	0.36
Expiratory force in millimeters of mercury	123.01	1.82

Cripps (5) reported the following data in a study of 950 men of the Royal Air Force:

	Average	Standard deviation	Coefficient of variation
Age.....years	23.80± 0.101	4.62	19.42
Standing height.....inches	68.82± .054	2.472	3.59
Weight.....pounds	139.6 ± .348	15.93	11.41
Vital capacity.....cubic centimeters	4,604.80±13.500	614.70	13.35
Expiratory force.....millimeters of mercury	133.13± .696	31.77	23.88

Schwartz, Britten, and Thompson (6), in a study of physical development reported the expiratory force in certain age groups as follows:

	Millimeters mercury		Millimeters mercury
18 years	135.2	30–34 years	133.9
19 years	136.3	35–39 years	140.2
20–24 years	133.7	40–44 years	148.0
25–29 years	135.7	45–49 years	140.5

The correlation of expiratory force with the capacity to lift, push, and pull was investigated and it was concluded that lung force followed the same trend as other strength tests in relation to age, height, weight, and vital capacity.

Experimental

The total number of men studied was 419. An apparatus was first set up to convince a large group of trainees of their ability to force air out through the flutter valve of the breathing bag of the lung against water pressure. This consisted of a long noncollapsible rubber tube of one-half inch interval diameter with a flutter valve on one end and a mouthpiece taken from a lung at the other extremity; the tube being attached to a vertical rod with a scale in inches inscribed thereon. The men were instructed to inspire through the nose and expire each breath through the tube as it was gradually lowered into the water until a point was reached where the subject was unable to forcibly exhale through the flutter valve. The men were carefully cautioned throughout all of these tests not to use the cheek muscles in forced expiration.

A second group of subjects was tested by means of a mercury manometer connected to a mouthpiece taken from a lung. A large number of trainees was studied by both of the above methods, the comparative results showing no essential differences. All of the data for expiratory force were therefore computed as in millimeters of mercury and the average for 419 trainees is presented in the following table together with other physical measurements indicated.

	Average	Standard deviation
Age, years	25.40±0.1602	4.904±0.117
Standing height, inches	68.73± .0830	2.532± .058
Weight, pounds	155.20± .4730	18.085± .420
Vital capacity, liters	4.52± .0633	0.620± .014
Expiratory force, millimeters of mercury	114.20± .7320	28.000± .655

The standing height and weight were taken without clothing. The vital capacity was determined by means of a standard type of balanced spirometer.

During the course of the collection of the above data the respective averages were computed for two different groups of 100 trainees each and compared. As the differences did not exceed one decimal place it appears conclusive that the final data rest on a sound foundation.

As explanatory of the above table, it will be noted that the average data for the successive factors and the respective standard deviation in each instance are followed by plus and minus decimal figures. The latter represent the probable plus or minus error. Thus, for expiratory force, the average of all data is 114.2 millimeters plus or minus a probable error of 0.7320; and a standard deviation of 28 millimeters, plus or minus a probable error of 0.655.

The average expiratory force of 114.2 millimeters is somewhat lower than that of 123 millimeters found by Schneider, whereas the average of 4.52 liters for vital capacity is slightly higher than his finding of 4.33 liters. A comparison of all average data with the study conducted by Cripps (5) shows that our average man was 1.6 years older, 15.6 pounds greater in weight, of practically the same height, and recorded only a negligible difference in vital capacity. Our group, however, fell considerably below in expiratory force, showing an average of 114.2, as against 133.1 millimeters.

The standard deviation is a measure of the variation from the norm or central tendency and represents the deviation of 68 per cent of the group. In the above data the standard deviation of expiratory force is 28 millimeters of mercury, which means that the expiratory force of 68 per cent of the men tested lies within the limits of 86.2 and 142.2, i. e., the average plus or minus the standard deviation.

Correlation coefficients.—The correlation coefficients of the expiratory force and the other factors were computed with the following results:

Expiratory force and age	0.07
Expiratory force and height	.01
Expiratory force and weight	.15
Expiratory force and vital capacity	.11

The correlation coefficient is a mathematical expression indicating the degree of relationship between the expiratory force and such variables as age, height, weight, and vital capacity. It will be noted, however, that the correlation coefficients, as determined in this study, have no significance, in that there is no fixed relation between expiratory force and the other variables considered. As 1.0 is the highest value and 0.3 is the lowest value showing any significant relation between the groups being compared, it will be seen that these factors fail to show any correlation with expiratory force. This can be further shown by a few extreme examples. The minimum expiratory force for the 419 subjects was 60 millimeters, the maxi-

mum 350 millimeters. A 230-pound man recorded an expiratory force of 74 millimeters, while a 148-pound man exerted a force of 180 millimeters. A man with a vital capacity of 3.3 liters exerted an expiratory force of 180 millimeters, while a man with a 6.0-liter vital capacity showed an expiratory force of only 74 millimeters.

Another aspect of the situation to be considered is the possible lack of cooperation of the experimental subjects. A few men obviously did not exert their most efficient effort, but on the whole this was an unusual situation as can be seen by the fact that the relatively high averages obtained for expiratory force compare favorably with the data in the literature.

The lowest expiratory force found was 60 millimeters of mercury, and the highest 350 millimeters. The latter individual recorded such remarkable expiratory force that the other findings are presented herewith: Age 29, height 67 inches, weight 173 pounds, chest expansion 4¼ inches, vital capacity 4.7 liters, blood pressure 112/69, and pulse 84.

Expiratory force and casualties in lung training.—This study of expiratory force should be considered in connection with certain lung training casualties which have been reported and discussed by Brown (7), Adams (8), and MacClatchie (9), and further summarized by Behnke (10). These studies, coupled with the research of Chillingworth and Hopkins (11) and (12), and the recent investigation of Polak and Adams (13), lead to the conclusion that it is not the increased intrapulmonic pressure alone that induces air embolism, but the pressure, plus the stretching which ruptures the lung tissue, thereby providing egress of air into the circulation. We can thus explain why an intrapulmonic pressure much greater than that necessary to drive air into the circulation of an anesthetized dog may have no effect upon a man, with the muscles of respiration consciously fixed as is the situation in the testing of expiratory force, thus preventing any material stretching of the lung tissue.

In three cases, however, we observed reactions resembling those of the typical Valsalva experiment (14) as reported by Mosler and Balsamoff. These men, while exerting their greatest possible expiratory force, lost consciousness for several seconds, still required physical support upon regaining consciousness and asked such questions as: "What happened?" "What did I do?" These three men registered 160, 152, and 128 millimeters, respectively.

A few men complained of momentary dizziness only, immediately following the expiratory test. Wide individual variation is seen from the fact that there were many whose expiratory force markedly exceeded that of any of the three suffering a reaction and yet they experienced no symptoms.

An important question arises: May breathing resistance either to expiration or inspiration be set up in the lung under unusual circumstances sufficient to cause marked respiratory distress to trainees? During excessively rapid ascent decided increase in expiratory resistance may result incident to the very rapid expansion of air in both the submarine and human lung. The lag in equilibrium between the outside sea pressure and the internal pressure, of course, accounts for this condition. On the other hand, overfilling of the canister of soda lime or clogging of the wire screening may induce an increase in inspiratory resistance. Wetting of the soda lime would raise the latter in still greater degree.

Haldane (15) points out that a resistance of 18 millimeters to inspiration and 10 millimeters to expiration may induce quick, shallow breathing in certain individuals or even fatigue of the respiratory center. He states: "The readiness with which a given resistance to breathing produces signs of fatigue of the breathing varies greatly in different individuals. In some persons a comparatively small resistance suffices to produce shallow breathing and rapid exhaustion of the respiratory center, though in other quite healthy persons a very considerable resistance is needed. Men with symptoms of neurasthenia are, as might be expected, particularly sensitive to resistance. This matter is, of course, important in connection with the design of respirators, etc. A respirator causing any considerable resistance may easily disable a man for muscular exertion." Whether respiration resistances of the order referred to by Haldane may be encountered with the submarine lung is a pressing question and is being investigated at present at the Submarine Base, New London.

Data have already accumulated to the effect that increased breathing resistance of the submarine lung in a normal individual causes nothing more than slow, deep respiration. However, in a subject of nervously unstable type this may prove to be a very upsetting factor, causing considerable difficulty in the preliminary training in the use of the lung, and subsequent disturbance during training escapes. This fact should be impressed upon instructors in training activities, who in turn should instruct trainees as to the cause of this resistance to breathing.

It is not considered that abnormally high resistance has necessarily been a direct cause of any of the lung accidents which have been reported, but it may have contributed to the tendency to hold the breath during ascent which has been shown to be a dangerous practice. When it is considered that 77 men, or 18.3 per cent, out of the 419 trainees in the present study, registered an expiratory force of only 90 millimeters of mercury or less, and that an inspiratory resistance greater than 90 millimeters might be induced by a disturbance

in the proper functioning of the submarine lung, it is imperative that a rigid preliminary inspection of the appliance be enforced at all times.

Summary

(*a*) The age, standing height, body weight, vital capacity, and maximum expiratory force were determined in 419 apparently normal trainees during a course of training with the submarine lung. The averages follow: Age, 25.4 years; standing height, 68.73 inches; body weight, 155.2 pounds; vital capacity, 4.52 liters; and expiratory force, 114.2 millimeters of mercury. The standard deviation of these data, representing 68 per cent of the group, was 4.9, 2.5, 18.1, 0.6, and 28, respectively. The probable plus or minus error of the average results and of the standard deviation was considered as negligible.

(*b*) The minimum expiratory force recorded was 60 millimeters and the maximum 350 millimeters of mercury. A total of 77, or 18.3 per cent, of the 419 trainees registered a reading of 90 millimeters or less. It is concluded that a figure of the general order of 114 millimeters is entirely representative of submarine personnel undergoing lung training.

(*c*) The correlation coefficients of expiratory force with age, height, body weight, and vital capacity were computed, but had no significance in that no fixed relationship between expiratory force and the other factors was found.

(*d*) The possible hazard to individuals with relatively low expiratory force, under circumstances of increased breathing resistance of the submarine lung incident to obstruction in the soda lime canister, or excessively rapid expansion of the contained air in unduly hurried ascent, was discussed.

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VITAL CAPACITY AND ITS RELATION TO CHEST EXPANSION

By C. W. SHILLING, Lieutenant (Junior Grade), Medical Corps, United States Navy

INTRODUCTION

As part of the physical examination for men undergoing training with the submarine "lung" at the Submarine Escape Training Tank, Submarine Base, New London, Conn., vital capacity has been taken by means of a standard type spirometer. This has been added to the routine examination along with the expiratory force test, with the object of determining what effect body structure and physical fitness have on individual reaction to "lung" training. The data are based on a study of 420 men.

During the early course of the investigation it was thought to be of interest to correlate this vital capacity with other body measurements, with special reference to chest expansion, since the latter is commonly used in service physical examinations as a rough indicator of the lung volume or vital capacity (1).

As pointed out by Meakins and Davies (2), interest in vital capacity has loomed large in medical literature. As has been shown by Myers (3) and Pratt (4), it has been a valuable means of estimating the functional disturbance in certain types of cardiac and pulmonary disease; and several writers have advocated its use in conjunction with other physical measurements for the quantitative estimation of physical fitness.

The earliest important contribution made was by Hutchinson (5). He devised a spirometer and collected a large volume of data on vital capacity and expiratory force, and their relation to chest circumference, height, weight, pulse rate, age, and other variables.

Arnold (6) published a monograph nine years later confirming the results of Hutchinson and showed in addition that vital capacity is correlated with chest expansion.

More recent workers have considered and extended almost every angle of the study of vital capacity and its relation to physical fitness and its correlation with various body measurements.

Peabody and Wentworth (7), in an extensive study, divided their male subjects into three groups as follows:

	Vital capacity, cubic centimeters
(1) 6 feet and over	5,100
(2) 5 feet 8½ inches to 6 feet	4,800
(3) 5 feet 3 inches to 5 feet 8½ inches	4,000

West (8) developed a formula which states that the body surface in square meters times a constant (2,500 in the case of men) yields the calculated vital capacity in cubic centimeters for men. He claims that this procedure is more accurate than any other mathematical method. He recommends for highly accurate work that vital capacity be predicted in this way, but considers that for rapid approximate results height may be used as a standard to predict the vital capacity, that is, height in centimeters times a constant of 25.

Dreyer (9) using a group of only 16 men and boys constructed elaborate power furmulæ for the prediction of vital capacity from weight, height, and chest circumference. He (10) again discussed the subject in a monograph published two years later. His work has been criticized because of the limited number of the group, but it is nevertheless important.

Lundsgaard and Van Slyke (11) studied the relation between the size of the thorax and lung volume and found a definite correlation to exist.

Lemon and Moersch (12) with the same grouping employed by Peabody and Wentworth (7) found their results to be slightly higher, namely, 5,450, 4,885, and 4,205 cubic centimeters for the three height groups. They confirmed West's formulæ (8) and considered that his was the most accurate and simple of any method. They also studied the effect of such factors as sex, age, deformities of the thoracic cage, and cardiac and pulmonary disabilities on vital capacity.

Arnett (13) made an exhaustive report on the literature up to 1925 and added his findings in connection with the vital capacity and various diseased conditions.

Although the above is but a brief outline of the more outstanding publications in the voluminous literature of this subject, it

is sufficient to indicate some of the trends in the study of vital capacity.

Experimental

The data for the arithmetic mean and standard deviation of the indicated factors in the present study of 420 men are presented in the following table:

	Arithmetic mean	Standard deviation
Age.....years..	25.4 ±0.1602	4.904±0.117
Standing height.....inches..	68.75 ± .063	2.532± .058
Weight.....pounds..	155.2 ± .473	18.085± .420
Vital capacity.....liters..	4.519± .0633	.62 ± .014
Body surface.....square meters..	1.84 ± .0019	.61 ± .0014
Chest expansion.....inches..	2.78 ± .023	.7 ± .0164
Expiratory force.....millimeters of mercury..	114.2 ± .732	28.000± .655

By the arithmetic mean is meant the average of all data obtained in each instance. From a comparison of the arithmetic mean found by the various workers already referred to, it is found that our group was representative of a good average. They were in every case men who had passed an especially rigid physical examination in order to qualify for the submarine service and were in excellent physical condition when examined and passed for "lung" training.

The standard deviation is a measure of the variation from the norm or central tendency and represents the deviation of 68 per cent of the group. In these data the standard deviation of vital capacity is 0.62, which means that the vital capacity of 68 per cent of the men tested lies within the limits of 3.899 and 5.139 liters; i. e., the arithmetic mean plus or minus the standard deviation.

(*a*) *Correlation coefficients:*

Vital capacity and chest expansion	0.392
Vital capacity and height	.531
Vital capacity and body surface	.465
Vital capacity and weight	.322

The correlation coefficient is a mathematical expression of the relation between any two factors and indicates the degree of relation between the variables in given groups. A complete correlation would be 1.0 and 0.3 is considered the lowest value showing any significant relation between two groups being compared. It is, therefore, evident from the above table that there is some relation between vital capacity and all of the four variables, with the relation between height and vital capacity being the most marked and weight and vital capacity the least marked.

(*b*) *Vital capacity predicted from body weight, height, and body surface.*—Myers (14) carried out a study of the relation between

weight and vital capacity on 1,280 men between the ages of 17 and 32 and arrived at a formula (weight in pounds×21.2+1,168=vital capacity in cubic centimeters) for predicting vital capacity from the body weight. He showed that 11.4 per cent of his men had a vital capacity below 85 per cent of the normal and 13.9 per cent had a vital capacity above 115 per cent of the calculated normal. We applied Myers' weight formula to our group of subjects and found 11.9 per cent below 85 per cent, 0.07 per cent below 70 per cent, and 13.09 per cent above 115 per cent. This is a remarkably close agreement which tends to show that our data are based on a standard group. Myers (14) also prepared certain charts and tables for rapidly determining the vital capacity without any arithmetical calculations.

West (8) constructed a formula for the prediction of vital capacity from height (height in centimeters×25=vital capacity in cubic centimeters for men). His study was applied to 129 individuals, of whom 85 were men. According to his data from men and women combined, 63 per cent were within 10 per cent of the standard, 15 per cent below 90 per cent, and 22 per cent above 110 per cent. We checked our 420 men according to this formula for height and found 58.5 per cent within 10 per cent of the normal, 14.1 per cent below 90 per cent, and 27.3 per cent above 110 per cent. This comparison was also quite close, but it must be remembered that women were not included in the present study. Our group showed a slightly higher percentage with a vital capacity above the predicted normal.

Formulæ based on body surface are reported by certain authors as furnishing the most accurate basis of predicting vital capacity. The calculation of body surface has been greatly facilitated by the chart prepared by DuBois and DuBois (15) (16) based on their formula: S. A.=Wt. 0.425×Ht. 0.725×71.84 where S. A. is the surface area in square meters, Wt. is the weight in grams, and Ht. is the height in centimeters. Hewlett and Jackson (17) used a group of 400 college students and arrived at a body surface formula for predicting vital capacity as follows: Vital capacity (cubic centimeters) =2,900×surface area (square meters) −1,000. They found in checking this group and three other groups that 10.5 per cent fell below 85 per cent and 0.6 per cent below 70 per cent. Using the DuBois and DuBois (15) chart for obtaining the surface area we checked our data from 420 men against the Hewlett and Jackson (17) formula and found 5.9 per cent to fall below 85 per cent, 0.02 per cent below 70 per cent, and 17.8 per cent to rise above 115 per cent.

West (8) has also proposed a body surface formula (body surface in square meters times 2,500 in the case of men) which we used

to check our men against and in this case we found 13.8 per cent below 85 per cent, 0.04 per cent below 70 per cent of the predicted normal, and 6.6 per cent above 115 per cent.

As pointed out above, certain observers have reported that there is a mathematical relation between vital capacity and height, weight, body surface, and chest expansion. It is concluded that our findings compare very favorably with those of previous workers when calculated according to various formulæ proposed for predicting vital capacity from height, weight and body surface.

(*c*) *Vital capacity in relation to chest expansion.*—The next plan was to determine the definite relation, if any, between vital capacity and chest expansion. In 1855 Arnold (6) did the fundamental work on this phase of the study of vital capacity. He reported his data under a heading which read, when translated, " Breathing capacity of 143 male individuals, ages 17 to 38 arranged according to their chest expansion." A part of his results follows:

Chest e pansion:	Vital capacity average cubic centimeters
3 centimeters	2, 944
4 centimeters	3, 275
4.5 centimeters	3, 401
5 centimeters	3, 369
5.25, 5.5, 5.75 centimeters	3, 304
6 centimeters	3, 580
6.5 centimeters	3, 490
7 centimeters	3, 701
7.5 centimeters	3, 790
8 centimeters	3, 729
8.5 centimeters	3, 750
9 centimeters	3, 960
9.5 centimeters	3, 991
10 centimeters	3, 998
11 centimeters	4, 065
11.5 centimeters	4, 200

It is interesting to note that in his study there was only one subject who had a vital capacity above 4,500 cubic centimeters and this reached only 5,200 cubic centimeters. This general tendency to a low vital capacity was in all probability due to the type of spirometer then in use.

Goodall and Belknap (18) conducted a study of the relation of chest expansion to vital capacity with 200 male hospital patients without organic disorders, a group of college students, and a group of enlisted men of the United States Navy and Marine Corps. The number of individuals in the last two groups was not stated. The

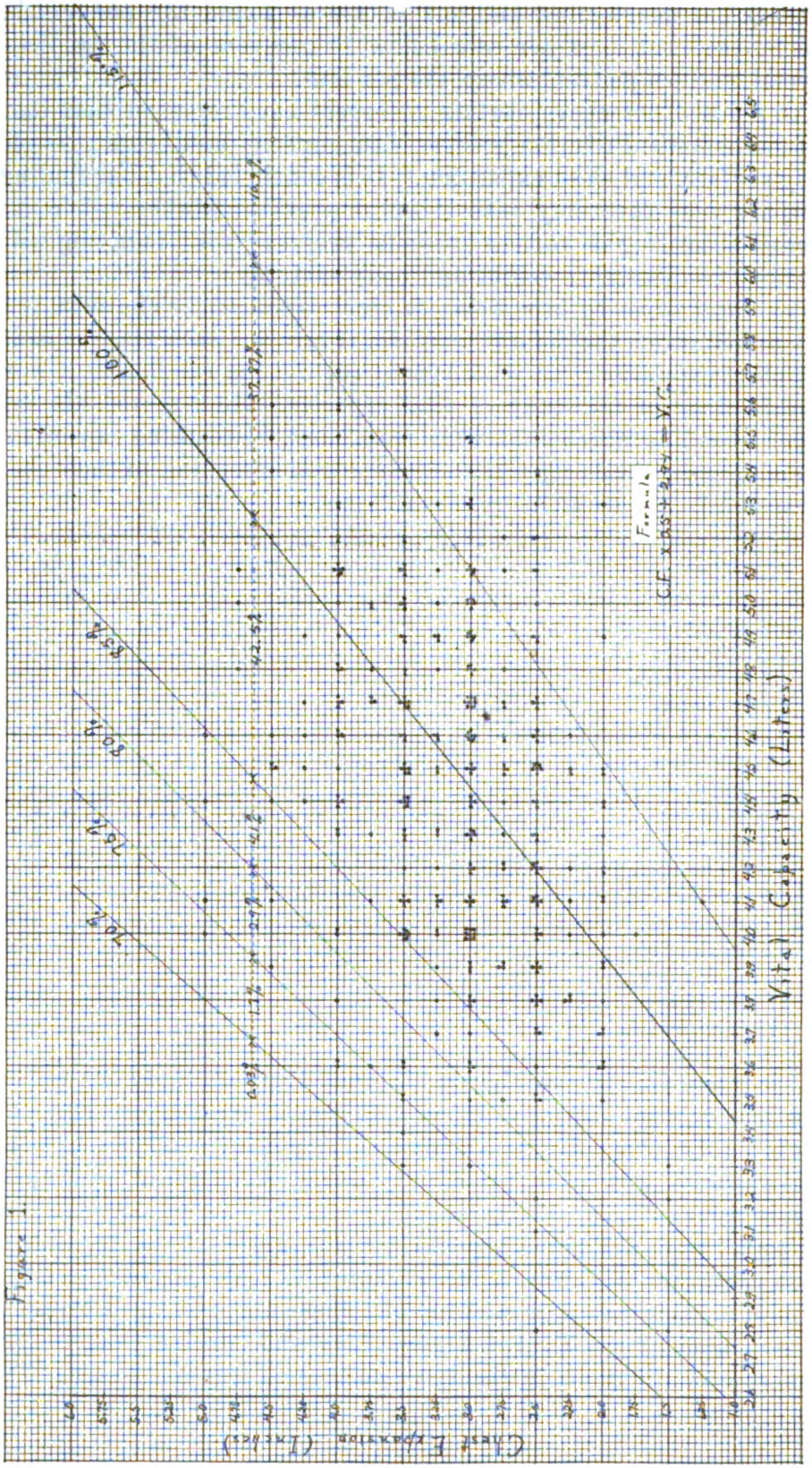

FIGURE 1.—Relation of Chest Expansion to Vital Capacity. (Shilling)

conclusion was reached that the measure of chest expansion has no constant direct relation to the lung capacity and that large measures only indicate good development of the muscles which expand the chest. These authors state that owing to individual variations normal standards can not be adopted. In normal individuals the measure of lung capacity was found to vary directly with muscular development provided the individual breathed properly. They claim that erroneous conclusions may be drawn from spirometer readings unless special attention is given to the method of breathing and that with both measurements large values only indicate good muscular development, while low values indicate inferior muscular development or improper breathing. In general, however, there was a tendency to increased vital capacity as the measure of expansion rose, although there were marked exceptions.

Jackson and Lees (19) took 55 body measurements on a group of 100 men and studied the correlation of the data with vital capacity. They found the chest expansion to have a correlation coefficient of 0.328 ± 0.060. As has already been shown in our group of 420 men, the correlation coefficient of chest expansion with vital capacity was 0.392.

The scatter diagram contained in Figure 1 presents the data for vital capacity by spirometer readings in conjunction with chest expansion for all subjects. The following formula was derived from this diagram to include the greatest possible number of cases within limits of 15 per cent above or below the calculated normal: Chest expansion in inches $\times 0.5 + 2.94 =$ vital capacity in liters. A number of diagonal lines will be observed on the diagram. The diagonal line marked "100 per cent" corresponds to the vital capacity calculated from the chest-expansion data listed in the column at the left by the formula just stated and is designated as the calculated normal. The remaining diagonal lines represent the calculated vital capacities by the same formula but in the indicated percentages of the normal.

It will be seen from a study of the diagram that 8.73 per cent had a vital capacity below 85 per cent of the calculated normal, 4.1 per cent of these falling between 80 to 85 per cent, 2.9 per cent between 75 to 80 per cent, 1.7 per cent between 70 to 75 per cent, 0.03 per cent below 70 per cent, and 10.9 per cent above 115 per cent of the normal. The remaining 80.37 per cent fell within 15 per cent of the calculated normal, with 42.5 per cent falling between 85 to 100 per cent and 37.87 per cent between 100 to 115 per cent.

This formula has been applied to the development of a chart by which the predicted vital capacity in liters can be immediately

Chest Expansion Inches	Vital Capacity in Cubic Inches.																																					Chest Expansion Centimeters
	170	176	183	189	195	201	207	213	219	225	231	237	244	250	256	262	268	274	280	286	292	298	305	311	317	323	329	335	341	347	353	359	366	372	378	384	390	396	
6.0														69	70	72	74	75	77	79	80	82	84	86	87	89	90	92	94	95	97	99	101	103	105	106	108	110	15.24
5.75														70	72	73	75	77	79	80	82	84	86	87	89	91	93	95	96	98	100	101	103	105	107	109	110	112	14.60
5.5												70	72	73	75	77	79	80	82	84	86	87	89	91	93	95	96	98	100	101	103	105	107	109	111	113	114	13.97
5.25												70	72	73	75	77	79	80	82	84	86	88	90	92	94	96	98	99	101	102	104	106	108	110	112	113	115	117	13.33
5.0									69	71	73	75	77	79	80	83	85	86	88	90	91	93	95	97	99	101	102	104	106	108	110	112	113	115	117	119	12.70
4.75									69	71	73	75	77	80	82	83	85	86	88	90	92	94	96	98	100	102	104	106	108	109	111	113	115	117	119	121	122	12.06
4.5									69	71	73	75	77	78	80	83	85	87	89	91	92	94	96	98	100	102	104	106	108	110	112	114	116	118	120	122	124	126	11.43
4.25									69	71	73	75	77	78	80	83	85	87	89	91	93	95	97	99	102	105	107	109	110	113	115	116	118	120	122	124	126	128	10.79
4.0							68	70	73	75	76	78	80	83	85	87	89	91	93	95	97	99	101	103	105	107	109	111	113	115	117	120	122	124	126	128	130	10.16
3.75						68	70	72	75	76	78	80	83	85	87	89	91	93	96	98	100	102	104	106	108	110	112	114	116	118	120	122	124	126	128	130		9.52
3.5					68	70	73	75	76	78	81	83	86	88	90	92	94	96	98	100	102	104	106	108	111	113	115	117	119	121	123	125	127	130					8.89
3.25				68	70	72	75	76	78	81	83	86	88	90	92	94	96	98	100	102	105	107	110	112	114	116	118	120	122	124	126	128	130						8.25
3.0			68	70	73	75	76	78	81	83	86	88	90	92	95	97	99	101	104	107	109	111	113	115	117	119	121	123	126	128	130								7.62
2.75			68	70	72	74	76	78	81	83	86	88	90	92	95	97	99	101	104	107	109	111	113	115	118	120	122	124	126	128	130								6.98
2.5		69	71	73	76	78	81	83	86	88	90	93	95	97	100	102	105	107	110	113	115	117	119	121	123	126	128	130											6.35
2.25	69	71	73	76	78	81	83	86	88	90	93	96	99	101	103	106	108	110	113	115	118	120	123	125	128	130													5.71
2.0	72	74	76	78	81	84	86	89	91	93	96	99	101	103	106	109	111	114	116	119	122	124	127	129															5.08
1.75	74	76	78	81	84	87	90	92	95	97	100	102	105	108	110	113	115	118	121	123	125	128	130																4.44
1.5	76	78	81	84	87	90	93	95	97	100	103	106	109	111	114	116	119	121	124	127	130																		3.81
1.25	78	81	84	87	90	93	96	99	101	103	107	110	113	115	118	121	124	127	129	131																			3.17
1.0	81	84	87	90	93	96	99	102	105	108	111	113	116	119	122	125	128	131																					2.54
	2.8	2.9	3.0	3.1	3.2	3.3	3.4	3.5	3.6	3.7	3.8	3.9	4.0	4.1	4.2	4.3	4.4	4.5	4.6	4.7	4.8	4.9	5.0	5.1	5.2	5.3	5.4	5.5	5.6	5.7	5.8	5.9	6.0	6.1	6.2	6.3	6.4	6.5	
Vital Capacity in Liters																																							

FIGURE 2.—The Actual Vital Capacity Expressed in percentages of the Calculated Vital Capacity. (Shilling.)

obtained without calculation from the chest expansion in inches, as follows:

Prediction of vital capacity from chest expansion

Chest expansion:	Vital capacity (liters)
6 inches	5. 940
5¾ inches	5. 815
5½ inches	5. 690
5¼ inches	5. 565
5 inches	5. 440
4¾ inches	5. 315
4½ inches	5. 190
4¼ inches	5. 065
4 inches	4. 940
3¾ inches	4. 815
3½ inches	4. 690
3¼ inches	4. 565
3 inches	4. 440
2¾ inches	4. 315
2½ inches	4. 190
2¼ inches	4. 065
2 inches	3. 940
1¾ inches	3. 815
1½ inches	3. 690
1¼ inches	3. 565
1 inch	3. 440

It was then planned to develop a graphic method by which the percentage of the calculated vital capacity based on chest expansion could be determined in terms of the true vital capacity measured by a spirometer. It is believed that this procedure will be of distinct value in connection with statistical studies of vital capacity. The chart presenting this method is contained in Figure 2.

The procedure of application of the method is as follows: Take the chest expansion in inches or centimeters. Locate in the extreme left-hand or extreme right-hand column of the chart the figure which approximates most closely to the chest expansion. Take the individual's vital capacity in liters or cubic inches with a spirometer. Locate in the horizontal column at the top or bottom of the table the figure which approximates most closely the vital capacity recorded. Now follow the vertical column from the figure representing the vital capacity to the point where it intersects the horizontal column leading from the figure representing the chest expansion. In the square where the two columns intersect will be found the percentage of the calculated vital capacity based on the chest expansion in terms of the actual vital capacity.

(*d*) *The value of a practical formula to predict vital capacity based on chest expansion.*—The subjects used in this study were

selected men physically, and at the time of the study were in normal condition as indicated by a general physical examination. The averages and standard deviations of the various variables are in good agreement with analogous results reported by previous workers. The mathematical coefficients of correlation for vital capacity and the other variables compare favorably with data of other studies. This also applies to the data based on the formulas developed by the various authors cited in the paper.

The formula (chest expansion in inches $\times 0.5 + 2.94 =$ vital capacity in liters), as has been shown, gives a total of 80.37 per cent of all subjects within 15 per cent of the calculated normal. Myers (14) in his weight formula quoted earlier found only 74.7 per cent within 15 per cent of the calculated normal, and in checking our group against this same formula we found 74.94 per cent within 15 per cent of the calculated normal.

In calculating our data against West's (8) height formula we found 77.2 per cent within 15 per cent of the normal. Using the body-surface formula of Hewlett and Jackson (17) as a standard of comparison we found 76.28 per cent of our group within 15 per cent of the standard; and in checking our group against the formula of West (8) for body surface we found 79.56 per cent within 15 per cent of the normal.

In view of these comparative considerations it is considered fair to assume that the results relative to chest expansion and vital capacity have an approximately universal application to normal men.

No previous research has been published in reference to a formula for predicting vital capacity from chest expansion, and therefore no comparison is afforded for the present group. It is believed, however, that the chest-expansion formula promises more favorable results than any of the other formulæ proposed. No method of predicting vital capacity is accurate enough to take the place of the spirometer. It is believed, however, that in its absence an approximate vital capacity can be predicted from the chest expansion as routinely measured in the naval service.

There are two general types of men who tend particularly to vary from this predicted normal, namely, the asthenic type and the heavy muscular type. The tall thin man will almost invariably register a vital capacity much greater than his chest expansion prediction and the muscular type will frequently show a vital capacity smaller than the prediction. We believe that this is due to the fact that the tall thin man tends to breath almost entirely by the use of his diaphragm, whereas the muscular or athletic type of individual is accustomed to thoracic breathing and, for this reason, in conjunction with his heavy musculature, can expand his chest out of proportion to his

vital capacity. This fact was pointed out by Goodall and Belknap (18) who gave a few extreme examples to demonstrate why chest expansion was of little value as one of the methods of physical examination. It is considered, however, that a great part of the difficulty can be obviated by directing the subject to take the greatest possible inspiration rather than by urging him to expand his chest to the greatest extent. In this way there will be less tendency to expand the chest out of proportion to the vital capacity.

It was found in this study that body structure and vital capacity had practically no effect on the individual reaction to "lung" training but, as reported elsewhere (20), expiratory force may have a significant bearing on individual reaction and to "lung" training under certain circumstances.

Summary

1. A study of 420 men was conducted in which vital capacity was correlated with height, body weight, body surface, and chest expansion. The following average data were obtained: Height 68.75 inches, body weight 155.2 pounds, body surface 1.84 square meters, chest expansion 2.78 inches, and vital capacity 4.519 liters. The standard deviation for these respective measurements was as follows: 2.53 inches, 18.08 pounds, 0.61 square meter, 0.70 inch, and 0.62 liter.

2. The following correlation coefficients of vital capacity with the indicated factors were found: Chest expansion 0.392, height 0.531, body surface 0.465, and body weight 0.322. It was concluded that a significant relation obtained between vital capacity and all four of these factors.

3. The following formula was developed for the prediction of vital capacity from chest expansion: C. E. $\times 0.5 + 2.94 =$ V. C.; C. E. indicating the chest expansion in inches and V. C. the vital capacity in liters; 80.37 per cent of the results for vital capacity measured by a spirometer fell within 15 per cent above or below the calculated findings by this formula.

4. A chart was constructed which presented the predicted vital capacity correlated with the chest expansion without the need of calculation.

5. A chart was also constructed by means of which the percentage of the individual's vital capacity measured by a spirometer, above or below the predicted value based on the chest expansion, may be quickly determined.

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TRAUMATIC LUNG LESIONS PRODUCED IN DOGS BY SIMULATING SUBMARINE ESCAPE[1]

By B. H. ADAMS, Lieutenant Commander, Medical Corps, United States Navy, and I. B. POLAK, Lieutenant Commander, Medical Corps, United States Navy

In a previous communication (1) a laboratory method was described for producing air embolism in dogs. In this paper the

[1] From the Department of Physiology, Harvard School of Public Health.

authors present experiments in which they have endeavored to approximate more closely normal conditions. The problem was to determine if a dog ascending from a given depth from a diving bell would hold his breath until lung injury occurred.

Method.—Two dogs were used for these experiments. The submarine escape training tank at the submarine base, New London, Conn., shown in the accompanying figure, which is used for training in the use of the submarine escape lung, gave sufficient depth to study the ascent of a dog.

Results of Experiments

Experiment 1.—On February 19, 1932, a dog weighing 9 kilograms was taken into the side lock of the submarine escape training tank at the 50-foot level. When the pressure was equalized with the 50-foot water level in the escape tank, the dog was put through the door of the lock and allowed to ascend unhampered to the surface. The ascent was made in 12 seconds, at a rate of about 4 feet per second. He was observed to vent air from his nose all the way to the surface. He appeared normal following his ascent, neither coughing or showing any signs of distress, and has subsequently shown no harmful effects from the experiment.

Experiment 2.—A second dog weighing 12 kilograms was taken down in the diving bell and put out at the 85-foot level indicated in the figure. He made the ascent in 40 seconds, or about 2 feet per second. It was difficult to observe the first part of his ascent because of poor visibility from the diving bell in which the observers followed him to the surface. During the last 20 feet of the ascent he was seen to vent. He was also making slow swimming movements and was evidently in distress. On emerging at the surface, frothy fluid appeared at his nose and he swam slowly to the edge of the tank.

When pulled out he stood with legs braced, and coughed and vomited about a pint of liquid. He continued coughing and showed marked dyspnoea and weakness. Auscultation of his chest revealed musical rales. The pulse rate was 100.

Twenty-four minutes after reaching the surface, the animal was chloroformed and an autopsy performed. It is entirely possible that the animal would have recovered from this injury. No bubbles were found in the coronary vessels or in the heart cavities. All the surfaces of the lungs revealed numerous hemorrhagic areas of about 1 centimeter in diameter. When the lung tissue was incised, considerable frothy fluid was found. No bubbles were found in the mesenteric vessels or in the large vessels at the base of the heart.

The brain was not examined owing to lack of facilities for an extensive autopsy. Microscopic study of the lung tissue revealed typical findings as demonstrated in our previous communication (1).

These changes included emphysema, rupture of the alveoli, and interstitial emphysema about the pulmonary capillaries. No hemorrhagic exudate was found in the interstitial tissue.

Summary

Experiment 2 demonstrates that a dog making an ascent from a depth of 85 feet, presumably with the lungs partly distended with air, may endeavor to hold his breath as long as possible. This is what a man tends to do under the same circumstances. Men using the submarine "lung" and finding it impossible to breathe into it will make a rapid ascent to the surface and have a tendency to hold the breath. Accidents resulting from this procedure have already been reported in the United States Naval Medical Bulletin (2).

These experiments therefore were made to clarify several points—especially to determine if a dog, while making an ascent, would hold his breath until injury resulted. The authors believe that the breath is held until the breaking point or until overstretching and tearing of the alveolar walls results, and that then and only then does either man or dog vent air, unless it is done voluntarily. We believe that some damage results before the animal is able to release the air from his lungs, as expansion of the air is very rapid as he nears the surface. The slower the ascent the less likely is he to hold his breath much beyond the breaking point. The unrestrained ascent of a man wearing the submarine "lung" is very rapid, and the breaking point of the alveolar walls may be passed so quickly that considerable damage may occur before he can adequately release the expanding air from the lungs. We believe that if the rapidity of the ascent were lessened, the subject, holding the breath, would release air from the lungs immediately upon the appearance of pain and thereby reduce the danger.

The failure of visible air emboli to appear at autopsy in our experiment was probably due to the immediate release of the air from the lungs at the time rupture resulted. If the dog had continued to hold his breath demonstrable air embolism would undoubtedly have resulted. The possibility of small air emboli in the brain was, of course, not excluded.

The writers wish to express their appreciation to Prof. C. K. Drinker for suggesting and outlining this investigation and to the commanding officer and diving tank personnel at the United States submarine base, New London, Conn., for their cooperation and assistance.

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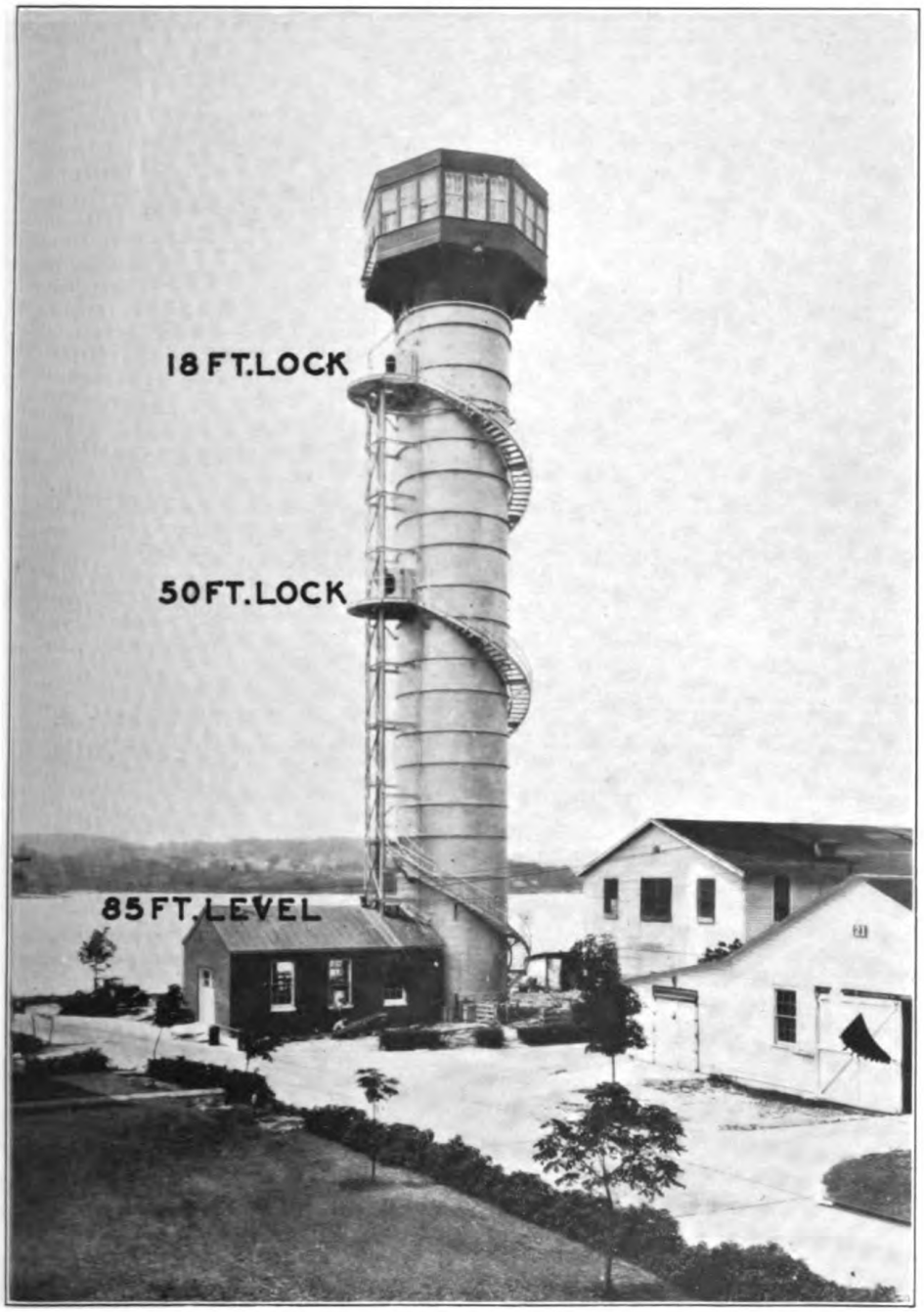

THE SUBMARINE ESCAPE TRAINING TANK AT THE SUBMARINE BASE, NEW LONDON, CONN.

Showing the 18-foot lock, 50-foot lock, and the 85-foot level. (Adams and Polak.)

THE DUTIES OF A MEDICAL OFFICER AFLOAT, UNITED STATES NAVY

By J. W. VANN, Lieutenant Commander, Medical Corps, United States Navy

During the years immediately preceding September, 1916, applicants for appointment in the Medical Corps of the Navy were given a preliminary physical and professional examination and those who qualified were sent to the Naval Medical School for a course of instruction. Those found physically and professionally qualified on examination after the course were then commissioned in the regular Navy. Early in 1917 and in 1918 the opportunity for enrollment in the Naval Reserve Force was extended to graduates of class "A" medical schools without professional examination, and many of these later entered the Medical Corps of the regular Navy after passing the required examinations.

Prior to September, 1916, the instruction given to each class at the Naval Medical School had extended over a period of from 6 to 7 months, but at that time it was found necessary to shorten the period of instruction to 4 months. It wasn't long, however, before it became necessary to further abbreviate the course, so that in 1917 it became approximately six weeks. These courses contained, along with other subjects of particular interest to naval medical officers, instructions in naval drills, naval routine, and duties of medical officers afloat and ashore, so that those who attended the school were fitted for the duties they were called upon to perform when they reported to their next assignment.

The Annual Report of the Surgeon General of the Navy for the fiscal year 1918 states that during the 10-year period from 1906–1916 there were 235 students given instruction at the school, and during the following year 175 students were in attendance. This was, however, but a small percentage of the number of medical officers who entered upon active duty for the first time during 1917, so obviously many reported to their assignments aboard ship or at hospitals and shore stations with but little knowledge of the problems they were to encounter. Fortunately the larger ships and the transports had for a senior medical officer an officer of the regular Navy with long experience, who knew the regulations and duties concerning his department, and from these men the newly commissioned medical officers learned, by precept and example, what they would have learned in normal times during the course of instruction at the Naval Medical School.

Should the Medical Corps of the Navy be called upon to expand again as in 1917, the same conditions may obtain, and many young medical officers may be embarrassed in the beginning on account of lack of knowledge of their duties. The information is all contained in the Naval Regulations, the Manual of the Medical Department,

General Orders, Uniform Regulations, and Circular Letters, but where to find it is often a problem.

An article entitled "The Duties of Medical Officers Afloat," by Surg. Capt. J. S. Dudding, Royal Navy, appeared in the Journal of the Royal Naval Medical Service of April, 1932. The subject matter and the outline of such duties impressed me so favorably that a similar outline of duties afloat of a medical officer, United States Navy, is furnished herewith as a guide and ready reference. An attempt will be made to keep the references up to date as changes in Navy Regulations and the Manual of the Medical Department occur, and it is hoped that it will be available for distribution in case of another rapid expansion of the corps. The abbreviations N. R. refer to the United States Navy Regulations, 1920, and M. M. D. to the Manual of the Medical Department, 1927.

For convenience the outline of The Duties of a Medical Officer Afloat, United States Navy, is arranged under four principal headings:

A.—Naval routine—customs and ceremonies. (Uniforms, drills, salutes, and other general information.)

B.—General Medical Department duties.

1. Organization of the Medical Department.
2. Care of the sick.
3. Care of the dead.
4. Sanitation and preventive measures.
5. Educational measures.
6. Preparation of reports.
7. Miscellaneous duties.

C.—Medical Department stores.

1. Sources.
2. Care and custody.
3. Disposition.

D.—Health records.

1. Opening.
2. Custody.
3. Disposition.

A.—NAVAL ROUTINE—CUSTOMS AND CEREMONIES

Titles—N. R. 148.

Uniforms—{Uniform Regulations. / N. R. 122.

Quarters for officers—N. R. 1428 and 1429.

Officers' messes—N. R. 1435 to 1440.

Salutes—{(*a*) The national anthem—N. R. 230 and 338. / (*b*) To colors and quarter deck—N. R. 285. / (*c*) Personal—N. R. 286 and 287.

Gangways—N. R. 284.
Boat etiquette—N. R. 291.

Drills—
- (a) General drills—N. R. 1302 and 1303; M. M. D. 861 and 862.
  - (1) Collision drill—N. R. 1304; M. M. D. 868.
  - (2) Fire drill—M. M. D. 867.
  - (3) Abandon ship—M. M. D. 869.
  - (4) Fire and rescue—M. M. D. 871.
  - (5) Landing force—M. M. D. 870.
  - (6) Gas defense—M. M. D. 872.
- (b) General quarters—N. R. 1301; M. M. D. 863, 864, and 865.

Courts and boards—
- Courts martial—see Courts and Boards.
- Board of medical examiners—see Courts and Boards.
- Board of inquest—see Courts and Boards.
- Board of investigation—see Courts and Boards.
- Court of Inquiry—see Courts and Boards.

Junior medical officers—
- Duties—M. M. D. 761.
- Absence from duty—M. M. D. 762.

Leave—
- Definition—N. R. 1725.
- General instructions—N. R. 1722 and 1731.
- Application for permission to leave the ship—N. R. 1730.

B.—GENERAL MEDICAL DEPARTMENT DUTIES (N. R. 1132 TO 1177; M. M. D. 701–707)

1. Organization of the Medical Department.
   - (a) Medical officer is head of Medical Department of ship—N. R. 1132; M. M. D. 821.
   - (b) Medical officer is in charge of surgeon's division—N. R. 1175, N. R. Chapter 30; M. M. D. 821.
   - (c) Watch, quarter, and station bills—N. R. 1106; M. M. D. 835.
   - (d) Sick bay—Detail of Hospital Corps men; Daily routine; Weekly routine. To be in writing.
   - (e) Battle stations—M. M. D. 873–887.
2. Care of the sick—M. M. D. 711.
   - (a) Sick call—(ship's routine).
   - (b) Junior medical officer's responsibility—M. M. D. 761.
   - (c) Senior medical officer's responsibility—M. M. D. 821.

(*d*) Binnacle list—M. M. D. 826.
(*e*) Sick list—M. M. D. 826.
(*f*) Transfer to naval hospital—{N. R. 1141 and 1142. / M. M. D. 829 and 2213(a).
(*g*) Transfer to other than naval hospital—{N. R. 1143. / M. M. D. 2213(b).
(*h*) Medical surveys—{N. R. 1141, 1197, 1198, and 1199. / M. M. D. 3426.

3. Care of the dead—M. M. D., chapter 19.
(*a*) Certificates of death—N. R. 909, 1144, 1843.
(*b*) Report of death—{N. R. 908. / Form N.
(*c*) Information required by the department—M. M. D. 2904.
(*d*) Investigation of deaths—M. M. D. 2921–2923.
(*e*) Embalming—M. M. D. 2931–2939.
(*f*) Transportation of remains—M. M. D. 2941–2976.
(*g*) Disposition of effects—M. M. D. 2977–2981.
(*h*) Battle casualties, disposition of the dead—M. M. D. 885.

4. Sanitation and preventive measures—M. M. D., chapter 17.
(*a*) Vaccination against smallpox—{N. R. 1152. / M. M. D. 2604.
(*b*) Vaccination against typhoid fever—{N. R. 1152. / M. M. D. 2605.
(*c*) Vaccination against other diseases—M. M. D. 2603.
(*d*) Inspection of food—{N. R. 1320. / M. M. D. 2608–2609.
(*e*) Water—{N. R. 1320. / M. M. D. 2610.
(*f*) Ventilation and lighting—M. M. D. 2611 and 2614.
(*g*) Communicable diseases—{N. R. 1133. / M. M. D. 2616.
(*h*) Lead poisoning—M. M. D. 2617.
(*i*) Venereal diseases—
(1) Prophylaxis—General Order No. 69.
(2) Concealment—{General Order No. 69. / N. R. 1136.
(3) Restriction—{General Order No. 69. / N. R. 1319(2).
(4) Misconduct status and loss of pay on sick list—{N. R. 1196. / M. M. D. Chapter 16.
(5) Transfer of men with—{N. R. 1142. / M. M. D. 703.
(*j*) Cleanliness, clothing, bedding—N. R. 1319.
(*k*) Fumigation—M. M. D. 2871–2886.
(*l*) Barber shops, sanitary rules for.

5. Educational measures.
   (*a*) Venereal diseases—M. M. D. 704.
   (*b*) General health measures—M. M. D. 704.
   (*c*) Instruction in first aid—N. R. 1137, 1155, and 1346; M. M. D. 836 and 882.
   (*d*) Instruction of Hospital Corps men—M. M. D. 704.
6. Preparation of reports—M. M. D., chapter 23.
   (*a*) General instructions—M. M. D. 3401 to 3406.
   (*b*) Medicine and Surgery lettered forms—M. M. D. 3411–3435.
   (*c*) Medicine and Surgery numbered forms—M. M. D. 3441–3442.
   (*d*) Miscellaneous forms and reports—N. R. 1153, 1154, 1322; M. M. D. 2694, 3551–3554.
   (*e*) Annual sanitary report—M. M. D. 2691 and 2698.
7. Miscellaneous duties.
   (*a*) Bills of health—N. R. 860 and 1172; M. M. D. 2831–2841.
   (*b*) Health of the port—N. R. 1134 and 1147.
   (*c*) Inspections of personnel—N. R. 1136; M. M. D. 825.
   (*d*) Inspections of living spaces—N. R. 216, 1160, 1161, and 1360(2); M. M. D. 825.
   (*e*) Swimming—N. R. 1323; M. M. D. 2618.
   (*f*) Diving—M. M. D. 1301–1305, and 1535.
   (*g*) Submarine duty—M. M. D. 1281–1290 and 1534.
   (*h*) Aviation duty—M. M. D. 1261 to 1266 and 1538 to 1566.
   (*i*) Drunkenness—M. M. D. 743.

C. MEDICAL DEPARTMENT STORES

1. Sources—M. M. D. 3020 to 3039.
   (*a*) Medical supply depots—M. M. D. 3442.
   (*b*) Naval Medical School—M. M. D. 3312 and 3327.
   (*c*) General storekeeper—M. M. D. 3024.
   (*d*) Open purchase—N. R. 1165, 1167, 1399, and 1608; M. M. D. 3040 to 3045.
   (*e*) Allotments—M. M. D. 3050–3054.
   (*f*) Intoxicating liquors and alcohol—M. M. D. 3032.
2. Care and custody.
   (*a*) General accountability—N. R. 1164 to 1166 and 1194; M. M. D. 3070 to 3081.
   (*b*) Medical storerooms—N. R. 1048 (2) (3), 1145, and 1434; M. M. D. 824 and 3076.
   (*c*) Inflammables and acids—N. R. 1347.

(*d*) Alcohol and narcotics—{N. R. 13, 118, and 1146. M. M. D. 739 and 3032.

(*e*) Dental—{N. R. 1182–1183. M. M. D. 234, 235, and 237.

(*f*) Inventory—M. M. D. 3416.

3. Disposition.

(*a*) By survey—{N. R. 1389. M. M. D. 3046 to 3049, and 3415.

(*b*) By transfer—{N. R. 1171. M. M. D. 3416 (d), (e), (f).

(*c*) On decommissioning of ship—{N. R. 1170. M. M. D. 3023 (b).

(*d*) Report of expenditures—N. M. S., Form E.

D. HEALTH RECORDS (N. R. 138)

1. Opening.

(*a*) Officers—M. M. D. 2202 (a) and 2203 (a).

(*b*) Nurses—M. M. D. 2205.

(*c*) Midshipmen—M. M. D. 2204.

(*d*) Men—{N. R. 1200 and 1201. M. M. D. 2206.

(1) Enlistment—{N. R. 1200 and 1201. M. M. D. 2206 (a).

(2) Reenlistment—M. M. D. 2206 (b).

(3) Extension of enlistment—M. M. D. 2206 (b).

2. Custody—{N. R. 1163 and 1195. M. M. D. 2208.

(*a*) Receipt of—M. M. D. 2209 and 2214.

(*b*) Record of—M. M. D. 2214.

(*c*) Entries—{N. R. 1195, 1196, 1200, and 1201. M. M. D. 2282 to 2292.

(1) Vaccination against smallpox—M. M. D. 2604.

(2) Typhoid—M. M. D. 2605.

(3) Syphilis—M. M. D. 2331.

(*d*) Verification of—{N. R. 826 and 1151. M. M. D. 2209.

3. Disposition—M. M. D. 2211 and 2214.

(a) To hospital—M. M. D. 2213.

(*b*) Expiration of enlistment—M. M. D. 2206 (d).

(*c*) Transfer—{N. R. 1201 (2). M. M. D. 2211 (a).

(*d*) Desertion—M. M. D. 2206 (d).

(*e*) Death—M. M. D. 2202 (d).

(*f*) Promotion—M. M. D. 2202 (b).

(*g*) Termination of service—M. M. D. 2202 (c).

CLINICAL NOTES

EPHEDRINE IN THE TREATMENT OF ADAMS-STOKES SYNDROME

REPORT OF CASE

By L. J. ROBERTS, Lieutenant Commander, Medical Corps, United States Navy, and T. H. TABER, Lieutenant Commander, Medical Corps, United States Navy

The occurrence of Adams-Stokes syndrome in a patient suffering from complete heart block must always be considered serious. The duration of life after this complication has supervened is usually brief. Death during a seizure is not uncommon. The importance of rendering these attacks less frequent or preventing their occurrence is therefore manifest. Only in rare instances, as in gumma of the bundle of His, may treatment of the lesion itself be expected to be successful. It is apparent, therefore, that any drug which is likely to abolish the Adams-Stokes seizures themselves is worthy of consideration, even though the underlying pathological condition remains unchanged.

The Adams-Stokes attacks are due to ventricular standstill for more than a few seconds. Standstill of the ventricles may occur during the period of transition from partial to complete heart block before the new idio-ventricular rhythm becomes established. It is due to depression of the idio-ventricular pacemaker which does not take up the control of the heart beat readily. Standstill may also occur after complete heart block has become established when the pacemaker becomes depressed.

The object of therapy in these cases should be to relieve this depressed state of the ventricles. In recent years several drugs have been used with this end in view. One of these drugs is barium chloride, which is known to act by increasing the irritability of muscle. In 1923 Cohn and Levine (1) reported its successful use in three patients with Adams-Stokes syndrome. In each instance the patients, who were suffering from frequent attacks, were rendered free from them by the administration of barium chloride by mouth. Since then barium chloride has come into general use in the treatment of this condition. It is not, however, always successful.

For a number of years it has been known that epinephrine will often prevent the recurrence of attacks when given subcutaneously

or stop them when injected into the heart itself. It is assumed that in these cases the drug exerts its action through the sympathetic nerve endings in the heart muscle. Epinephrine has the disadvantage that its action is of short duration, requiring subcutaneous injection several times a day.

It was, therefore, natural that ephedrine, with its epinephrinelike action, should be tried in Adams-Stokes disease, inasmuch as its action is more prolonged and it has the additional advantage over epinephrine of not requiring subcutaneous injection. Hollingsworth (2) in 1927, Stecher (3) in 1928, Parade and Voit (4) in 1929, and Wood (5) in 1932, reported the successful use of ephedrine in this disease. In Stecher's case barium chloride was not entirely successful. In Wood's case the attacks actually increased in number and severity in spite of its use. Both, however, obtained complete relief by the use of ephedrine.

In the following case the use of ephedrine appeared to be attended with marked success when, following the lengthening of the time interval between doses, sudden death supervened.

Case Report

H. J. R., chief yeoman, United States Navy, was admitted to the United States naval hospital, Mare Island, Calif., on May 30, 1931. He had considered himself in good health until 10 weeks prior to admission. At that time he first noticed dyspnea even on slight exertion and occasional attacks of vertigo. A week later he had a syncopal attack. He was put to bed, but, as he was feeling well the next day, he was returned to duty. He continued on duty for about two weeks and noticed some dyspnea and momentary attacks of vertigo which were relieved by lying down. Six weeks before entering the hospital he had his first severe attack, during which he became unconscious for about 15 seconds. Later that evening he developed severe headache and had repeated attacks of dyspnea and syncope. He was then transferred to U. S. S. *Relief*, where he was retained as a patient until transferred to Mare Island. In the meantime his attacks had become more frequent and more severe.

The family history was unimportant, except that his father died at the age of 57 of congestive heart failure. There was nothing of importance in the past history. He denied chancre and had never received antiluetic treatment.

Physical examination upon admission revealed a well-developed adult male, who appeared somewhat apprehensive. Venous pulsations, 88 per minute, were visible in the neck. The pupils were equal and reacted to light and accommodation. The ears, nose, mouth, and throat were normal. The lungs were normal. There was slight præcordial bulging. The area of cardiac dullness was increased outward and to the left and downward to the sixth intercostal space. The point of maximal intensity of the heart beat was in the fifth intercostal space, 12½ centimeters from the midsternal line. There were forcible pulsations at the apex occurring about 24 to the minute. A soft blowing systolic murmur, which was transmitted outward to the axilla and upward toward the neck, was present at the mitral area. The aortic second sound was accentuated. Auricular systolic sounds could be heard anteriorly in the left

second intercostal space. Blood pressure: Systolic, 170; diastolic, 60. The abdomen and genitala were normal. There was a scar on the right shin. The skin was pale and moist; reflexes were normal.

The urine was normal. Blood Kahn test was four plus. Chemical examination of the blood resulted as follows: Nonprotein nitrogen, 54.5 milligrams; urea nitrogen, 25 milligrams; uric acid, 6 milligrams; creatinine, 4 milligrams; sugar, 100 milligrams.

An electrocardiogram taken June 1 showed complete heart block with auricular rate 99 and ventricular rate 23. The Q–R–S interval was 0.14 second.

In view of the positive Kahn test it was considered that antisyphilitic treatment was indicated. Potassium iodide in increasing doses, bismusol intromuscularly, and mercury rubs were started. Arsenicals were considered contraindicated.

Progress notes.—Following admission to hospital he continued to have rather frequent Adams-Stokes attacks during which his ventricular rate dropped to seven or eight per minute. These seizures were usually preceded by the warning signs of dyspnea and vertigo, and he soon learned to recognize their approach. His symptoms were relieved and attacks were aborted or prevented by the administration of 1 cubic centimeter of 1 to 1,000 epinephrine subcutaneously in conjunction with oxygen inhalations. In the intervals between attacks his pulse rate averaged about 24 per minute and never rose above 30. About the middle of June he had a series of severe attacks. On June 18, after a short course of digitalis administered rather intensively, an electrocardiogram showed no essential change except that the ventricular rate showed slowing to 11 per minute at times. One ventricular systole arose in a focus other than the usual one. Oral administration of barium chloride, one-third grain three times daily, was begun and continued for 10 days. While this drug was being administered, perceptible improvement was noted. The attacks occurred less frequently and were more transitory. After the first two days they ceased and did not recur until after the drug was stopped.

At the end of 10 days' treatment with barium chloride its regular administration was discontinued and the patient was given the drug whenever he noted the characteristic premonitory symptoms which usually ushered in an attack. During the early days of July he had a few mild attacks, but none of these compared in severity with those prior to the administration of barium chloride. On July 5 he was given ephedrine sulphate, 25 milligrams, four times a day for one week. During the next two weeks he received 25 milligrams three times a day. This drug wholly controlled the attacks from the beginning of its administration. After July 25 ephedrine was given only with the advent of premonitory symptoms. All antisyphilitic treatment was discontinued at that time.

An electrocardiogram taken July 30 showed the ventricular rate to average about 25, but its rhythm was irregular and the shifting focus previously noted was still present. The patient continued to feel very well. He had only a few mild attacks during the month of August. On account of this, late in the month he was given 25 milligrams of ephedrine twice a day. On August 9 he developed severe abdominal pain. The physical signs and laboratory data strongly suggested renal infarction due to embolism. The symptoms subsided in a few days.

By the middle of September the syncopal attacks ceased and he began to feel much better. Early in October he was allowed up each day in a wheel chair. About that time the frequency of ephedrine administration was reduced

so that he received 0.025 gram once daily, usually at night. No further attacks occurred until October 28 when, while sitting in bed talking to some other patients, he suddenly lost consciousness. Epinephrine hypodermically and oxygen inhalations were given but he died in a few seconds.

Necropsy was performed by Lieut. Commander H. E. Ragle, Medical Corps, United States Navy.

Necropsy protocol

The body is that of a well-developed, well-nourished white male about 35 years of age.

Chest.—Pericardial sac contained 60 cubic centimeters clear straw-colored fluid; all chambers of the heart large and full of dark fluid blood; musculature of left ventricle averages 15 millimeters in thickness and shows a moderate fibrosis; musculature of left ventricle rather flabby; valves good; coronaries not sclerosed and are patent, there are, however, several areas of narrowing; ascending aorta normal; arch and descending aorta show numerous yellow plaques in linear arrangement; left lung adherent posteriorly by fibrous adhesions; right lung free, cut sections show some congestion and œdema; peribronchial glands markedly enlarged.

Abdomen.—Gastro-intestinal tract normal; liver somewhat swollen, cut section shows passive congestion almost to the nutmeg stage; spleen normal; gall bladder normal; pancreas normal; right-kidney capsule stripped easily, cut section shows acute congestion, markings good; left kidney the same as the right except that in the center of the greater curvature there is an old fibrous infarct 40 by 20 by 6 millimeters; ureters normal, urinary bladder and prostate normal; calvarium not opened.

Gross pathological diagnosis.—(1) Myocarditis, chronic; (2) chronic passive congestion of liver and kidneys; (3) syphilitic aortitis; (4) chronic fibrous pleurisy; (5) passive congestion and œdema of lungs.

On section the myocardium showed considerable fibrous change and a few areas of round cell infiltration.

COMMENT

Three drugs, epinephrine, barium chloride, and ephedrine were apparently effective in preventing ventricular standstill. It is significant that while ephedrine was being administered as frequently as twice a day no Adams-Stokes attacks occurred. It was considered fortunate in this case that the occurrence of symptoms giving warning of an oncoming seizure seemed to make unnecessary frequent administration of the drug. The terminal attack, however, came without warning almost 24 hours after the last previous dose. It is not unlikely that this attack would have been prevented and the patient's life prolonged by continuous and more frequent use of ephedrine.

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PRIMARY CARCINOMA OF INTRAHEPATIC BILE DUCTS

Report of Case

By Lucius W. Johnson, Captain, Medical Corps, United States Navy, and W. W. Hall, Lieutenant Commander, Medical Corps, United States Navy

We have to report a case which we believe to be one of the rare primary carcinomas of the intrahepatic bile ducts arising in an obstructive biliary cirrhosis and finally producing an almost complete obstruction and a very high-grade jaundice. It is interesting to note that as the symptoms gradually developed the patient sought medical advice, was thoroughly examined by competent men, and nothing could be found on which to base a diagnosis.

Case Report

A man, aged 54, was admitted to the hospital complaining of progressive, painless jaundice of about two months' duration.

About four months before admission he began to feel that he was not so well as usual, but was unable to describe any definite symptoms. He was submitted to a thorough physical examination on the hospital ship, with complete laboratory examinations. No defect whatever was found.

About two months before admission he noticed that he had lost his appetite and that his urine was highly colored. This was followed by urticaria, muscle pains, and general malaise. He took calomel and salts but there was no improvement. A few days later he noticed a yellow color of his skin and his stools became clay-colored. The jaundice increased gradually but he had no pain, fever, chills, or sweats. These symptoms developed slowly during a period of about five weeks and at the end of that time he was admitted to the sick list and sent to the hospital ship, where he was again thoroughly studied.

The liver was found slightly enlarged, the edge being about two finger-breadths below the costal margin. No irregularity could be felt and there was no tenderness on pressure. The rest of the physical examination was essentially negative. The icterus index was 200 and varied, during his stay, between 190 and 330. Examination of the feces showed no bile present. The urine was highly colored and contained bile but no albumin or casts. The blood picture, on three occasions, was within normal limits. The Kahn test was negative. A flat X-ray picture of the abdomen showed an indefinite mottling in the gall-bladder region. The gastrointestinal X-ray series showed spasticity in the ileocecal region and a filling defect in the pyloric area. During his stay on the hospital ship gaseous distention of the intestines and dyspeptic symptoms became pronounced.

On admission to the hospital his icterus index was 220, the Van den Bergh reaction was immediate and direct with a quantitative of 225 milligrams per liter. The temperature, pulse, and blood picture were approximately normal.

We agreed with the doctors on the hospital ship that the diagnosis lay between cancer of the head of the pancreas, causing obstruction of the common bile duct; cancer of the ampulla of Vater; a silent stone in the common duct; and chronic pancreatitis.

After giving intravenous calcium chloride for three days to reduce the danger of hemorrhage, operation was done under spinal anesthesia. Sodium amytal was used as preliminary medication. Transfusion of 500 cubic centimeters of citrated blood was given during the operation. All the tissues were found to be deeply bile stained. The liver appeared somewhat smaller than normal, and the middle portion seemed harder than the edges, though similar in color. The gall bladder was soft, thin walled, and not distended. The common duct was somewhat larger than normal. In the head of the pancreas was a hard mass about the size of a hen's egg, surrounding the common bile duct. A cholecystogastrostomy was done. On opening the gall bladder it was found to be filled with lemon-colored mucus.

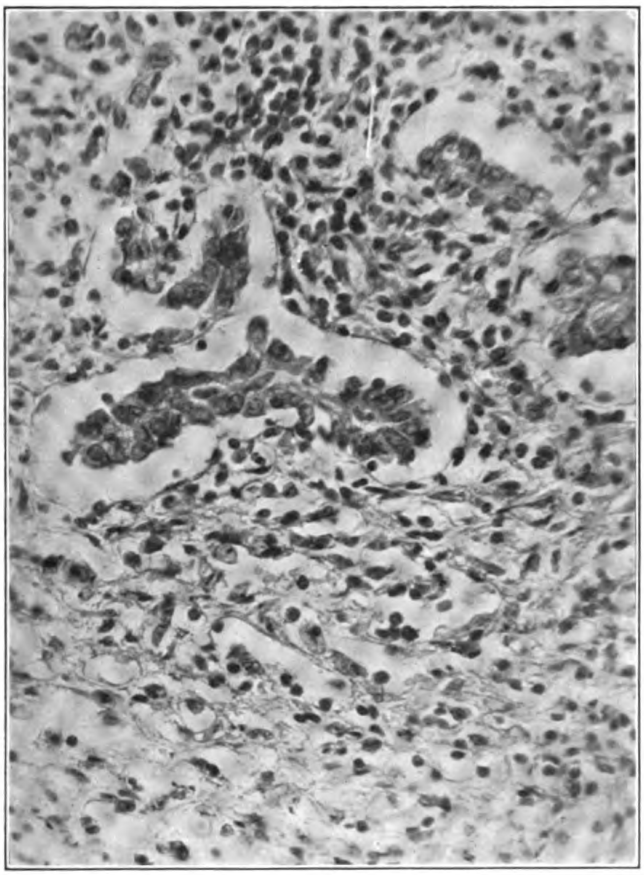
We interpreted the lack of distention of the gall bladder and common duct with the finding of lemon-colored mucus in the gall bladder as indicating that the trouble was within the liver itself and not obstruction of the extrahepatic ducts.

The day after the operation found the patient very well and he continued to improve for several days. He ate well and his stools changed from clay color to the normal brown, but the jaundice did not decrease. On the eleventh day after the operation the last of the sutures were removed and, a few hours later, the wound edges separated, so that it was necessary to resuture them. He died on the thirteenth postoperative day.

Autopsy findings

At autopsy the anastomosis was found to be patent and there was no leakage. The stomach and duodenum were opened and explored. No abnormality was noted. The small and large intestine presented no abnormality. The common bile duct, the cystic and hepatic ducts were opened and showed no evidence of obstruction. The ampulla of Vater was normal and patent. The mass in the head of the pancreas was sectioned and found to be hard and fibrotic, but the appearance of the cross section was similar to that of the rest of the organ. It did not obstruct the common duct. The liver weighed 1,300 grams. The organ showed increased resistance to cutting.

Histopathological findings.—Microscopical study revealed the pancreatic acini normal in size and number; the fibrous interlobular stroma was considerably increased in amount. The liver showed a cirrhosis of the obstructive biliary type with some proliferation of the bile ducts, some typical, others rather atypical, in structure. Toward the margin of the liver and along the inferior surface of the right lobe there was a zone about 1 centimeter in width (which appeared light in color grossly), which was composed entirely of a scirrhous carcinoma. The fibrous stroma was abundant; the epithelial masses scattered through the stroma represented more or less atypical ductlike structures. The majority of these showed no lumen or only a suggestion of a lumen, occasionally solid masses of cells were encountered.



PRIMARY CARCINOMA OF INTRAHEPATIC BILE DUCTS

High power field showing atypical duct structures and abundant stroma. (Johnson and Hall.)

Comment

Ewing (Neoplastic Diseases, third edition) says of multiple carcinoma of the intrahepatic bile ducts: "Eggel found that 32 per cent of primary carcinomas of the liver were of this origin. Pepere on much the same data reduced the proportion to 14 per cent. * * * In the literature definite reports of bile-duct carcinoma are much less common than cases of liver-cell carcinoma. * * * Bile-duct carcinomas appear chiefly in adult life, in subjects of cirrhosis. A history of severe disturbance in evacuation of bile * * * is relatively common. Gross anatomy: The liver is usually enlarged, intensely jaundiced, and often the seat of advanced cirrhosis, chiefly of the biliary type. The tumor process affects most or all of the organ, and produces very numerous, usually small, firm nodules which may become confluent. Fischer found the most extensive growth in the region of the hilus. * * * In most cases it is impossible to locate any single primary focus, and the origin is multicentric. * * * The outlying portal canals commonly exhibit marked proliferation of bile ducts as in biliary cirrhosis * * *. From such precancerous overgrowth every stage up to true adenocarcinoma has been traced. * * * Metastases are not commonly observed except with the very active tumors."

Our case, we believe, represents one in which biliary obstruction began as a result of the biliary cirrhosis and finally became almost complete with the gradual constriction of the tributary hepatic bile ducts by the scirrhous carcinoma primary in the intrahepatic bile ducts.

The observation of bile in the stool in the postoperative period indicates that the obstruction was not absolute. The liver in this case was not enlarged and the carcinoma did not occur in nodules as is usually the case.

CONGENITAL POLYCYSTIC KIDNEYS

Report of Two Cases

By R. G. Davis, Commander, Medical Corps, United States Navy

Although congenital polycystic disease is the most common type of cystic degeneration attacking the kidneys, the condition is comparatively rare. The first case reported in this paper reveals some variations from the average textbook picture, such as insidious progress to almost complete destruction of kidney tissue, remaining wholly localized without marked signs of chronic interstitial nephritis and terminating suddenly in uremic dyspnea. The second case also terminated suddenly, but was due to hemorrhage into the

pons. Cystic degeneration of the liver and cerebrum was associated with that of the kidneys. The disease is met with in infants, at times requiring the destruction of the foetus before delivery; but is rare in childhood. Most of the cases occur between 40 and 60, and the condition is nearly always bilateral with one kidney usually more affected than the other. The etiology is unknown, but several theories have been advanced, such as inflammation and plugging of the tubules, cyst adenomas, and congenital malformations. Embryologically the secretory and collecting tubules develop from two different layers of the Wolffian system. Failure to later unite and form a channel is considered a possible cause of cyst formation. No part of the kidney tissue is immune from attack, and the size of the kidney depends upon the number of cysts scattered through it. The normal contour is usually maintained in spite of the hypertrophy and surface cysts cause a knobby appearance. The parenchyma is thinned by pressure atrophy of the cysts containing serous or bloody material. Chemically the cystic fluid has been found to contain albumin, blood crystals, cholesterin, triple phosphates, and fat droplets. Urea and uric acid are rarely found. Flattened epithelium line the cysts. Malformations are often produced elsewhere in the urinary tract, and the liver is commonly cystic. The symptoms are usually those of dull pain, hematuria, and signs of chronic interstitial nephritis. Enlargement of the kidneys may be the first sign for months or years, but pain is eventual from capsule stretching, paroxysmal at first and later continuous, dull dragging in character, increased on exertion and relieved on lying down. When hematuria is present renal colic may be produced by the passing of clots. It would seem an easy matter to palpate the irregular surfaced tumors particularly in extensive hypertrophy, but the intervening colon makes palpation and percussion misleading. Hematuria may be the initial symptom varying from occasional intermittent traces to a profuse flow proving fatal. The pressure of the cysts produces a chronic nephritis associated with arteriosclerosis and hypertension. The urine is increased, specific gravity lowered and relatively fixed, and contains blood, albumin, and casts. After a gradual progressive course (lasting 36 and 35 years in the cases here reported) death occurs suddenly from uremia or circulatory complications such as cerebral hemorrhage. The diagnosis may be difficult, as the growth is slow, but it is based on bilateral enlargement of the kidneys associated with pain and nephritis; however, the latter may so dominate the picture that the tumors escape recognition. Renal function tests and pyelography are aids in the diagnosis, and malignancy is suspected if the tumor is unilateral. The treatment is essentially that of chronic nephritis. Surgical puncture of the cysts may be done, but the mortality is high. Surgery should be reserved for profuse

hemorrhages or infections such as perinephritic abscess and peritonitis. The disease is rapidly fatal in the new born but may be latent for years in the adult.

Case Reports

Case I.—C. T. M., age 36, Veterans' Administration patient, was admitted February 8, 1932, for observation complaining of weakness and fatigue of six months' duration. Occupation, ironworker.

Past and family history.—Negative.

Present history.—For the past 18 months, following a heavy cold, general weakness of lower extremities and easy fatigability has been noted. Some dyspnea upon exertion has been present for several years and the past three months swelling of the right ankle has been present. Thinks he had symptoms of uremia one year ago.

Physical examination.—Appearance, pale; development, slender; expression, anxious; weight, 150 pounds (normal, 155 pounds); skin, dry; no adenopathy; head, neck, and thorax, negative; abdomen is distended; no rigidity or tenderness. There is a definite, hard, slightly movable mass on the right side at the level of the umbilicus which does not move with respiration. Irregular in outline and measuring about 15 by 8 centimeters. No evidence of liver tissue between the mass and the costal margin. There is a mass on the left side which appears to be notched and part of the spleen, but descending colon makes differentiation indefinite. Nervous system: Reflexes sluggish; left knee jerk decreased when compared with the right; Romberg and Babinski, negative. Genitalia, negative except for an old scar on penis.

February 11, 1932: Electrocardiogram denotes myocardial change.

February 12, 1932: X ray of lungs and heart, negative; no ova or parasites in stool; Van den Berg, delayed; icterus index, 6; red blood corpuscles, 2,370,000; white blood corpuscles, 5,700; Hb., 55 per cent; polys, 81; lymphs, 1; eosins, 2; urine: albumin, one plus. Kahn test, two plus.

Clinical impression.—Syphilis of the liver, bilateral cystic kidneys; malignancy of liver, colon, or kidneys.

February 18, 1932: Urea nitrogen, 28 milligrams per 100 cubic centimeters; creatinin, 2.1 milligrams per 100 cubic centimeters; Kahn, three plus.

February 20, 1932: Patient developed a chest cold; lungs clear except for few subcrepitant rales, right side; throat slightly injected.

February 21, 1932: Patient suddenly became very dyspneic; heart sounds of fair quality; pulse, regular; rate, 110; temperature subnormal; right chest filling up; death occurred suddenly.

Autopsy report

The body was that of a well-developed, fairly well nourished white male, apparent age 35 years, and has been embalmed. No significant external findings were noted.

Chest.—Pleural cavities free from fluid. Both lungs appeared normal throughout, except for slight emphysema.

Heart.—The pericardial cavity contained no free fluid. The heart was fairly large, but within normal limits. The myocardium appeared normal. There were small atheromatous plaques on the mitral valve and in the ascending portion of the aorta. The coronary arteries showed a very slight sclerosis, as did all the arteries of the body.

Abdomen.—Liver, spleen, pancreas, adrenals appeared normal.

Kidneys.—Both kidneys were polycystic and greatly enlarged. The right was approximately 5 by 5 by 14 inches and weighed 2,670 grams. The left kidney was 5 by 5 by 11 inches and weighed 2,070 grams. On section there was almost total absence of kidney structure. All tissue studded with small and large multilocular cysts.

Pathological diagnosis.—(1) Bilateral polycystic kidneys; (2) early generalized arteriosclerosis.

Case II.—W. D. G., chief electrician's mate, age 35, was admitted September 14, 1929, complaining of numbness over the entire right side of body, mental haziness, impaired vision of right eye, impaired hearing and general weakness.

Past history.—Fifteen years' service. Operated upon in May and August, 1924, for cysts of the liver. At that time cysts of the kidney were noted. Some of the liver cysts were evacuated, but the kidney cysts were not touched. On the sick list from March to November. Since that time has been able to do duty as long as he was careful about over exertion. He had attacks of severe deep-seated pain in back and on left side with frequent vomiting. Headaches were frequent. No attacks of vertigo, no shortness of breath, no definite gastrointestimal symptoms, except vomiting. Always exceptionally well until onset of present condition. Has had two indefinite attacks of jaundice. Blood pressure has been high for several years, ranging from 230/170 to 204/160. Two plus albumin has been noted in the urine. Specific gravity slightly below normal. Patient has a congenital polycystic condition of both kidneys and liver associated with a marked arterial hypertension and present attack is probably result of a small cerebral hemorrhage on the left side.

Physical examination.—Well-developed, well-nourished man of 35, mentally clear and rational.

Head and neck.—Negative, except impaired vision and hearing.

Chest.—Symmetrical, normal mobility.

Heart.—Moderate enlargement to the left. Apex beat forceful. Rythm regular, rate normal. No murmurs or thrills. Blood pressure 208/160.

Lungs.—Negative.

Abdomen.—Old surgical scar right upper abdomen. Upper abdomen distended and filled with a firm, hard, nodular mass emerging from right costal margin and extending to the umbilicus. Upper border of liver dullness at fourth rib. Left kidney palpable, enlarged, and tender. Tenderness over liver.

Genitalia.—Negative.

Nervous system.—Pain, temperature, and light-touch sensation diminished over right half of body and right arm and leg. Grip in right hand weaker than left. Slight weakness of right leg. Reflexes normal.

Placed on general régimé for chronic nephritis. Blood chemistry remained essentially normal. Albumin from 1 to 2 plus. Phenolsulphonephthalein output 45 per cent. Showed improvement in general condition at the end of three months, but hypertension remained constant. Surveyed to continue treatment. During the next three months a chronic appendix was removed and 16 teeth were removed due to periapical abscesses. Nephritis increasing.

June 28, 1930: Died suddenly.

Autopsy report

The body presented for examination is that of an adult white male, about 35 years of age. Rigor mortis is present. The body is embalmed. The pupils are equal and dilated; the nose, ears, and mouth are negative. The abdomen is full, and a mass can be palpated in the upper left quadrant. There is a right rectus scar about eight inches in length. The skin and subcutaneous tissue are normal.

Abdomen.—On opening the abdomen, numerous adhesions are found. The diaphragm is high, the dome on the right side reaching to the level of the third rib and including half of the right chest cavity. The left chest is occluded in practically the same manner.

Lungs.—The left lung is free. The right lung is bound by adhesions. The left lung is normal in size, contains air throughout and floats in water. There is moderate anthracosis. The right lung is slightly smaller than normal. There are interlobar adhesions. It contains air throughout.

Heart.—There is a normal amount of pericardial fluid. The heart is larger than normal. The left ventricle is markedly hypertrophied, and there is slight sclerosis of the mitral valves. The aortic valves and aorta are normal. The coronaries are patent and slightly sclerosed.

Liver.—The liver is firmly bound in the upper abdomen to the anterior abdominal wall, diaphragm, transverse colon, the stomach and the ascending colon. It is markedly enlarged, being half again as large as normal. It is covered with cysts varying in size from a millet seed to that of a baseball. The cysts are multilocular and extend throughout the liver substance. The edge of the liver is rounded. The right side of the dome of the liver is one mass of cysts. The gall bladder is absent.

Spleen.—The spleen is slightly enlarged. The capsule strips easily; on section it appears engorged.

Kidneys.—The left kidney is three times as large as normal, and is studded with cysts. The capsule is adherent. On cutting into one of these cysts, a brownish-red fluid is evacuated. On section, the cortex and medulla are seen to be obliterated by cysts which take up the entire kidney substance. The right kidney presents the same picture. The pelvis is very small.

Bladder.—Distended and contains approximately 500 cubic centimeters of clear urine. The mucosa is normal.

Prostate.—Normal.

Pancreas.—Normal.

Brain.—The calvarium is normal in thickness. The cerebral hemispheres appear normal. The base of the brain is covered by a recent hemorrhage. The arteries are markedly sclerosed. The pons is very soft and fluctuates, and on opening contains about 10 cubic centimeters of blood clot. The right side of the brain appears normal. On opening the ventricle of the left brain, a cyst, about 4 centimeters long and 2 centimeters wide in the region of the corpora striatum and just lateral to the internal capsule, is seen. It contains a brownish exudate; the rest of the brain is normal.

Pathological diagnosis.—(1) Multiple cysts of the kidneys and liver; (2) hemorrhage into pons; (3) cystic degeneration of the left cerebrum.

Immediate cause of death.—Hemorrhage into pons.

Comment

It seems remarkable that such an extensive destruction of a vital organ can be present with little or no symptoms. Eisendrath and Rolnick report that the kidney normally uses one-seventh of its capacity, with the remaining six-sevenths in reserve. The usual sudden termination either by uremia or circulatory complication is well illustrated in the above cases. Only one of the cases reported exhibited nephritic symptoms, but not to a marked degree.

TORSION OF THE SPERMATIC CORD WITH GANGRENE OF THE TESTICLE AND EPIDIDYMIS

Report of Case

By French R. Moore, Lieutenant, Medical Corps, United States Navy

Torsion of the spermatic cord with gangrene of the testicle and epididymis is infrequent. One hundred and twenty cases had been reported in the literature up to 1920 (1). Since then, 17 cases have been added, all from the Bellevue Hospital, New York.

Torsion of the cord is usually seen in young adult life, but may occur at any age. The torsion may be incomplete and subside spontaneously, or complete, resulting in gangrene of the testicle and epididymis.

The etiology is based upon congenital malformation affecting the anchorage of the testicle and epididymis to its tunical bed. Scudder (2), who collected 32 cases, found that 47 per cent occurred in cryptorchids. Torsion usually follows a severe strain but may occur during sleep. Two types of torsion have been described by Young (3): (*a*) The extravaginal in which the testicle, epididymis, and tunica rotate as one mass within the scrotal wall, twisting the cord in its extravaginal position; and (*b*) the intravaginal, where a long, poorly developed mesorchium allows the testicle and epididymis to twist around the cord. The latter type is the more common.

The pathology seen is a moderate unilateral enlargement of the scrotum. There is moderate edema and thickening of the skin and subcutaneous tissues. When the tunica is opened the testicle and epididymis are found to be gangrenous, and a few drams of reddish black fluid escapes. Torsion of the cord is seen near the lower pole of the epididymis or perhaps involving part of the epididymis. The cord may have one or more twists.

The onset of symptoms is sudden, with severe pain, followed by moderate swelling of the testicle, and there is low-grade fever. There may be nausea and vomiting. Pain subsides after the first day and the patient may only complain of moderate swelling and slight pain on palpation of the testicle. Torsion of the cord may be differentiated from acute epididymitis by finding the testicle and not the epididymis enlarged, and absence of a lower urinary tract infection. When torsion of the cord occurs in undescended testicle the diagnosis between this and strangulated hernia may be difficult, but with the absence of the testicle in the scrotum the diagnosis should be reasonably certain.

The treatment is orchidectomy, unless the case is seen immediately, when an attempt may be made to untwist the cord. At operation

the other testicle should be sutured to its bed to prevent loss of the remaining testicle by a similar misfortune.

Case Report

A. N. G., seaman, second class, United States Navy, age 18, was admitted to the urological service, United States naval hospital, Mare Island, Calif., April 19, 1932, complaining of pain and swelling of the right testicle. Upon arising three days before he was seized with severe pain in the testicle. The pain gradually decreased to a dull ache in the course of a few hours. Swelling appeared shortly after onset of the pain; increased the first day; and then remained stationary.

Past history.—No history of a venereal disease. One year before he experienced a similar attack lasting a few hours. The next day the testicle was normal.

Physical examination.—Temperature, 99° F.; pulse, 82. The right side of the scrotum was moderately swollen and edematous. The right testicle was twice its normal size and slightly tender. It did not transilluminate. The epididymis was not enlarged. There were no signs of urinary-tract infection.

Diagnosis.—1. Torsion of the spermatic cord. 2. Hematocele.

Treatment.—Under spinal anesthesia the right side of the scrotum was incised. Upon opening the parietal tunica, two drams of chocolate-colored fluid escaped. The testicle and epididymis were gangrenous and twice their normal size. The cord was twisted in one complete turn at the lower pole of the epididymis. Orchidectomy was performed and the other testicle was sutured to its bed. Convalescence was uneventful.

Conclusions

1. Torsion of the spermatic cord with gangrene of the testicle and epididymis is infrequent.
2. Differentiation from acute epididymitis is to be made by finding the testicle and not the epididymis enlarged and by the absence of a lower urinary tract infection.
3. Orchidectomy is required unless the patient is seen soon after torsion has occurred, when an attempt may be made to untwist the cord.

REFERENCES

(1), (2) Lewis: Practice of Surgery, vol. 9 (Urology), ch. 28. (3) Young: Practice of Urology. 1926, vol. 2, p. 164.

DISSECTING ANEURYSM

Report of an Unusual Case

By R. G. Davis, Commander, Medical Corps, United States Navy, and W. W. Hall, Lieutenant Commander, Medical Corps, United States Navy

Case Report

A man aged 45 years, who had been retired about two years previously for arterial hypertension, was admitted to the United States naval hospital, San

Diego, Calif., at 12.30 p. m., May 6, 1932, with the diagnosis of poisoning, lead, acute (ethyl gasoline), not misconduct. Complaints were acute abdominal cramps and loss of sensation of left leg.

At about 10.30 a. m., that date, while attempting to syphon some ethyl gasoline from the tank of his car by mouth suction he accidentally swallowed a mouth full of the liquid and within 10 minutes began having acute stomach distress. On his way to a civilian doctor he stopped at a drug store, where milk and eggs followed by mustard were given. Violent vomiting resulted. Following this, a stomach tube was passed, lavage performed, and one ounce of saturated solution of magnesium sulphate left in the stomach.

Blood pressures since retirement had been 180/126 to 190/110. Patient had been free from any illness, except occasional headaches on exertion. Headaches and dizziness have been more frequent the past two weeks before the present admission.

Physical examination on admission revealed acute abdominal pain, loss of sensation of left leg below the knee, and paraesthesias of left leg. Pupils normal. Ears, nose, mouth, negative. Mucosa pale; no excoriation of lips or tongue. Neck negative. Chest negative. Heart tones regular and of good quality. Blood pressure 120/80. Abdomen moderately rigid, not tender. Extremities: Deep reflexes absent, left patellar and Achilles regions. All other deep reflexes hyperactive. Marked ankle clonus on right, none on left. Vessels—general arteriosclerosis. Blood: Red blood corpuscles, 4,310,000; hemoglobin, 80; white blood corpuscles, 9,600; polymorphonuclears, 69; lymphocytes, 31. Red and white cells normal in morphology and staining reaction. Routine treatment of acute lead poisoning instituted; viz, lactates, milk, heat, and sedatives.

Four hours after admission patient was unable to move left leg. Absence of pulsations noted in left dorsalis pedis and posterior tibial arteries. Intermittent nausea and increased abdominal pain more acute in left lower quadrant. Thrombi of iliac, femoral, and mesenteric vessels were considered. Blood pressure rose at this time to 265/90. Blood examination: White blood corpuscles, 13,600; polymorphonuclears, 90; lymphocytes, 9. Temperature, 101; pulse, 112; respirations, 40. Urine: Dark amber color; specific gravity, 1.026; albumin, double plus; sugar, negative; many erythrocytes, few leucocytes; lead negative. Patient became somewhat irrational during the night, but general condition remained good until 7.30 a. m., when he became more restless, went into shock, left eye dilated widely, and death occurred at 8.20 a. m.

Autopsy Report

Chest.—Each pleural cavity contains some excess of blood-tinged fluid.

Lungs.—On section, both lungs show dark red, raised areas, which are interpreted to be early infractions. The intervening pulmonary tissue is greatly congested.

Heart.—Pericardial sac contains approximately 100 cubic centimeters of bloody fluid. The tissue around the root of the pulmonary artery and aorta is filled with blood. No definite break in the epicardial layer, accounting for the appearance of blood in the pericardial sac, is noted, however. On removing the heart and several centimeters of aorta, there is seen to be a split in the intima of the aorta which apparently has begun at a softened atheromatous spot about 3 centimeters above the aortic valve. This split has continued sharply, as though cut with a knife, around the entire aorta, allowing the lower section of the intima and a portion of the media to retract downward as an elastic cuff with curled-over edges. The dissection of the aortic wall has

progressed downward into the interventricular septum and there is a split in the endocardium of the right ventricle 2 centimeters long, through which the intramural aortic split communicates with the right ventricular cavity. A thrombus is present in the right auricle, the probable source of the pulmonary emboli. The aorta above the point thought to be the primary break is split into two tubes, the inner elastic and the outer largely adventitia. The two layers of the aorta are separated in an irregular fashion all the way around the arch and down through the thoracic and abdominal aortas. The left common iliac also shows separation and between the inner and outer tubes in the left common iliac and in the left common carotid, an accumulation of clotted blood sufficiently large to collapse the inner vascular tube and occlude its lumen, is present. The amount of atheromatous change present in the aortic arch and thoracic aorta is only moderate for a man of 40 years of age. The aorta still retains considerable elasticity.

Abdomen.—There is some increase of peritoneal fluid present and there is a definite tinge of blood throughout. No source of hemorrhage was located, however. There is a small area of marked injection on the margin of the omentum. The loops of the small intestines are quite dark and on opening are seen to be filled with blood-stained fecal contents. The mesenteric vessels were examined and no area of infarction discovered. The liver is soft and shows the nutmeg mottling of a passive congestion. The spleen is congested. The kidneys show no gross abnormalities other than congestion. The pancreas is negative. Pelvis is negative.

Head.—The dura is densely adherent to the calvarium. The brain shows no gross abnormalities. The cerebral arteries show large atheromatous plaques.

Pathological diagnosis.—1. Dissecting aneurysm of aorta. 2. Occlusion of left common carotid and left common iliac by intramural thrombi. 3. Intracardial rupture (right ventricle) of dissecting aneurysm. 4. Pulmonary infarction.

Comment

So-called dissecting aneurysms occur when the intima or both the intima and media tear. These tears usually occur at the time of some sudden rise in blood pressure, such as occurs when lifting some heavy burden, and develop at a weakened spot when the fatty degenerated atheromatous intima parts. Cases have been reported as developing after great emotional excitement or terror. (Kaufmann's Pathology.) MacCallum (Textbook of Pathology, fourth edition) says: "Such a tear is most common in the arch of the aorta and has sharp edges as though cut with a knife. In the sclerotic arteries, in which it most often happens, it is easy to split the wall in the middle of the medial coat, and blood presses into the space thus formed and burrows its way down the length of the aorta and along the branches, converting each as far as it goes into two tubes, one within the other, the inner formed of intima and half the media, the outer of the other half of the media and the adventitia."

Aside from the rarity of such dissecting aneurysms, our case presents some unusual features; viz, firstly, the dissection downward

from the root of the aorta into the interventricular septum and rupture into the right ventricle below the pulmonary valve; secondly, the great extent of the separation involving the entire aorta and its major branches at each extremity; thirdly, the occlusion of the left common carotid and left common iliac by intramural thrombi.

The toxic effect of the ethyl gasoline swallowed obviously was nil. It simply initiated a train of circumstances (fear, excitement, the administration of emetics and gastric lavage, retching, and vomiting) which served to sharply raise an already dangerously high blood pressure.

NAVAL RESERVE

MEDICAL CORPS

APPOINTMENTS, THIRD QUARTER, 1932

Name	Rank	Appointed
Cellino, Joseph F	Lieutenant (junior grade), MC-V(G)	June 11, 1932
Kles, Benjamin B	do	May 23, 1932
Longstreth, Clyde M	do	June 14, 1932
Michael, Paul	do	May 27, 1932
Parsons, William C	do	Feb. 11, 1932
Zimmerman, Edward F	do	June 1, 1932
Reinertsen, Bernhard R	Lieutenant, MC-V(8)	May 31, 1932
Kalez, Marion M	Lieutenant (junior grade), MC-V(G)	July 7, 1932
Hornthal, Henry A	do	Aug. 9, 1932
McCaffery, Glenn	do	Aug. 4, 1932
McGrail, M. A	do	Aug. 1, 1932

PROMOTIONS

Name	From—	To—
Cowlbeck, Harry D	Lieutenant (junior grade), MC-V(G)	Lieutenant, MC-V(G).
Haight, Harry H	do	Do.
Agnew, John Robert	do	Do.
Hall, Llewellyn	do	Do.
Keck, Herman	Lieutenant (junior grade), MC-F	Lieutenant, MC-F.
McCartney, James L	Lieutenant (junior grade), MC-V(G)	Lieutenant, MC-V(G).
Whalen, John M	Lieutenant, MC-F	Lieutenant commander, MC-F.
Bleasby, Charles B	do	Do.
Bowser, Frank E	Lieutenant (junior grade), MC-F	Lieutenant, MC-F.
Childress, Calvin H	Lieutenant, MC-F	Lieutenant commander, MC-F.
Cooke, Harry H	Lieutenant (junior grade), MC-V(G)	Lieutenant, MC-V(G).
Ellet, Wm. C	Lieutenant (junior grade), MC-F	Lieutenant, MC-F.
Hemmingsen, T. Charles	Lieutenant, MC-V(G)	Lieutenant commander, MC-V(G).
Ivews, W. C	do	Do.
Thomas, Charles C	Lieutenant (junior grade), MC-V(G)	Lieutenant, MC-V(G).

TRANSFERS

Name	From—	To—
Garnett, Herman M	Lieutenant (junior grade), MC-V(G)	Lieutenant (junior grade), MC-F.
Zimmerman, Edward F	do	Do.
Stedman, Harold E	do	Do.
Watkins, Harry C	do	Do.

DENTAL CORPS

APPOINTMENTS, THIRD QUARTER, 1932

Name	Rank	Appointed
Levin, David	Lieutenant, DC-V(8)	Sept. 7, 1932
Otto, Bertram B	Lieutenant (junior grade), DC-V(G)	Sept. 15, 1932
Proctor, Charles M	Lieutenant commander, DC-V(8)	July 11, 1932

PROMOTIONS

Name	From—	To—
Beazley, William A	Lieutenant (junior grade) DC-V(G)	Lieutenant, DC-V (G).
Carbinier, Charles F	do	Do.
Denney, Frank C	Lieutenant, DC-V(G)	Lieutenant commander, DC-V(G).
Foppert, Edwin C	Lieutenant (junior grade), DC-V(G)	Lieutenant, DC-V(G).
Graham, DuVern E	do	Do.
Hoke, Leslie L	do	Do.
Huguley, Herbert T	do	Do.
Levy, Joseph L	do	Do.
Pepin, Ernest M	do	Do.
Plagman, Archibald A	do	Do.
Shone, Lloyd B	do	Do.
Woodworth, Earl B	do	Do.

NOTES AND COMMENTS

NAVAL MEDICAL BULLETIN

On account of the decrease in the appropriation for printing and binding, the BULLETIN, during the fiscal year 1933, will contain less pages than heretofore, and can contain only a very limited number of illustrations. Authors may expect to find their articles appearing with fewer illustrations than those submitted. The section heretofore devoted to book notices has been discontinued.

It is hoped, of course, that the appropriation for the next fiscal year will permit the BULLETIN to resume its former size and some day even to appear as a monthly, as it has done during some periods in the past.

LETTERS OF COMMENDATION

In accordance with the statement made in the preface of the BULLETIN, the Surgeon General of the Navy appointed a board to select the papers published in the BULLETIN during the year 1932 which it considered to be worthy of letters of commendation. As a result of the selections made by the board, letters of commendation have been sent to the officers named below for the papers which appear opposite their names:

Capt. G. F. COTTLE, Medical Corps, United States Navy, Mortality in appendicitis.

Lieut. R. R. KRACKE, Medical Corps, United States Naval Reserve, The significance of the leukopenias.

Lieut. Commander J. J. WHITE, Medical Corps, United States Navy, Carbon monoxide and its relation to aircraft.

Lieut. Commander I. B. POLAK and Lieut. Commander B. H. ADAMS, Medical Corps, United States Navy, Traumatic air embolism in submarine escape training.

Lieut. (Junior Grade) A. R. BEHNKE, Medical Corps, United States Navy, Analysis of accidents occurring in training with the submarine lung.

Lieut. Commander D. FERGUSON, Medical Corps, United States Navy, Reinvestigation of the value of active pneumococcus immunity—preliminary report.

ANNUAL REPORT OF THE SURGEON GENERAL, UNITED STATES NAVY, 1932

The Medical Department activities discussed in this report occurred during the fiscal year ended June 30, 1932. The statistics concerning morbidity and mortality rates and other data pertaining to health conditions of the Navy are for the calendar year 1931.

Death rate.—A total of 391 deaths occurred, making the death rate from all causes 3.47 per 1,000.

This is a slight advance over the rate for the preceding year (3.31) and is due almost entirely to the material increase in deaths from motor-vehicle accidents, practically all of which resulted from injuries received while the individuals were on leave or liberty. Deaths from this cause greatly exceeded those from drowning—which, prior to 1930, was the usual leading cause of death in the Navy.

The rate for drowning (35 per 100,000), while slightly higher than last year, was, however, with this exception, lower than in any year since 1887, the first year in which complete statistics were published in the Annual Report of the Surgeon General.

The death rate from injuries and poisons has exceeded that from diseases during each year since 1922. This year the death rate for injuries and poisons was 1.95 and for diseases 1.53.

The leading causes of death for the last three years are shown in the following table:

Leading causes and number of deaths

	1931	1930	1929		1931	1930	1929
Motor vehicle accidents	81	58	48	Influenza	10	4	14
Drowning (all causes)	40	37	50	Neoplasms	9	8	12
Suicides	29	32	29	Burn, multiple	9	9	6
Diseases of circulatory system	26	17	27	Intracranial injury	8	7	8
Pneumonia	23	14	15	Cerebrospinal fever	4	11	20
Tuberculosis	17	28	22	Syphilis	4	7	9
Abscess	10	7	8	Poisoning	3	11	14
Appendicitis	10	14	9				

Flight hazards.—There has been an almost progressive decline in aviation fatalities per unit of flying hours during the last 11 years. The following table shows the fatalities per 10,000 flying hours since 1920:

1921	9.8	1927	2.5
1922	3.8	1928	1.5
1923	3.2	1929	1.0
1924	6.9	1930	0.9
1925	3.7	1931	0.7
1926	2.3		

Morbidity rates.—The general admission rate per 1,000 from all causes was 565.92, as compared with 520.61 in 1930. This increase is

accounted for principally by the higher prevalence during the year, both afloat and ashore, of influenza and the group of so-called common respiratory diseases.

The number of sick days per person, however, was smaller than for any previous year since 1913, except 1917 and 1925.

There were 27 different conditions, each of which caused more than 10,000 sick days during the year. They are listed in the order of frequency of admissions. It is noted that, with the exception of bronchitis in 1931 and influenza in 1930, the same 10 conditions caused each more than 1,000 admissions for 1930 and 1931.

	1931		1930	
	Admissions	Sick days	Admissions	Sick days
Catarrhal fever, acute	10,693	57,034	8,188	49,712
Gonococcus infections	8,761	130,090	8,659	123,737
Accidental injuries and poisonings	7,188	153,368	7,240	154,340
Tonsillitis	4,356	60,365	4,957	67,655
Chancroid	3,739	14,097	4,033	17,319
Syphilis	2,871	62,013	2,940	66,470
Influenza	2,384	15,735	209	2,079
Cellulitis	1,783	18,349	1,882	23,364
Appendicitis	1,494	46,752	1,585	48,809
Bronchitis	865	21,184	1,141	28,690
Angina (Vincent's infection)	688	10,375	845	12,930
Malaria	674	13,583	447	12,337
Trichophytosis	587	16,443	495	16,236
Hemorrhoids	467	10,293	507	11,580
Cholangitis	450	11,823	433	10,910
Deviation, nasal septum	427	10,751	461	12,039
Bubo, inguinal (nonvenereal)	413	15,373	537	18,583
Otitis, media	408	12,655	441	14,722
Hernia	380	21,764	415	23,593
Arthritis	309	19,416	304	20,573
Pneumonia	225	12,154	253	12,300
Tuberculosis	192	35,979	225	45,451
Absence, acquired, teeth	170	13,253	190	13,384
Psychoneuroses	140	14,558	166	19,113
Ulcer, duodenum	123	11,544	100	12,523
Dementia præcox	57	14,866	69	25,870
Chancroidal lymphadenitis	46	11,127	80	12,655

With the exception of catarrhal fever for 1930, it is noted that the same five conditions caused each more than 50,000 sick days during each of the last four years.

	Sick days			
	1931	1930	1929	1928
Accidental injuries and poisonings	153,368	154,340	145,532	150,307
Gonococcus infections	130,090	123,737	121,585	111,634
Syphilis	62,013	66,470	55,229	60,398
Tonsillitis	60,365	67,655	76,803	75,022
Catarrhal fever, acute	57,034	49,712	56,273	71,963

Administration of arsenicals in the treatment of syphilis.—There has been a gradual increase in the use of arsenicals since 1925, as shown in the accompanying table. The syphilis rate, while showing

a decline from 1927 to 1929, inclusive (24.57, 22.69, and 21.64 admissions per 1,000 men), advanced in 1930 and 1931 to 25.03 and 25.46, respectively.

	Number of injections			Number of injections	
	Arsphenamines	Tryparsamide		Arsphenamines	Tryparsamide
1925	47,717	1,160	1929	83,051	2,383
1926	63,163	1,232	1930	88,460	4,418
1927	74,478	2,054	1931	97,984	5,927
1928	80,000	2,551			

TRAINING OF MEMBERS OF THE HOSPITAL CORPS OF THE NAVY IN MEDICAL DEPARTMENT SPECIALTIES

Rear Admiral C. E. Riggs, Medical Corps, United States Navy, the Surgeon General of the Navy, has, during the past year, instituted in the medical service of the Navy a plan by which selected hospital corpsmen are trained as enlisted assistants in certain medical department specialties, and one in which the expenses of this training are reduced to the minimum in accordance with the present economic policy of the Navy Department.

The necessity of having available for the medical service a larger number of specially trained assistants in the hospital corps has long been apparent. With the advances in the medical profession and the coincident training in special fields of selected medical officers a speeding up of the program for providing enlisted assistants was mandatory.

Formerly the major portion of specialty instruction for enlisted men was given at the United States Naval Medical School, Washington, D. C., and at civilian institutions. This involved a considerable charge against Navy Department funds in that these men received subsistence allowance of approximately $1.95 per day for each man under instruction, in lieu of quarters and rations, and limited the number of men who could be assigned to courses of instruction.

As all major naval medical department activities and certain other naval units were considered excellently qualified and equipped for instruction of enlisted personnel and afford a variety of courses of instruction, it was decided to utilize them. Groups to receive special training were assigned to these activities, and, so as not to appear as a charge against the overhead of the institution, they were carried on the muster rolls as "under instruction." Thus, there was no necessity for subsistence in lieu of quarters and rations. Men so assigned are available for additional duty, and in times of emergency, epidemics, etc., their services are available.

A careful study of the requirements of the naval service resulted in the establishment of the following summarized data:

Hospital corpsmen trained in special fields

Specialty	Required	In service	Shortage	At sea	Foreign shore	In United States
Aviation medicine	75	67	8	28	10	29
Commissary	65	100		54	12	34
Medical aspects of deep-sea diving	6	0	6	0	0	0
Dentistry (general)	190	158	32	46	22	90
Dentistry (prosthetic)	36	33	3	13	3	17
Electrocardiography	30	29	1	10	3	16
Embalming	40	27	13	4	5	18
Laboratory	185	158	27	50	23	85
Medical field service	50	23	27	3	6	14
Physiological methods for diving and chemical-warfare research	6	2	4	0	0	2
Physical therapy	50	20	30	6	4	10
Property and accounting	338	300	38	238	12	50
Roentgenology	75	48	27	14	10	24

To illustrate the progress that has been made by this program, attention is invited to the data appearing on page 33 of the United States Naval Medical Bulletin of January, 1932. A comparison of those data with present developments reveals with what satisfaction the future may be faced.

Under the present system, training of Hospital Corps men is continuous and assures the Medical Department of an ample number of qualified assistants.

The attrition of this group is more or less constant in that enlisted men separate themselves from the service by transfer to the fleet reserve at the end of 16 and 20 years' service and by not reenlisting. Provision has been made whereby a Hospital Corps man holding a qualification certificate may, at his own request, after a period of 5-year service in a specialty receive training in another field or revert to the general service of the Hospital Corps. This enables the corpsmen to qualify in more than one subject, a factor of material benefit to him on separation from the service to enter the fleet reserve.

A bureau circular letter, dated July 5, 1932, invited the attention of all medical officers and Hospital Corps men to the advantages offered by these special courses of training. As a result of that letter a large number of requests for special courses has been received. Officers attached to the personnel office of the bureau were designated as a board of review to recommend men for these courses. In this selection, consideration is given to previous education, service records, adaptability to the service, statements from commanding officers, interest in work, quarterly efficiency reports, and duration of service on completion of the courses.

The following chart indicates the extent to which this special training is being conducted at various naval activities. It is planned to continue this special training until the service requirements have been met and then to keep under instruction a sufficient number of men to meet the attrition in the various specialties.

Hospital corpsmen under instruction

Station	Aviation medicine	Commissary	Medical aspects of deep-sea diving	Dentistry	Electrocardiograph	Embalmers	Laboratory	Medical field service	Physiological methods for diving and chemical warfare service	Physical therapy	Property and accounting	Bronchoscopy	Röntgenology	Number under training
Hospitals:														
Annapolis, Md		2									2			4
Canacao, P. I														
Charleston, S. C														
Chelsea, Mass				1			3			1			2	7
Great Lakes, Ill		5									1			6
League Island, Pa		1		1	1		4			2	1		3	13
Mare Island, Calif		5		1			3				4		1	14
Newport, R. I		4		1							3			8
New York, N. Y		1		3		6	6			7	6	2	3	34
Norfolk, Va		2		1	1		4				3		2	13
Parris Island, S. C														
Pensacola, Fla														
Portsmouth, N. H														
Puget Sound, Wash		1		3	1		1			1	2			9
San Diego, Calif		2		6	1	2	5			4	4		3	27
Washington, D. C										1			3	4
Naval air stations:														
Norfolk, Va	2			1										3
Lakehurst, N. J	7													7
Pensacola, Fla	3													3
San Diego, Calif														
Naval training stations:														
Norfolk, Va				4										4
Newport, R. I														
Great Lakes, Ill														
San Diego, Calif				1										1
Naval Academy, Annapolis, Md				3										3
Marine Barracks, Parris Island, S. C														
Marine Barracks, Quantico, Va				2										2
Marine Corps Base, San Diego, Calif								1						1
Naval Medical School, Washington, D. C							7							7
Harvard University, Boston, Mass									2					2
Deep diver's school, navy yard, Washington, D. C			1											1
Embalming school, west coast (receiving ship, San Francisco, Calif)						2								2
Carlisle Barracks								4						4
Total	12	23	1	28	4	10	33	5	2	16	26	2	17	179

Upon successful completion of courses in training, certificates are issued indicating the qualification as listed below and appropriate entries made on bureau records.

Technician—Aviation medicine.
Enlisted assistant to commissary officer.
Graduate—Course in medical aspects of deep-sea diving.
Technician—Dental (general).
Technician—Dental (prosthetic).
Technician—Electrocardiography.
Technician—Embalming.

Technician—Laboratory (clinical).
Technician—Laboratory (chemical).
Technician—Laboratory (biochemical).
Graduate—Noncommissioned officers' course, medical field service school.
Technician—Physical therapy.
Enlisted assistant to property and accounting officer.
Technician—Roentgenology.

Previous to the institution of the present system, instruction in embalming was given at an embalming school in one of our larger cities at a cost of $250 per man and a subsistence allowance of $1.95 per day for four to six weeks. Under the present arrangement 10 men are undergoing instruction in this specialty at no expense to the Government. This was accomplished by arrangements made by the district medical officers with reputable undertaking establishments in New York, San Francisco, and San Diego.

Formerly, men were assigned to various business colleges for training in accounting and finance. At present this instruction is given by warrant officers and men in the service who have completed these courses at civilian institutions.

Instruction leading to the qualification of "Technician—Aviation medicine," formerly given at the Naval Medical School, Washington, D. C., is now being conducted at the various aviation fields. Instruction in laboratory technique, formerly given at the Naval Medical School, is now being conducted also at major naval hospitals. Only advanced courses in this specialty are given at the school.

Gratifying results have been obtained by this policy of educating Hospital Corps men in medical department specialties. The commanding officers of the naval hospitals and the senior medical officers of naval activities have cooperated most enthusiastically and beneficial result to the service is assured.

The advantage of this system of training is shown by the fact that the long-existing deficit in the number of specially qualified Hospital Corps men is at last being overcome with speed and with satisfaction to all concerned.

SECOND CONFERENCE OF THE INTERNATIONAL BUREAU OF DOCUMENTATION OF MILITARY MEDICINE[1]

The second conference of the International Bureau of Documentation of Military Medicine was held at Liege, Belgium, from June 23 to 25, 1932. It constituted the continuation of an interesting assemblage as an institution established one year ago with serious and commendable purpose, being an outgrowth of the International Congress

[1] Report upon the second conference of the International Bureau of Documentation of Military Medicine by Capt. W. H. Bell, Medical Corps, United States Navy, and Commander W. S. Bainbridge, Medical Corps, United States Naval Reserve, delegates from the United States.

of Military Medicine and Pharmacy and organically identified with it in that the permanent committee of the latter is also the permanent committee of the other. Indeed, it is one of the activities of the Congress under the administration of the permanent committee, and one of the matters covered during this year's conference was the clarification of the relationship of the bureau of documentation to the congress and the permanent committee. Liege, Belgium, is its permanent headquarters, where offices have been provided by the municipal government and where its services are available at all times to those seeking information concerning publications, of medico-military import, or introduction and guidance in making contact by correspondence or in person with the nationals of different countries who have contributed to medico-military literature on various subjects or to advances in medico-military science. Meeting annually it serves also to prepare the way for the biennial meeting of the International Congress of Military Medicine and Pharmacy.

The United States delegates arrived in Liege in advance of the opening date of the conference and were in attendance at all sessions. Twenty-two nations were represented, including, Belgium, Chile, Colombia, France, Great Britain, Holland, Hungary, Italy, Lithuania, Luxemberg, Morocco, Mexico, Persia, Poland, Portugal, Rumania, Spain, Switzerland, Czechoslovakia, United States, Venezuela, and Yugoslavia. In addition, the League of the Societies of the Red Cross, the International Committee of the Red Cross, and the International Bureau of Labor were officially represented, and Germany, although not formally represented in the conference, was, nevertheless, present in the person of the consul general, as a representative of the ambassador of Belgium, for social participation only. Central Europe has been represented in the International Congress of Military Medicine and Pharmacy for years, but this is the first year that Germany has completed a gesture in that direction, and the fact is regarded as of agreeable significance by the International Office of Documentation of Military Medicine in its ambition that all nations capable of contributing to its purpose and high aims may seek to do so and come to be counted among those adhering.

Each of the nations mentioned was represented by one or more delegates, Belgium (the host), France, Holland, and Spain, in the order named, having the largest delegations. There was a total of 159 delegates, and it is eminently noteworthy that, as at the first conference of the International Office of Documentation of Military Medicine and the several sessions of the International Congress of Military Medicine and Pharmacy, a large proportion of the participants were of high military rank and distinction, bespeaking the importance with which the organizations and their functions are regarded. There were, among other representative personages, the

surgeon general or medical director general of the French Army, Polish Army, Italian Army, and Belgian Army, and there were also other general officers as well as diplomatic representatives.

The sessions were held in the assembly hall of the provincial palace and before the hour fixed for the conference to begin the delegates gathered in the façade in respect of the provincial governor.

The opening session of the conference was altogether impressive and also indicative of the great hope for increasing international understanding and good will as a special and actual by-product of its specific scientific and humanitarian purposes.

The first address was by the governor of the Province, who welcomed the delegates in behalf of the King and who in the course of his remarks stressed the international aspect of the gathering and read into it great and definite possibilities of influence toward the peace of the world.

The incoming medical director general of the Belgian Army, who was to preside at some of the sessions, was the next to speak and he sounded the note of the conference.

Then came in succession the representative of the bourgemaster of Liège, who spoke on the legal aspect of military medicine in its humanitarian service; the secretary general of conference, who recalled the past and projected the future of the organization and cautioned against any neglect of opportunities to further international comity through the intercourse offered by periodic gatherings incident to the activity of the International Office of Documentation of Military Medicine and the International Congress of Military Medicine and Pharmacy; and the Spanish delegate, member of the permanent committee, who spoke of the next meeting of the International Congress of Military Medicine and Pharmacy to be held in the capital city of his country next year. Finally, the member of the general staff for chemical warfare of the Belgian Army spoke at length and with great force on the seriousness of poison gas, as a weapon, to belligerent forces and civilian populations and expressed the conviction that until the leading nations of the world had placed it beyond the pale it was a problem for the military medical profession to solve. With this and a reception to the conference by the governor of the Province, immediately following, the morning programme of the first day was concluded.

Succeeding sessions were devoted to the presentation of the scientific papers and the agenda was quite faithfully observed. All subjects were presented in full except one and, without exception, were ably treated. The resultant total contribution to the advancement of our knowledge and increase of our resources in the medico-military field of practice is weighty.

Special entertainments during the conference were numerous and varied, and the city vied with individuals in extending hospitality to the delegates. The most brilliant affair was given the evening of June 24 in the Hotel de Ville by the bourgemaster, who was particularly cordial to the delegates from the United States.

Throughout the period of the conference both at the sessions and in social intercourse, whether at entertainments or otherwise, the spirit was delightfully fraternal and marked by a manifest desire to establish real and enduring friendships. These international gatherings of primary, medical, and humanitarian import seem especially fruitful of wider results in international amity than is wittingly projected, for the discussions and exchanges are always of that free and frank nature common to a profession whose individual ideas and discoveries or whatnot for the amelioration of suffering and the welfare of mankind belong to every member of it by ethical right. The frequent assembly of men and women with such high principles and generous disposition should be encouraged and facilitated and fostered in the interest of our highest hopes for civilization.

RADIATION IN BUERGER'S AND RAYNAUD'S DISEASE

Medical officers who have been called upon to treat cases of Buerger's and Raynaud's disease and have been discouraged by the results obtained from the various therapeutic measures so far recommended will be interested in the following communication recently received from Capt. Lucius W. Johnson, Medical Corps, United States Navy.

We have, at the naval hospital, San Diego, many cases of Buerger's and Raynaud's disease. The results of treatment have been most unsatisfactory, though we have employed all the methods that any of us could discover. Recently I read in a French medical periodical of the treatment by radiation of the ganglia. The writer stated that relief of pain was immediate and prolonged and that several limbs had been saved from amputation. I have since been unable to locate this reference or to find any mention of it in the literature.

I submitted the idea to Lieut. Commander O. B. Spalding, our Röntgenologist, and he was glad to experiment with it. The results have been most gratifying in three cases of Buerger's and one case of Raynaud's disease. One case of Buerger's disease showed only temporary slight improvement. The treatment is followed by prolonged vasodilation and relief of pain. Ulcerations, in two cases, are beginning to heal. The patients are most enthusiastic over it because of the complete relief of pain. They had previously had all

kinds of treatment in many clinics and one of them had ganglionectomy with little relief.

Doctor Spalding gives one-quarter of an erythema dose on alternate days for three doses and continues on alternate days with a gradually reducing dose.

We believe, though the evidence is by no means complete, that the radiation is of greatest value in the same type of case that is helped by injection of the ganglia and ganglionectomy.

Editor's note.—K. H. Beall and J. Jagoda writing in the Texas State Journal of Medicine, vol. 26, p. 279, August, 1930, also report excellent results from irradiation of the sympathetic ganglia in Buerger's disease and Raynaud's disease. They believe that a more or less permanent increase in the blood supply of an extremity can be caused by irradiation of the corresponding sympathetic ganglia.

ATEBRIN IN TREATMENT OF MALARIA

The annual report of the medical department of the United Fruit Co. for 1931 contains three articles on the treatment of malaria with atebrin.

These articles report the results obtained by physicians of the company who were provided with sufficient quantities of atebrin to enable them to conduct experiments on series of cases. The drug, a synthetic product prepared by a German firm, was supplied in compressed tablets, each tablet containing 0.1 gram of atebrin.

A total of 320 cases were treated—278 aestivo-autumnal, 35 tertian, 1 quartan, and 6 mixed infections. As a rule, the dosage prescribed by the manufacturer—0.3 gram daily, divided in three doses, for three consecutive days—was followed; however, in many instances the drug was given in larger daily doses, or greater length of time, or both.

The results of treatment, according to the reports, were uniformly gratifying, and the following conclusions reached by Doctors Phelps and Jantzen of the Truxillo division, agree in general with those of the other investigators.

1. Atebrin, in daily doses of 0.3 gram for six days, is an efficient therapeutic agent in clearing the blood of the vegetative forms of benign tertian or malignant tertian (E. A.) malaria. It successfully and promptly relieves the symptoms of malaria. It may be used in smaller doses, but the treatment is more prolonged.

2. The toxicity of atebrin is low, with a wide margin of safety between the effective therapeutic doses and the toxic dose.

3. Treatment with atebrin has decided advantages over that with quinine. There is no tinnitus; digestive organs are not upset; and the tablets are small and very soluble, thus giving assurance that the medicine can be taken and that prompt absorption will follow.

4. Atebrin seems to have no destructive effect on the gametes in the blood, and, for this reason, it should be supplemented with plasmochin.

5. Photosensibility does not occur; but a yellow discoloration of the skin may result, and especially if the treatment is extended over six days.

6. Hemoglobinuria does not seem to be provoked by atebrin, nor does there seem to be any contraindication to giving atebrin to cases which have developed hemoglobinuria.

7. It is our impression that relapses do not occur over a period of months following the completion of atebrin treatment.

FURTHER STUDIES ON THE TRANSMISSION OF ENDEMIC TYPHUS FEVER

Several months ago the announcement was made by the United States Public Health Service that endemic typhus fever, which has been recognized for several years in the United States, had been shown to be transmitted by fleas.

Additional studies indicate that the rat flea is the agent that transmits this condition. This work has been proven by laboratory experiments and by field studies, all of which have been conducted by the Public Health Service. There seems to be ample evidence that endemic typhus fever is spread from rat to rat by the rat flea, and from rat to man by the same agency.

IS THE "APPALLING INCREASE" IN HEART DISEASE REAL?

Under this title C. F. Bolduan and N. S. Bolduan present in the Journal of Preventive Medicine, July, 1932, the results of a study of the mortality statistics of New York City covering the past 30 years.

Their summary and conclusions follow:

A study of New York City's mortality statistics has been made to determine the truth of the statements that there has been an "appalling increase in the death rate of heart disease."

The character of the basic data (death certificates) is analyzed and sources of errors to be guarded against are pointed out.

The difficulty of classifying deaths in which there is more than one cause of death is discussed, and the practice of statistical offices is described.

The pathological changes and the etiological factors in heart disease are shown not to be limited to the heart, but to include also the arteries and kidneys.

The registered increase in heart disease is admitted and shown to be accompanied in New York City by a registered decrease in apoplexy, Bright's disease, and deaths charged to senility.

Statistics are presented which show that in New York City, for the population as a whole, the death rate from cardio-arterio-renal disease is no higher now than it was 30 years ago.

Similar statistics are presented for the four important age groups, and only in the group aged 65 years and over do these show any increase in the death rate from cardio-arterio-renal disease.

Conclusions

1. In New York City, for the population as a whole, there is no evidence of any increase in the real death rate from heart disease.
2. The registered rise in the death rate from heart disease is largely, if not wholly, fictitious.
3. Statistics based on the registered deaths from heart disease alone are fallacious. Deaths registered under apoplexy, arterial disease, kidney disease, and senility must be taken into account.
4. The great prevalence of cardio-arterio-renal disease presents a very important health problem. Its solution demands accurate statistics.
5. In the United States registration area as well as in New York City the specific death rates even in the higher age group have declined since the beginning of the century, a fact which it is difficult to reconcile with any considerable increase in the mortality of heart disease.
6. A detailed statistical study of the course of the death rate from cardio-arterio-renal disease in the United States registration area should be made, due consideration being given in such a study to the various sources of error discussed in this paper.

POSTVACCINATION ENCEPHALITIS, WITH SPECIAL REFERENCE TO PREVENTION

Writing in the July 22, 1932, number of the United States Public Health Service weekly report, Dr. Charles Armstrong presents the results of a study of postvaccination encephalitis, with special reference to prevention.

As a designation for this complication, the author states that "postvaccination encephalitis is deemed preferable to postvaccinal or postvaccinial encephalitis, because the complication follows the vaccination but usually appears at the height of, rather than after, the vaccinia. The term postvaccination is, moreover, noncommital as to the vaccinal or nonvaccinal nature of the ailment."

The summary of the results of the investigation follows:

The only practicable means so far suggested for preventing the encephalitis occasionally noted following smallpox vaccination have to do with the vaccination procedure.

A suitable vaccination technique is defined as one employing a small superficial insertion, never over one-eighth inch in greatest diameter and which employs no routine dressing.

Infancy is the best time for performing primary vaccinations in so far as the prevention of postvaccination encephalits is concerned.

Evidence is presented which suggests that inoculation with diphtheria toxoid tends to render mice somewhat more resistant to vaccine virus subsequently administered intracerebrally.

It is suggested that primary vaccinations, especially after the first year of life, be deferred until contemplated immunization against diphtheria or other diseases by means of inaminate antigens has been accomplished. (It is suggested that the first dose of diphtheria toxoid be given preferably at 6 months of age and the second dose 1 month later; vaccination against smallpox to follow the second inoculation in from 3 to 4 weeks. The same procedure is suggested for older children also.)

The hope is expressed that a recent preliminary exercise or mobilization of the immunity or defense forces may lead to a more efficient anti-vaccine-virus response, with the result that the ensuing reaction may tend to simulate primary infant or secondary vaccinations in their comparative mildness and freedom from postvaccination encephalitis. The suggestion is made that possibly the high percentage of poliomyelitis cases recorded among diphtheria-susceptible children in New York in 1916 may be due in part to an increased resistance to poliomyelitis among children immune to diphtheria.

THE PROGRESS OF THE ELEVENTH REVISION OF THE UNITED STATES PHARMACOPŒIA

The subcommittee on scope, of the committee of revision of the United States Pharmacopœia XI has announced its first decisions on what is commonly spoken of as "Admissions and deletions." The members of the subcommittee have diligently pursued their part in the work of revision and their discussions and conclusions cover almost 400 pages of subcommittee bulletins. In addition to this, the subcommittee has held three personal conferences when titles were discussed and many decisions reached.

The following lists are announced for the information of members of the medical and pharmaceutical professions and the general chairman invites a free discussion of these first decisions. Comments received by him will be issued in the official circulars under the author's name and referred to the subcommittee for their consideration. General statements are of little help to the committee. Criticisms or comments should deal with specific items and offer, if possible, facts to support the opinion expressed.

Deletions proposed.—The following United States Pharmacopœia X titles, 108 in number, have not been admitted to the United States Pharmacopœia XI:

Aconitina.
Antitoxinum tetanicum crudum.
Benzaldehydum.
Buchu: Fluidextractum buchu.
Calcii glycerophosphas.
Calumba: Tinctura calumbæ.
Cambogia.
Cimicifuga: Fluidextractum cimicifugæ.
Cinchonidinæ sulphas.
Colchici cormus:
    Extractum colchici (cormi).
    Fluidextractum colchici (seminis).

Colocyntnis:
Extractum colocynthidis.
Extractum colocynthidis compositum.
Cotarninae chloridum.
Cubeba.
Elaterinum.
Emplastrum capsici.
Emplastrum plumbi oleatis.
Eucalyptus: Fluidextractum eucalypti.
Ferri carbonas saccharatus.
Ferri chloridum.
Ferri phosphas solubilis.
Ferri sulphas exsiccatus.
Fluidextractum belladonnæ foliorum.
Fluidextractum cinchonæ.
Fluidextractum hyoscyami.
Fluidextractum rhei.
Fluidextractum scillæ.
Gambir: Tinctura gambir composita.
Glyceritum phenolis.
Granatum: Fluidextractum granati.
Guaiacolis carbonas.
Hydrargyri iodidum rubrum.
Hydrastis: Fluidextractum hydrastis.
Hyoscyaminæ hydrobromidum.
Infusum digitalis.
Ipomœa: Resina ipomoeæ.
Jalap: Resina jalapæ.
Krameria: Tinctura krameriæ.
Linimentum calcis.
Liquor arsenii et hydrargyri iodidi.
Liquor ferri et ammonii acetatis.
Liquor plumbi subacetatis.
Liquor potassii arsenitis.
Liquor potassii citratis.
Liquor potassii hydroxidi.
Liquor sodæ chlorinatæ.
Liquor sodii hydroxidi.
Liquor zinci chloridi.
Lobelia: Tinctura lobeliæ.
Manna.
Mistura glycyrrhizæ composita.
Morphinæ hydrochloridum.
Oleoresina capsici.
Oleum cajuputi.
Oleum cari.
Oleum tiglii.
Paraformaldehydum.
Pepo.
Phosphorus.
Pilocarpinæ hydrochloridum.
Pilulæ asafœtidæ.
Pilulæ hydrargyri chloridi mitis compositæ.

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Pilulæ phosphori.
Plumbi monoxidum.
Pulvis jalapæ compositus.
Pulvis rhei compositus.
Quassia.
Quininæ hydrobromidum.
Quininæ hydrochloridum.
Quininæ tannas.
Rhus glabra: Fluidextractum rhus glabra.
Salicinum.
Senega:
    Fluidextractum senegæ.
    Syrupus senegæ.
    Syrupus scillæ compositus.
Strontii salicylas.
Strophanthus: Tinctura strophanthi.
Sulphonmethanum.
Syrupus rhei.
Tinctura asafœtidæ.
Tinctura cardamomi.
Tinctura cinchonæ.
Tinctura rhei.
Tinctura valerianæ amoniata.
Trochisci acidi tannici.
Trochisci ammonii chloridi.
Ulmus.
Unguentum iodoformi.
Unguentum plumbi oleatis.
Uva ursi: Fluidextractum uvæ ursi.

New admissions.—The following new admissions have also been announced:

Acriflavine.
Antimeningococcus serum.
Antipneumococcus serum, type 1.
Antiseptic iodine solution (exact formula to be determined).
Carbon dioxide.
Chiniofon (sodium iodoxyquinoline sulphonate).
Digitalis solution for injection.
Diphtheria toxin for Schick test.
Diphtheria toxoid.
Emulsion of mineral oil.
Ephedrine.
Ephedrine sulphate.
Ethylene for anesthesia.
Fluorescein.
Histamine acid phosphate, solution of histamine acid phosphate.
Iron arsenate (standardize for ampul manufacture).
Iron arsenite (standardize for ampul manufacture).
Iron citrate (green) (standardize for ampul manufacture).
Liver extract.
Phenobarbital soluble.
Rabies vaccine.

Tetraiodo-phenolphthalein sodium.
Tuberculin, old.
Typhoid vaccine.

A few additional titles have been tentatively admitted, subject to the adjustment of possible patent or trade-mark complications. In this list are insulin and viosterol and other similarly involved substances. It is hoped that arrangements can be made for the admission of most of these.

Comments should be sent to the general chairman of the committee of revision of the pharmacopœia of the United States of America, E. Fullerton Cook, Forty-third Street and Woodland Avenue, Philadelphia, Pa.

PARROT FEVER WARNING ISSUED BY PUBLIC HEALTH SERVICE

The United States Public Health Service advises all persons to avoid contact with recently shipped or acquired birds of the parrot family. Several cases of psittacosis, or parrot fever, are being reported in various parts of the United States. Reports of five cases and one fatality have recently been received from Minneapolis, Minn. Another case has been reported from Boise, Idaho. There have been 12 cases of parrot fever, with 6 deaths, reported in California between December 1, 1931, and February 10, 1932.

Upon the recommendation of the Public Health Service, the Secretary of the Treasury has recently issued an order amending the interstate quarantine regulations so as to limit the interstate transportation of birds of the parrot family by common carriers to those certified by the proper health authority of the State as coming from aviaries free from infection.

A medical officer of the Public Health Service at the invitation of the California State Department of Public Health, within the recent past made a careful study of the situation in California with reference to parrot-fever infection and the breeding of birds of the parrot family in that State. Conclusive evidence was thus obtained which indicates that psittacosis, or parrot fever infection is present in some of the breeding aviaries of southern California. Parrots and parrakeets from this source have probably been one of the important means of spreading the disease to other States. The cases occurring in Minneapolis and Boise were traced to California birds as well as previous cases this year reported from New York City and Oregon.

An outbreak of psittacosis or parrot fever occurred in the United States during the winter of 1929–30. One hundred and sixty-three cases were reported at that time, with 33 deaths. Practically all of these cases were traced to association with recently acquired parrots and parrakeets.

THE DIVISION OF PREVENTIVE MEDICINE

O. J. MINK, Captain, Medical Corps, United States Navy, in charge

DOPE POISONING AS A POTENTIAL HAZARD IN SPRAY COATING AIRPLANE WINGS

By CLIFTON A. YOUNG, Lieutenant, Medical Corps, United States Navy, in collaboration with A. C. GELATTE, Chief Pharmacist's Mate, United States Navy

Since the stimulus of chemical research was brought about by the World War, and incidentally since the coming of the airplane into prominence as an important item industrially, numerous chemicals of varied physical and chemical properties have made their appearance in the form of solvents, plasticizers, etc., for use in the manufacture of spray lacquers. It is apparent that many of these organic compounds may be met with in the process of doping airplane wings. Much has been written concerning the toxicity of benzene (benzol) as a health hazard in the lacquer and other industries, covering both acute and chronic poisoning by this substance, and the "toxic jaundice" producing power of tetrachlorethane dopes seems to be well known. The former, while still in use to some extent in the Navy and elsewhere, appears to have been largely excluded from the better grades of American lacquers, and no formula was found for a dope now in use that contained the latter. From this fact it would appear that tetrachlorethane has been eliminated as a present-day constituent in airplane dopes. However, there are many chemicals still in use as lacquer ingredients that may be considered dangerous substances with respect to health hazards, especially when the lacquers are applied by the spray-gun method.

The men employed in spray coating the wings of airplanes in the dope shop of a naval air station had been drinking large quantities of milk as a prophylactic measure against possible poisoning by the toxic ingredients that might be contained in the spray lacquers. The milk was being furnished by the station's welfare service, the expense of which was becoming more and more a drain on its funds until the amount of the monthly bill for milk used for this purpose by three or four men mounted to approximately $40. The effectiveness of milk as a preventive became a question that brought about a search through available literature for information on this subject together with what could be learned regarding the toxicity of the constituents of airplane dopes.

The first problem was to determine the ingredients of the airplane dopes. This could not be readily accomplished, because different contractors did not furnish dopes of the same composition and the dopes received from the same contractor at different times were not necessarily always the same. It was learned, however, that the dopes being furnished the station consisted in general of various proportions of the compounds summed up as follows:

Film-base compound.—Cellulose nitrate; cellulose acetate.
Flexilizers.—Triphenyl phosphate; triacetin; tricresyl phosphate; castor oil; camphor.
Solvents.—Acetone; methyl acetate; ethyl acetate; methyl-ethyl ketone (for cellulose acetate); butyl acetate; amyl acetate (for cellulose nitrate).
Diluents.—Ethyl alcohol; toluol; xylol; mineral spirits; butyl alcohol.
Dryer.—Ethyl lactate; diacetone alcohol; cellosolve; benzol alcohol; methyl cellosolve; cellosolve acetate.

While most of the compounds in the above groups were found in the literature, nothing applicable to the health hazards of spray-coating could be found concerning triphenyl phosphate, triacetin, methyl acetate, methyl-ethyl ketone, and ethyl lactate. Furthermore, no information concerning mineral spirits was located under that name, though data found on certain petroleum distillates may apply to this diluent. In the endeavor to locate data on these substances the search finally resolved itself into the subject of lacquer solvents in general, because it appeared that most of the solvents used in spraying lacquers might also be used in airplane dopes.

No standard formula for Navy airplane dopes seems to have been prescribed, the object of the specifications being to obtain a lacquer that would produce a properly finished coating. However, it is stated in the specifications for cellulose nitrate and acetate dopes that the dopes shall contain no chlorinated compounds or other toxic or saponifiable chlorine derivatives, and that the vapors shall not cause serious discomfort or injury to the workmen engaged in the application of the dope. The specifications for pigmented and semi-pigmented nitrate dopes prohibit the use of benzene (benzol) in addition to the above. Thus it may be possible for any of the numerous solvents, except "chlorinated compounds or other toxic or saponifiable chlorine derivatives," and in most cases benzene, to become an ingredient in Navy dopes, unless it is known that the vapors will cause "serious discomfort or injury" to the doper. With the elimination of the substances prohibited by specifications it will still have to be conceded that a good lacquer must necessarily contain substances of a more or less poisonous nature, that its constituents are subject to frequent changes and that precautions are necessary to prevent possible dope poisoning in the process of spray coating with it.

In the matter of controlling health hazards various regulations for spray painting, committee reports, special articles, etc., stress the point that carefully regulated ventilation is paramount, and recommend air masks, helmets, etc., as the only adequate protection when the exposure is high or continuous. Articles on this particular phase of the subject are quite voluminous and the essential points appear to be well covered in the conclusions of the National Research Council of Canada quoted below. This report also contains an extensive bibliography and much valuable material, collected from many sources, on the physiological effects of the more common constituents of spray paints, varnishes, and lacquers, and the means of controlling health hazards.

After a careful study of all the information found in the accompanying report, the committee is of the opinion that there are certain definite health hazards connected with spray painting, and the committee offers the following conclusions and recommendations for the consideration of the constituted public authorities who are responsible for dealing with such matters.

1. The more toxic or pathogenic ingredients in the material to be sprayed should be reduced to a concentration below that which constitutes a health hazard. This will involve especially lead (which present knowledge would indicate should not exceed in concentration 1 per cent of the material used for spraying), benzol, turpentine, and silica, and certain alcohols (methyl, butyl, allyl, and amyl), benzine, gasoline, toluol (toluene), xylol (xylene), tetralin, and tetrachlorethane. When a satisfactory painted surface can be produced otherwise a number of these ingredients should be eliminated altogether from the material used for spraying. All paints containing lead, benzol, turpentine, and free silica for use in spray painting should be labeled in such manner as clearly to indicate the percentage of each such substance in the container. Whether other ingredients now used or other compounds which may in the future be so used should be included in the foregoing list can only be determined after careful investigations have been made as to the possibility of their constituting more or less serious health hazards in spray painting.

2. The spray booth and its equipment should be such as to provide the maximum safety possible for the spray operator. The exhaust fan should provide a sufficient current of air, varying according to the size of the booth, but adequate to exhaust the fumes. To prevent the exit of the fumes or air charged with the ingredients of the sprayed material other than through or by the exhaust fan the air entering the room in which the booth is located should not exceed the supply of air removed from the booth by the exhaust. The face of the booth should be provided with doors capable of being adjusted to reduce the open face of the booth, according to the size of the object to be sprayed.

Where spray painting is done within buildings, it should be carried on only in a part of the building set aside exclusively for such work. Objects freshly sprayed should be immediately removed for drying to special apartments not used for other purposes, and proper equipment for exhausting the fumes from such apartments should be provided.

3. The air pressure used in the spray machine should be as low as will provide a satisfactory painted surface. The nozzle of the spray machine should be held at least 1 foot distant from the object sprayed, in order to avoid unnecessary rebound (beat back) of the spray.

4. The operator should, and, when he is engaged in interior spray painting, must, wear a mask into which uncontaminated air under pressure is supplied.

5. No one should be engaged as an operator who is under 20 years of age, and before beginning employment as such should undergo a medical examination to determine whether he is in proper condition to serve as an operator. Thereafter, where possible, every three months he should be examined medically to determine whether he has been affected in any way by the material used in spraying, and if found affected should only continue as operator on medical advice and under medical supervision. No woman should be employed in spray painting during pregnancy.

6. Provision should be made requiring spray operators personally to observe regulations intended to minimize the health hazard. (2, p. 36.)

As stated previously, the primary object of this search was to determine the effectiveness of milk drinking as a preventive measure against possible dope poisoning, but it appears from the results of the search that the establishing of properly regulated ventilation and the use of other safety devices are the important items of prevention when it becomes necessary to spray paint with toxic substances. However, in the matter of milk drinking as a preventive measure, Alice Hamilton (1, pp. 4–5) has touched on the subject under the heading of Food as follows:

It is, of course, a fact well known to pharmacologists that drugs are absorbed more quickly by the fasting stomach than when administered after a meal, but although almost anyone knows this to be true with regard to medicines, few people realize that it is equally true with regard to industrial poisons. Experience in the lead trades taught the English years ago that one of the best preventatives of lead poisoning was the presence of food in the stomach, and in English lead works it has long been customary to supply the workmen, free of cost, a glass of milk or a cup of cocoa the first thing in the morning, and in their more recent industry of anilin dye manufacture the English follow the same rule. The Germans' experience during the war, when the food blockade demonstrated on an unprecedented scale the effects of lack of food, has proved conclusively that lowering the body nutrition results in a marked lowering of the resistance to poisons * * *.

This question of the influence of proper and sufficient food in warding off industrial poisoning has received less attention in the United States than one would expect. In all my experience I have known of but one white-lead works and one anilin dye works which give milk to the workmen, and in the case of the dye factory it is given only to those employed in the most dangerous department, the one in which dinitrobenzene is handled. It is not usual, even in the poisonous trades, to find provisions for a wholesome hot meal at noon, and the indigestible cold lunch is as customary in American factories where poisons are used as is the lunch room with hot food and hot tea or coffee in the German and the British factories * * *.

The remainder of the reference deals mostly with milk versus lead, in which case it appears that milk is effective. While lead is considered one of the principal dangers in spray painting, no reference was found including it as an ingredient in the cellulose-derivative lacquers as used in airplane dopes. Therefore, eliminating lead, it

could not be determined whether or not milk will play a more important part in the prevention of dope poisoning than to prepare the individual against a possible increase in the action of the poison, due to lack of resistance brought about by the fasting stomach, or to aid the body nutrition and thus assist in warding off industrial poisons in general.

Data on Toxicity of Compounds Used in Spray Lacquers

Data on the toxicity of many of the compounds used in spray lacquers are lacking. The report of the National Research Council of Canada (2) lists 15 substances about which no information applicable to spray painting was found in the literature. Their review covered literature published up to February 1, 1929. However, interesting information concerning the toxicity of many substances that might be used as ingredients in airplane dopes has been gathered from available literature and is presented below.

Toxic constituents.—Toxicity due to substances in spray painting involves an effect from the introduction of foreign substances into the body, resulting in derangement, temporary or permanent, of normal function. Sometimes the toxicity will be of negligible effect and sometimes it will be severe, involving impairment of body function with consequent ill health.

The spray-painting machine, which ejects under an air pressure of 30 to 100 pounds, pigments and solvents used partly because they evaporate rapidly, subjects the worker to an atmosphere containing these substances as fine particles, vapor, or droplets. They enter the system, mainly by inhalation, to a limited extent by ingestion, and in rare instances through the surface of the skin.

From this standpoint, the results of investigations to determine the toxicity of many of the substances are hardly applicable to spray painting, since conclusions regarding the effect of the inhalation of these substances can not be drawn from experiments using ingestion, subcutaneous, or intravenous injection methods (2, p. 11).

Cellulose nitrate and cellulose acetate.—Celluloseacetate and nitrate are both quite free from poisonous properties, but their solvents are not. A study of the risks involved in airplane manufacture resolves itself into a study of dope solvents (3).

Benzene (Benzol)[1].—The final report of the committee of the National Safety Council on benzene contains an excellent review of the literature with report of experiments on white rats as to the effects of benzene and its homologues following intraperitoneal and subcutaneous injection, and upon inhalation.

The National Safety Council spray-painting report states that benzene is being discontinued by practically every manufacturer because of its recognized health hazards.

In 1922, Dr. Alice Hamilton published an article entitled "The Growing Menace of Benzene (benzol) Poisoning in American Industry." In 1928, the same author entitles an article, "The Lessening Menace of Benzene Poisoning in American Industry."

[1] A commercial term applied to various mixtures of aromatic hydrocarbons obtained from coal tar. The terms benzene and benzol are often used synonymously.

In the Pennsylvania survey it appeared that, "the benzol content of lacquers and similar finishes is highly variable and uncertain, and even when not originally present, benzene is frequently added in the form of cheap lacquer thinners."

Collection in air may be made by an absorption tube improved in the course of the Pennsylvania survey. Estimation of benzene in air is made by Elliott and Dalton's modification of Pfeiffer's method. This method was tested to show that the presence of toluene did not interfere with the benzene determinations. In the National Safety Council survey, titanous chloride was substituted for stannous chloride for the reduction of nitro groups with improved practical results. Benzene in air can be determined by this method in concentrations of 10 parts per million. Tests showed the method to be sufficiently accurate at concentrations to be expected in practice.

In the Pennsylvania survey, 91 samples of air averaged 100 p. p. m. benzol in air including one with 2,000 p. p. m. and 28 negative samples. In the National Safety Council survey, 85 samples averaged 918 p. p. m., including one with 6,500 p. p. m. and 5 negative samples. In tests made in spray booths there was an inverse correlation between air velocity and benzene content of air.

Rambousek states that 0.015 cubic centimeter per liter may produce confusion in one-half hour. Legge states that 550 p. p. m. is likely to be associated with some definite clinical poisoning.

Injury to the blood-forming organs appears with concentrations of 100 p. p. m. of benzene in air. A concentration of 460 p. p. m. destroys the white cells of the blood, while even 2,440 p. p. m. is not fatal in adult white rats. Chronic poisoning was reported from New Jersey with exposure to 100 p. p. m.

In regard to physical effects, acute poisoning is characterized by violent irritation of the nerve centers and consequent asphyxiation and fails to produce the effect on the cell content of the blood characteristic of chronic poisoning. Evidence of benzene absorption was found in many sprayers of lacquer, substitute shellac, and stain. It occurred to a less degree in paint sprayers and a few varnish sprayers, with no evidence of absorption in shellac sprayers. The number of symptoms increased with the length of service.

The symptoms of chronic poisoning include headache, dizziness, weakness, loss of appetite, pallor due to true anemia, shortness of breath, and a disturbed blood picture.

The Pennsylvania and National Safety Council surveys took 9,000 white cells per cubic millimeter of blood as the normal white-cell count for the industrial group and agreed with the benzol committee that a white-cell count below 5,625 per cubic millimeter may be taken as a fair index of this disease. The importance of the total white-cell count, the polymorphonuclear count, and the lymphocyte count is stressed. The lymphocyte count rises at first and eventually drops, but not so rapidly as the other white-cell counts giving a relative lymphocytosis. With heavy exposure, this initial stimulation is absent.

In the plants with high exposure to benzene there were more workers showing low red-cell counts and more workers showing low white-cell counts, i. e., below 5,625 per cubic millimeter.

In the National Safety Council survey in 127 lacquer-sprayer examinations over 5 per cent of the white-cell counts were indicative of benzene poisoning and over 39 per cent gave a disturbed blood picture.

In the body 15 to 30 per cent of benzene absorbed is oxidized to phenol and dihydroxybenzenes which combine with sulphuric acid and glycuronic acid to

be eliminated through the urine. A small amount of benzene is also broken down into muconic acid.

According to Gadaskin, benzene poisoning is the composite effect of the toxicity of benzene and toluene. In dogs benzene gives an immediate rise in sulphur output and a rise in the sulphur-nitrogen ratio with a compensatory fall later, suggesting the formation of mercapturic acid. The administration of toluene gives no indication of the formation of mercapturic acid.

Subcutaneous injection of benzene gives a sharp early rise in creatine, and to a less extent in total nitrogen, far in excess of that found in rabbits after starvation. Benzene, chloroform, toluene, turpentine, and xylene in vitro exhibit hæmolytic power in the order named.

Experiments on poisoning with rabbits correspond to the effects produced in cases of poisoning in workers.

Extracts of liver and bone marrow from animals poisoned by benzene are more strongly leukolytic than normal. Later this leukolytic power disappears and injury to bone marrow is more marked. Some animals with repeated doses of benzene became hypersensitive and were poisoned by amounts which they tolerated before (2, p. 13).

Absolute protection can not be guaranteed where benzol lacquers are used, so the recommendation is made that no lacquers be used for spraying which contain over 0.5 per cent of benzol. Where materials containing benzol have to be used a sprayer should have an efficient air mask or helmet. It would be safest to discontinue the spraying of any paints or lacquer coats containing over 1 per cent of lead. The final conclusion was that manufacturers of paints, lacquers, shellacs, varnishes, and vitreous enamels to be used in spray coating, should as far as possible eliminate benzol, lead and free silica, and containers should be labelled accordingly (2, p. 29).

Turpentine.—Gasoline, benzene, white spirits, and tetraline are used as substitutes for turpentine.

H. A. Gardner conducted inhalation experiments with six grades of turpentine. He states that, "paints and varnishes thinned with mineral spirits, gum spirits of turpentine or highly refined steam-distilled wood turpentine are nontoxic from the standpoint of vapors of the solvent used." This generalization is based on the assumption that good ventilation exists. "Refined steam-distilled turpentine showed slight organic changes, crude steam-distilled wood turpentine and destructively distilled wood turpentine gave toxic effects possibly due to the presence of aldehyde."

Spirits of turpentine can certainly exert a toxic action on organs. It is capable of causing complications in the kidneys and blood vessels.

Armstrong and Klein, after experimental work, conclude that some of the physical effects appearing among painters, usually attributed to lead, are in fact due to turpentine. In brush painting, Armstrong and Klein found 3.2 milligrams turpentine per liter of air in the vicinty of the painter's mouth. Lehmann has shown by animal experiments that 3 or 4 milligrams vapor per liter of air produce severe symptoms.

On the other hand, Agasse-Lafont maintains that, while turpentine may exert an effect on the cardio-renal system, the effects from painting usually attributed to lead are really due to lead.

The English registrar-general's figures to the commission of enquiry into industrial painting give no indicaton that turpentine is the cause of the excessive nephritis among painters. In the previous 10 years, there had been no death certificate with turpentine poisoning given as the cause of death.

Smyth and Smyth found that 715 p.p.m. steam-distilled turpentine for 45 to 58 exposures produced only slight changes in the liver and scattered tubular degeneration in kidneys in guinea pigs, corresponding closely to findings with similar exposure to gum spirts of turpentine. According to Albaugh, 0.003 gram per liter produced local symptoms and 0.006 gram per liter produced poisoning in 1 to 4 hours.

It is a local irritant, and, absorbed through the lungs, has an exciting effect on the central nervous system. With large doses, the symptoms are dyspnoea, palpitation, giddiness, stupor, convulsions, pain in the chest, and inflammation of the kidneys, which last also arises from the chronic action of turpentine.

This summary indicates that a satisfactory decision regarding the physiological action of turpentine has yet to be obtained. Turpentines vary exceedingly in composition, and some of the effects may be due to special constituents (2, p. 14).

Turpentine, according to Alice Hamilton, causes poisoning chiefly, if not wholly, through the inhalation of vapors. Prosser White quotes McCord, according to whom, with the use of gum spirits of turpentine as a solvent for paint, there were in 12 years 3 cases of rash yearly. He found that steam-distilled wood turpentine is far more irritating than any other type. The destructively distilled wood turpentine is less irritating than any other type until thoroughly oxidized. Prosser White says that tetralin and decalin also are obnoxious to the skin (2, p. 42).

Methyl alcohol.—Boiling point 65° C. A progress report is made of an experimental study of the toxicity of methyl alcohol by inhalation and skin absorption.

Two hundred and sixty-five animals have been utilized to date, including 31 monkeys, 58 rabbits, and 176 rats.

The materials for test purposes have included crude, 95 per cent, and highly purified natural methyl alcohols, and also synthetic methyl alcohols derived from three sources of manufacture.

In skin-absorption experiments, methyl alcohol was applied under conditions that precluded concurrent inhalation. Methyl alcohol applied to the skin has invariably led to damage consistently like that arising from the oral intake of this substance. This evidence of absorbability of methyl alcohol has been noted in optic atrophy observed clinically by ophthalmologists, and after death as observed in lipoidal degeneration. The regular recovery of methyl alcohol on the distillation of all organs derived from skin-treated animals, and the absence of similar findings in control animals, constitutes inescapable proof of absorbability.

The threshold of danger following skin absorption of methyl alcohol is near 0.5 cubic centimeters per kilogram of animal weight, applied four times daily. This quantity produces illness in monkeys within 24 hours, during which time four applications of the specified amount were made, with eventual death. One and three-tenths cubic centimeters per kilogram of weight will produce death within 48 hours when such applications are made at the rate of four per day.

If these results obtained from monkeys may be applied to man, approximately 1 ounce (31 cubic centimeters) of methyl alcohol repeatedly in contact with the human body, under conditions favorable to retention and evaporation, constitutes a threat to well-being.

Inhalation experiments have been carried out with the three kinds of animals mentioned, in concentrations ranging from 1,000 to 40,000 p. p. m. of methyl-alcohol vapor, in gassing chambers under controlled conditions providing suitable changes of air. All of these mentioned concentrations have produced death

among exposed animals. One thousand p. p. m. of air have killed some but not all animals, the shortest exposure time being 41 hours at the rate of 18 hours per day. Rabbits—and particularly black rabbits—are much less susceptible to the action of methyl alcohol than other animals. Marked variations in individual susceptibility have been observed.

The duration of exposure in hours per day constitutes a definite factor in the experimental toxicity from methyl alcohol. An exposure of one hour daily to 40,000 parts of methyl alcohol vapor per 1,000,000 of air causes scant evidence of immediate impairment among animals but eventually kills. Four hours of such exposure promptly kills all animals. A few animals will survive for weeks when exposed for seven hours daily to 10,000 p. p. m. of air, but quickly succumb to the same exposure over 18 hours per day.

Animals subjected to the inhalation of methyl alcohol have regularly yielded methyl alcohol on distillation of organs, blood, urine, muscle tissue, etc.

The varied tests made upon the distillates from organ tissues appear to establish that methyl alcohol and not formaldehyde is the principal recoverable foreign substance. Very rarely have even traces of formaldehyde been detected. Brain, lungs, heart muscle, skeletal muscle, liver, spleen, pancreas, kidneys, blood, and urine have consistently yielded methyl alcohol after exposure either to skin absorption or inhalation. No organs taken from control animals have yielded even traces of methyl alcohol.

The threshold of danger by inhalation is well below 1,000 p. p. m. of methyl-alcohol vapor. If this degre of toxicity obtained from monkeys applies to man, the vapors from 1 ounce of methyl alcohol entering the human body constitute a threat to life even when the exposure is distributed over 2 or 3 days.

In a later publication detailed reports will be made upon tissue pathology. Note is here made of extensive peripheral nerve damage.

On the basis of exposure, cubic centimeter for cubic centimeter, methyl alcohol is at least as toxic through inhalation or skin absorption as it is following oral intake.

In view of the very small amounts necessary to produce dire clinical conditions and death in animals most like man, it is reasonable to assume that practical hazards for human beings may be produced under conditions of apparently trivial exposures (4).

The toxic dose is difficult to fix. In air containing 0.15 per cent methyl alcohol, animals are not affected appreciably after 24 hours.

Loewy and Heide state that if the air that is breathed contains 0.2 per cent of methyl alcohol, it has a toxic effect on animals when inhaled for a sufficiently long period.

There has been found 0.005 to 0.6 per cent in workrooms. It is recommended that the concentration be kept below one part per 10,000 parts of air, i. e., 100 p. p. m. Some of the effect of methyl alcohol in industrial use is probably associated with its impurities, pyridine bases, and furfural.

Sollmann states that the impurities of wood alcohol play only a minor part in chronic intoxication, the methyl alcohol itself being the dominant toxic agent.

Macht says that when wood alcohol is introduced into the alimentary canal, it does not act as such, but exerts its most deleterious effects through its decomposition products, especially formaldehyde and formic acid. It is these secondary products that render wood alcohol more poisonous than ethyl alcohol when taken by mouth. When injected into the vein, however, and acting acutely upon the heart and brain, methyl alcohol is considerably less toxic than ethyl alcohol. In discussing the toxicity of alcohols, a distinction should

be made between the acute or immediate and the secondary or remote effects of the drugs.

In large doses, there is a marked drop in temperature, rapid breathing and then retardation, irregularity of the heart, convulsions and paralysis. In large doses, methyl alcohol is less toxic than ethyl alcohol. In small doses, methyl alcohol is more toxic than ethyl alcohol, probably due to the fact that it is more slowly excreted. The physical effects are irritation of the mucous membranes, eyes and respiratory passages, headaches, nausea, vertigo, loss of weight and poor appetite, with optic neuritis due to oedema and degeneration of the ganglion cells or interstitial neuritis of the optic nerve (2, p. 20).

Ethyl alcohol.—Boiling point 78° C. Air containing 300 p. p. m. causes no effect and at this concentration can be used safely in lacquers. * * * Said to cause an excessive amount of sugar to pass into the blood in rabbits (2, p. 21).

Butyl alcohol.—Boiling point 117° C. Air containing 100 p. p. m. produces a decrease in the red-cell count with relative and absolute lymphocytosis, also early liver degeneration and marked renal degeneration. * * * Experimentally * * * said to increase diastase liberation (2, p. 21).

Amyl alcohol.—Boiling point 126° to 132° C. Of all forms of alcohol used, amyl alcohol is the most toxic. One and five-tenths grams suffices to kill an animal averaging 1 kilogram in weight, while it requires 7.75 grams absolute ethyl alcohol and 6 grams methyl alcohol to produce the same results. The toxicity of amyl alcohol increases when evaporation is favored by the simultaneous evaporation of ether or ethyl alcohol. Amyl alcohol produces severe and uncomfortable irritation of the throat, headache, vertigo, and can not be tolerated in repeated doses.

Habituation to amyl alcohol is produced by its administration to dogs and rabbits (2, p. 21).

Diacetone alcohol.—Boiling point 164° C. It has a greater soporific and a less irritating effect than acetone. In dogs and rabbits it always retards respiration. It is stated that there is no previous reference to toxicity in the literature (2, p. 21).

Isopropyl alcohol.—Boiling point 82° C. It is of mild toxicity. Intoxication is similar to that of ethyl alcohol and tolerance is usually quickly established in animals, chicken and dog more than cat. Human beings reported increased ability to use their eyes during experimental ingestion. Acetone was present in the urine. Sensations of warmth, dizziness, tingling, and drowsiness were reported. It is soon tolerated. No harmful effect was found.

Macht states that "Efron found that isopropyl alcohol is less depressant than normal propyl alcohol for nerve fibers, and his observations harmonize with findings of the present investigation." (2, p. 21.)

Benzyl alcohol.—(Boiling point 206° C. It is oxidized readily to benzoic acid and excreted as hippuric acid with no bad effect. No good results from its therapeutic use are reported, but it is harmless (2, p. 21).

Esters.—Aliphatic esters and essential oils in paints tend to decrease thyroid activity in rats. The effect is similar to that of the alcohols and perhaps of other narcotics (2, p. 22).

Ethyl acetate.—Boiling point 77° C. Smyth and Smyth report as follows: "With ethyl acetate (used in largest amounts of all nitrocellulose solvents), at 2,000 p. p. m. of air, animals showed no effect." Four cubic centimeters per kilogram of body weight by mouth was injurious to dogs; 10 cubic centimeters per kilogram of body weight produced symptoms of salivation, somnolence, deranged coordination. Dogs receiving 100 to 300 milligrams daily for 4 to 6 days

showed urine nitrogen increased by 11.5 per cent; the increase was attributed to toxic action on the protein body as well as to an increase in metabolism. Small doses produce little effect on the circulation while large doses lower the blood pressure with a slight decrease in pulse rate. (2, p. 22.)

Butyl acetate.—Boiling point 125° C. Smyth and Smyth indicate that concentrations of 900 to 1,000 p. p. m. of air would appear to be reasonably safe. Apparently, the vapors caused an increase in total leukocyte count which was followed by a drop below normal. Lungs and kidneys showed beginning degeneration with exposure of guinea pigs to 500 p. p. m. 3 hours daily for 36 days (2, p. 22).

Amyl acetate.—Boiling point 138° to 142° C. This is considered by Smyth and Smyth to be among the safest of the solvents. Exposure to the vapor at 500 p. p. m. for 3 hours daily for 36 days showed no effect. In man a concentration of 60 milligrams in 12 cubic meters of air produces at the end of an hour cough and irritation of the throat.

Lehmann reports 20 milligrams per liter of air inhaled for 9.5 hours produced no effect except salivation in cats. In a cat, inhalation of 35 milligrams per liter of air caused loss of equilibrium and partial narcosis in 7 hours. In a man 5 milligrams per liter produced only an irritation of the mucous membrane; pulse remained normal and headache was absent.

Inhalation of the vapor causes nervous symptoms, headaches, nausea, torpor, inflammation of the respiratory tract and slight fainting attacks, sensation of oppression, and pain in the head. Symptoms disappear in two to three hours and recur on repeated exposure, but no case of chronic poisoning has been reported. In protracted animal experiments it irritates the air passages and causes fatty degeneration of the liver, but this has not been demonstrated in human beings (2, p. 22).

Alice Hamilton reports that workers making leather upholstery and auto tops using a solution of nitrocellulose in benzene, amyl acetate, and butyl acetate showed furunculosis, redness, tiny blebs, and small ulcers on the skin (2, p. 43).

Ethylene dichloride.—Boiling point, 83.5 C. The acute physiological response of guinea pigs exposed to air containing ethylene-dichloride vapors was determined. The concentrations of vapor and periods of exposure ranged from those which produced death in a few minutes to those that caused no apparent effect after several hours. The symptoms, gross pathology, and fatality are given, together with a brief discussion of potential health hazards.

1. In the order of occurrence the symptoms produced in guinea pigs by inhalation of ethylene-dichloride vapor are eye and nose irritation, vertigo, static and motor ataxia, retching movements, semiconsciousness and unconsciousness, accompanied by uncoordinated movements of the extremities, and death if exposure is continued. Exposure to 6 per cent vapors causes all these symptoms, excepting death, to occur in less than 10 minutes and death in about 30 minutes. Exposure to 1 per cent causes all the symptoms to appear in 25 minutes, with the possibility of death occurring a day or more following an exposure of about 15 to 20 minutes. Exposure to 0.12 per cent did not cause apparent symptoms or death following an exposure of eight hours. Tables in the report should be consulted for intermediate and additional data.

2. The gross pathological findings were hyperemia, congestion and edema of the lungs, with secondary degenerative changes in the kidneys. The severity of the pathology increased with the concentration of vapor and duration of exposure. The lung lesion was the most prominent and probably the greatest causative factor in death. No serious pathology was found for the following concentrations of vapor and periods of exposure: 6 per cent for 5 minutes,

1.7 per cent for 10 minutes, 0.4 per cent for 30 minutes, 0.2 per cent for 120 minutes, and 0.11 per cent for 480 minutes. Also these concentrations and exposures did not cause death of the animals.

3. The summarized physiological response given in the four degrees usually reported are 10 to 20 per cent kills in a few minutes; 0.4 to 0.6 per cent, dangerous in 30 to 60 minutes; 0.35 per cent maximum amount for 60 minutes without serious disturbances; 0.1 per cent, slight symptoms after several hours.

4. A comparison of the results obtained with those reported in the literature for other compounds indicates that for single exposures and periods of an hour or more the toxicity of ethylene dichloride appears to be of about the same order as gasoline, benzene, carbon tetrachloride, and chloroform. For periods of less than an hour it is less toxic than these compounds.

5. The odor of ethylene dichloride is distinct and noticeable, and warning symptoms are produced by relatively safe concentrations (5).

Dioxan or diethylene dioxide.—The second ether of ethylene glycol. Melting point, 11° C.; boiling point, 101.1° C. It forms a constant boiling mixture, 80 per cent dioxan and 20 per cent water, with a boiling point 86.8° to 86.9° C. at 742 millimeters of mercury. The odor is faint and pleasant, described by some persons as similar to absolute ethyl alcohol.

The acute physiological response of guinea pigs to air containing dioxan vapor was determined. The concentration of vapor and periods of exposure ranged from those which produced death to those which caused no apparent effect after several hours' exposure. The symptoms, gross pathology, and fatality are given, with a discussion of potential hazards.

1. The symptoms are principally those of eye and nasal irritation, with signs of lung irritation after long exposure, and narcosis with high concentration.

2. The principal gross pathological findings were congestion and edema of the lungs and hyperemia of the brain.

3. Because of the comparatively low vapor pressure of dioxan it is not possible to attain concentrations in air at 20° C. which will kill guinea pigs in less than two to three hours' exposure. Concentration of 1 per cent did not cause death after eight hours' exposure.

4. Dioxan vapor possesses warning properties manifested as eye, nose, and throat irritation. Persons exposed to 0.16 per cent in air, by volume, immediately experienced a slight irritation of the eyes and nose, with lacrimation. Exposure to 0.55 per cent produced a marked and discomforting degree of the same symptoms with the addition of a burning sensation in the throat.

5. Considering the comparatively low toxicity of dioxan vapor and the warning intensity of concentrations below those which produced serious harm to guinea pigs, it appears that health hazards from breathing the vapors are slight under ordinary conditions of usage and reasonable exposure. As in the case of practically all comparatively nontoxic volatile liquids, however, dioxan presents a hazard to life under conditions of exposure to air confined over the liquid in tanks, vats, and similar places where high concentrations would accumulate (6).

Ethyl benzene.—Boiling range, 95 per cent between 135.2° and 136.5° C. The acute physiological response of guinea pigs to air containing ethyl benzene vapor was determined. The concentration of vapor and periods of exposure ranged from those which produced death to those which caused no apparent effect after several hours' exposure. The symptoms, gross pathology, and fatality are given, with a discussion of the potential health hazards.

1. In the order of occurrence, the symptoms observed were eye and nose irritation, and apparent vertigo, static and motor ataxia, apparent unconsciousness, tremor of extremities, rapid jerky respiration, then shallow respiration, and finally slow, gasping respiration, followed by death. Exposure to 1 per cent caused all these symptoms and death in from two to three hours; 0.5 per cent caused all the symptoms up to and including tremor of extremities, but not respiratory disturbances and death during or after exposure of eight hours; 0.2 per cent caused all the symptoms up to and including ataxia in eight hours; 0.1 per cent did not cause symptoms other than eye irritation during eight hours.

2. The gross pathological findings were congestion of the brain and congestion and edema of the lungs. These were most severe for the exposures to 1 per cent concentration of vapor until death ensued. A more moderate degree of the same type of pathology was found in the animals killed for autopsy immediately after exposure to 0.5 per cent and to a less degree after exposure to 0.2 per cent. Gross pathology was not found in animals exposed to 0.1 per cent for eight hours. The degree of pathological changes increased in severity with increase in period of exposure to a given concentration of vapor. The pathology, however, decreased in severity during the 4-day period of observation following exposure and was absent in most cases after eight days.

3. From the standpoint of acute poisoning, as produced by a single exposure, the relative toxicity of ethyl benzene appears to be slightly less than that of gasoline and benzene in the range of high concentrations, and practically the same as that of gasoline and benzene in moderate and low concentrations.

4. Ethyl-benzene vapors are irritating to the eyes and upper respiratory passages in concentrations below those causing serious response. Also, other warning symptoms, such as vertigo, occur in advance of serious response from a single exposure.

5. The relatively low vapor pressure of ethyl benzene mitigates health hazards. Saturated air at 20° C. contains less than 2 per cent vapor.

6. It was not possible at room temperatures to attain a concentration high enough to kill guinea pigs in a short time. Exposure of from 30 to 60 minutes to 1 per cent by volume produces marked symptoms and is dangerous to life following exposure; 0.7 per cent is the maximum amount for 60 minutes' exposure without the occurrence of death and 0.3 per cent the maximum for 60 minutes without serious symptoms; 0.1 to 0.2 per cent is the maximum concentration for a single exposure of several hours (7).

Acetone.—Boiling point, 57° C. It is generally agreed that acetone is of low toxicity. Kagan found that repeated doses of 3 to 5 milligrams per liter of air caused no visible harm but did indicate that the animals became more tolerant. Doses of 8 to 10 milligrams to cats produced irritation of the visible mucous membranes, while with doses above 10 milligrams there was numbness and drowsiness after 0.5 hour, but with doses of 80 to 100 milligrams there was dizziness and incoordinate movements, i. e., alcohol stimulation with no narcotic stage.

Carozzi states that 0.590 gram per liter of air produced little effect on guinea pigs, and quotes Kohn-Abrest to the effect that the toxicity of acetone vapor is negligible compared with that of benzene and chloroform.

Acetone vapor in a concentration of 20,600 p. p. m. of air kills mice after 10 minutes' exposure. Prolonged exposure to the vapor has not the destructive action on tissues exerted by carbon bisulphide, benzol, or chloroform.

Experiments showed that 3.4 cubic centimeters of 5 per cent solution of acetone per kilogram per day was more poisonous to guinea pigs than ethyl

alcohol. One and eight-tenths cubic centimeters of 2.5 per cent solution per kilogram per day by mouth to rats was not fatal, even after four months' administration.

As to the potency of acetone, it is evident from Salant and Kleitman's investigation that its action is not very strong but is nevertheless considerable. It is particularly interesting to note that its effect was greatly augmented when a number of subminimal doses were given intravenously.

Intravenous injections in cats and dogs produced a marked fall in the blood pressure owing to cardiac depression, with prolonged apnoea. Acetone lowers the body temperature.

The physical effects are much the same as those of amyl acetate, a hot feeling with vertigo, slight fainting attacks, irritation of the throat and coughing. Kagan breathed air that had passed though a 10 per cent solution of acetone and on account of a burning sensation in the throat was unable to continue for more than five minutes. (2, p. 22.)

Cyclohexanone (not to be confused with hexaline or cyclohexanol).—One of the new solvents with a high boiling point (150° C.). Has an odor of peppermint. (See hexalin.) (2, p. 23.)

Tetrachlorethane.—Nonexplosive (boiling point 147° C.). Used as a solvent for fats and resins. It has a pleasant odor.

Its action is slow but the vapors are four times as toxic as those of chloroform. One attack makes the individual especially susceptible to another. Laboratory experiments on white rats produce the same picture as appears in man, i. e., obstructive jaundice with fatty degeneration of the liver and kidneys. In Lehmann's experiments with cats, 30 to 60 milligrams per liter of air for 5 to 10 minutes produced narcosis; 10 milligrams per liter of air for 30 to 60 minutes produced narcosis and convulsions; 5 milligrams for 30 to 60 minutes produced no effect. In early poisoning there occurred sweating, loss of appetite, general malaise, somnolence, vertigo, nausea, and abdominal pain, bad taste, and constipation. After some days jaundice, headache, and vomiting develop with final stupor and delirium; 7,300 p. p. m. are rapidly fatal with short exposure.

Minot and Smith state that even in the absence of clinical symptoms, characteristic blood changes appear. The large mononuclears are increased to 12 per cent and even to 40 per cent with increased platelets and slight diminution of red blood cells (2, p. 23).

Furfural.—(Boiling point 161° C.) It is an aldehyde with high boiling point. Its use is limited as a solvent. Experiments on gold fish show it to be one-third as toxic as formaldehyde and half as toxic as phenol. Large doses by mouth produce a paralytic action on frogs, similar to chloral. In increasing doses in white mice and rabbits there is unsteady gait with final paralysis, and marked cyanosis. Reflexes may be increased at first and then decreased with larger doses. In cats slight drowsiness was caused by 0.6 cubic centimeters of 5 per cent solution per kilogram of body weight; 0.12 cubic centimeter of the pure drug caused increased irritability, increased respiration and salivation with strychninelike convulsions. The effect in dogs is similar (2, p. 23).

Cellosolve.—(Boiling range: Not less than 95 per cent distills over from 130° to 136° C. at 760 millimeters) (8). (Monoethyl ether of ethylene glycol) (8) (2). The use of these compounds as solvents is recent but is increasing rapidly. Cellosolve acetate is used in high-grade automobile lacquers. Butyl cellosolve is chiefly used for brushing and dipping lacquers. They all have a mild odor.

Ethylene glycol itself injected intravenously in dogs rapidly lowers the blood pressure. This is followed by a marked increase in the amplitude of heart beat with slight increase in heart rate. Respiration at first is markedly increased followed by slowing with increased amplitude (2, p. 24).

The acute physiological response of guinea pigs to air containing cellosolve (monoethyl ether of ethylene glycol) vapor was determined. The concentration of vapor and periods of exposure ranged from those which produced death to those which caused no apparent effect after 24 hours' exposure. The symptoms, gross pathology, and fatality are given, together with a discussion of potential health hazards.

1. The symptoms exhibited after 18 to 24 hours' exposure to air saturated with cellosolve vapor (0.6 per cent by volume) were inactivity, weakness, dyspnea, and death. Exposure to 0.6 per cent for 24 hours caused death at the end of the exposure; 0.3 per cent for 24 hours caused death in 24 hours following exposure; and exposure to 0.6 per cent for 10 hours, 0.3 and 0.1 for 18 hours caused occasional death in from 1 to 8 days following exposure. Exposure to 0.6 per cent for 1 hour, 0.3 for 4 hours, and 0.05 for 14 hours caused no apparent harm.

2. The principal gross pathological findings were congestion and edema of the lungs; distention of the stomach, with numerous reddish-brown petechiæ scattered over the mucous membrane; and congestion of the kidney. The contents of the stomach were also discolored reddish brown. All these occurred in the animals that died during or soon after exposure. The congestion and edema were the principal findings in the animals that died 24 hours following exposure and broncho-pneumonia in the animals that died three days following.

3. Due to comparatively low vapor pressure of cellosolve it is not possible to create atmospheres at ordinary room temperatures which will produce serious acute poisoning in an hour.

4. Air saturated with cellosolve vapor at room temperatures produces a disagreeable odor and a moderate eye irritation. If these properties are heeded as warning of the occurrence of a potentially dangerous atmosphere it is believed that acute poisoning will not occur (8).

Ethylene oxide (CH_2CH_2O).—A colorless gas at ordinary room temperatures (boiling point 10.7° C.). Ethylene oxide is principally used as an intermediate in the synthesis of other compounds as methyl, ethyl, and butyl cellosolve (9).

The acute physiological response of guinea pigs to air containing ethylene oxide was determined. The concentration of vapor and periods of exposure ranged from those which produced death to those which caused no apparent effect after several hours' exposure. The symptoms, gross pathology, and fatality are given, with a discussion of the potential health hazards.

1. In the order of occurrence the symptoms produced are nasal irritation, eye irritation, blood-tinged, frothy, serous, exudate from nostrils, unsteadiness on feet and staggering, inability to stand, respiratory disturbances, dyspnea and gasping, and death. Most of these symptoms occurred with exposures to concentrations of 8.5 to 0.3 per cent by volume. Eye and nose irritation were the principal symptoms with exposure to 0.13 and 0.06 per cent; no distinct symptoms were observed with exposure to 0.025 per cent.

2. The principal gross pathological change was marked irritation of the respiratory system. This was most prominent in animals that died within a few hours following exposure. Lobar and lobular pneumonia and parenchymatous changes in the kidneys were noted in the animals that died two to six days following exposure.

3. Exposure to 5 to 10 per cent causes death after a few minutes' exposure; 0.3 to 0.6 per cent for 30 to 60 minutes is dangerous to the life of guinea pigs; 0.3 per cent is the maximum for 60 minutes without serious disturbances; and 0.025 per cent is the maximum allowable concentration for several hours without serious disturbances.

4. From the standpoint of relative toxicity (concentrations causing acute harm) ethylene oxide is less harmful than hydrogen chloride and sulphur dioxide, more harmful than chloroform and carbon tetrachloride, and similar to ammonia.

5. Ethylene oxide does not possess enough odor to give distinct warning of harmful concentrations, but it causes intolerable irritation to the eyes and nose when present in high concentrations, and moderate though distinct irritation in comparatively safe concentrations. This irritation must, however, be taken as warning of a dangerous atmosphere to avoid serious injury (9).

Petroleum naphtha.—The Condensed Chemical Dictionary published by the Chemical Catalog Co., New York, states that the term "naphtha" is now generally held to include all those petroleum fractions which boil below 280°, but that best practice dictates that the designation be confined to mixtures of light hydrocarbons. Reference is made to petroleum naphtha, benzene, and petroleum ether synonymously, as derived from petroleum and with a boiling point of 40° to 70° C.

Trade designations for volatile solvents and thinners have led to considerable confusion, particularly when one attempts to collate the results of investigations of physiological action as reported in the literature (2, p. 24).

All the petroleum naphthas * * * are stated by Prosser White to produce the same result on the skin, i. e., dermatitis. The skin becomes reddened, contracts, and feels dry and unpleasant (2, p. 43).

Benzine (not to be confused with benzene).—It is mixture of paraffin hydrocarbons which are liquid around 20° C.

According to Albough, with naphtha, gasoline, and petroleum benzine at a concentration of 0.02 gram per liter of air, local symptoms were produced and a concentration of 0.05 gram per liter of air caused poisoning. Substitution of this mixture for benzene in rubber factories is imprudent because while benzine is less toxic, it gives rise to slow cumulative poisoning. Benzine causes deviation of complement and if removed, the complement again takes its part in the haemolysis phenomenon (2, p. 24).

Gasoline.—Smyth and Smyth indicate that a 1,500 p. p. m. of air narcotic effects were produced in animals, also diarrhoea and an early temporary albuminuria. At the end of the test period they seemed to be in good condition although there was evidence of degeneration in the liver. There were no characteristic blood changes and the lungs were clear. The early disturbance would seem to show that 1,500 p. p. m. was about the limit of safe concentration.

Gasoline in concentrations of 11,000 to 22,000 p. p. m. is dangerous for even short exposure. The margin between the concentration which produces anæsthesia and that which causes death is narrow. Acute poisoning produces severe muscular twitchings during the stage of excitement (2, p. 24).

Toluene (toluol).—Inhalations of 1,600 p. p. m. proved fatal to rats within a few days, while 2,440 p. p. m. of benzene (benzol) did not, "due entirely to the narcotic effect of these solvents in accordance with the general increase in narcotic effect with added methyl radicals." When present in from 620 to 1,100 p. p. m. toluene (toluol) produced no measurable effect on animals.

At 1,250 p. p. m. it produces marked toxic degeneration of the liver and also pulmonary inflammation in animals. Smyth and Smyth suggest that within

reasonable exhaust ventilation, 1,000 p. p. m. will not be exceeded and at this concentration animals are not materially affected.

The benzol committee of the National Safety Council found that with intraperitoneal injections up to 0.7 cubic centimeter per kilogram of body weight (white rats), there were no symptoms except apathy, in the nature of narcosis rather than nerve irritation. With subcutaneous injections of 1 cubic centimeter per kilogram of body weight there were no constitutional symptoms except slightly lessened activity.

Commercial toluene (toluol), xylene (xylol) and petroleum ether are all toxic increasing in the order named but all less than benzene. "Aëration experiments, therefore, were not attempted because a high concentration of benzene is necessary to obtain definite poisoning under these conditions." The haemolytic action of serum is destroyed by these compounds but if the volatile chemical is removed, the hæmolytic power is restored.

A leukocytosis was present after the inhalation of toluene (toluol) by dogs. In dogs killed by inhalation there was more toluene in the spleen than elsewhere. Toluene is oxidized to benzoic acid which is then combined with glycocoll to form hippuric acid and excreted in the urine in that form.

Toluene vapors irritate the mucous membrane of the respiratory passages and the conjunctivæ more than the other more toxic substances (2, p. 24).

Xylene (xylol).—Smyth and Smyth found that at 450 p. p. m. of xylene (xylol) in air one animal died and two others were prostrated. At 300 p. p. m. there was not much evidence of definite harm. They indicate that with a moderate degree of exhaust ventilation to keep the concentration below 300 p. p. m. no harm should be produced. The benzol committee of the National Safey Council found its action the same as that of toluene (toluol) for the same concentration. At 1,600 p p. m. there was instability and incoordination with signs and symptoms of mucous membrane irritation, and narcosis; at 998 p. p. m. instability and incoordination, mucous membrane irritation, urine decreased and sirupy, no narcosis, spleen, and bone marrow hyperplastic, little actual specific damage; at 620 p. p. m. slightly, if at all toxic.

Xylene is oxidized on only one side chain and forms toluic acid which is excreted in combination with glycocoll as toluric acid (2, p. 25).

Tetralin (tetrahydronaphthalene).—This compound is used as a substitute for turpentine. It has a tendency to revert back to naphthalene in the body. It is apparently of mild toxicity

Pohl and Rawicz report that it may be fed in doses of 5 to 6 grams to rabbits of 2 kilograms without any noticeable effects, while Lewin states that rabbits die from the administration of 5 to 6 milligrams, narcosis first resulting.

Two painters using tetralin had early irritation of the mucosa, headaches, stupor, and dark green urine (2, p. 25).

Hexalin (hydroxyhydro aromatic compound—cyclohexanol).—The toxicity for frogs decreased as the number of hydroxyl groups increased. Some departures from this generalization occurred for individual organs.

In rabbits, hexalin gives increased excretion of ethereal sulphates and the presence of glycuronic acid in the urine. In biological behavior hexalin as a hydroxy-aromatic compound constitutes a link between the aliphatic alcohols and the aromatic phenolic substances.

Cyclohexanol acetate was well tolerated by animals (cat and rabbit) in amounts up to 4, 5, and 9 milligrams per liter of air. Above 9 milligrams narcosis was produced. Those with narcosis nearly all died while no changes were produced by nonnarcotic doses. In man, doses of 3 milligrams per liter produced only irritation of the mucous membrane, no headache, and a sweet taste perceptible from removal from vapors. There were no changes in animals

exposed to 6.5 to 9.5 milligrams per liter of air for five days of nine hours each. Measured by norcotic effect, it is about three times as toxic as amyl acetate (2, p. 25).

Dipentine (Boiling point 181° to 182° C.)—According to the Condensed Chemical Dictionary, published by the Chemical Catalog Co., New York, it is synonymous with dipentene, $C_{10}H_{16}$.

Gardner caused rabbits to inhale the vapor from 30 cubic centimeters of dipentene with a boiling point range of 116° to 200° C. in a dish placed in the cage. He reports no clinical effects except mild irritation of mucous membranes and production of some anaemia (2, p. 26).

A spraying lacquer distillate.—This consists of composite vapors from a lacquer distillate which may contain both paraffin and cyclic hydrocarbons.

Smyth and Smyth found that at 6,100 p. p. m. there were produced lung irritation and inflammation, and toxic degeneration of the liver and kidneys. With moderately good exhaust ventilation this concentration can be lowered (2, p. 26).

Coal tar naphtha.—The replacement of benzene by toluene has resulted in toluene scarcity with an increased use of its higher boiling point homologues.

The Condensed Chemical Dictionary, published by the Chemical Catalog Co., New York, defines these naphthas in part as follows:

High-flash naphtha.—Boiling Point 150° to 200° C. Flash point not below 37.8 C. Derived from coal tar or illuminating gas.

Heavy naphtha.—Boiling point 160° to 220° C. (about 70 per cent at 200 C.). Flash point about 78.3 C. Derived from coal tar or illuminating gas.

Solvent naphtha.—A mixture of small quantities of benzene and toluene with xylol and higher homologues from coal tar. Boiling Point of grade A—80 per cent at 160° C.; grade B—90 per cent at 160° C. Flash point about 25.6 C. Derived from coal tar or illuminating gas.

It is indicated that the term naphtha is loosely used, and is synonymous with "mineral naphtha."

The benzol committee of the National Safety Council tested the toxicity of high-flash naptha with a boiling point range of 156.6° to 212° C. Its composition is not definitely known but it will contain one or more of the following: Xylol, trimethylbenzene, tetramethylbenzene. At 567 p.p.m. it was somewhat irritating to rats; at 312 p.p.m. there was little, if any, effect. It is relatively nontoxic except for an irritating effect on the mucous membrane at higher concentrations. It has a pungent odor. The benzol committee of the National Safety Council found that with doses of 1 cubic centimeter per kilogram of body weight given subcutaneously there was lessened activity and slight loss of weight with a slight and transient reduction in red blood cells. With a 0.5 cubic centimeter dose given intraperitoneally, there was limpness and paresis and lowered body temperature, finally deranged equilibrium and weakness. The narcotic effect was similar to that produced by toluene and xylene (2, p. 26).

* * * Benzene from coal, benzine from paraffin and other light distillates are stated by Prosser White to produce the same result on the skin, i. e., dermatitis. The skin becomes reddened, contracts and feels dry and unpleasant (2, p. 43).

Tricresyl phosphate.—Commercial tricresyl phosphate as used in lacquers is largely, if not wholly, the phosphoric ester of ortho cresol. Smith, Elvove, and Frazier (10) found by animal experiments that tri-ortho-cresyl phosphate is the cause of "ginger" paralysis. The following have been abstracted from their articles (10) (11) on the subject.

Tri-ortho-cresyl phosphate stands out toxicologically apart, quantitatively as well as qualitatively, from either phenol, or the three isomeric cresols, or the phosphoric acid esters of phenol and para cresol.

The minimum lethal dose of tri-ortho-cresyl phosphate in the rabbit is 100 milligrams per kilogram, and as little as 50 milligrams per kilogram may result in definite symptoms which may occasionally prove fatal.

It seems likely that the results will ultimately show that the lethal dose of tri-ortho-cresyl phosphate in the chicken may be about one-half or 1 gram per kilogram, while 50 milligrams per kilogram or even less may be sufficient to produce paralysis.

The action of the ortho ester is persistent and cumulative.

Rabbits may survive 1 to 1.6 grams of tri-para-cresyl phosphate.

Summing up the results of the experiments on rabbits it may be concluded that tri-ortho-cresyl phosphate differs from the corresponding cresol or the other cresols or phenol or their phosphoric acid esters, first in toxicity and second in the manner of action.

The systemic action of tri-ortho-cresyl phosphate is slow.

Tri-ortho-cresyl phosphate given orally or intramuscularly produces in the calf, after a long latent period, a paralysis of the extremities comparable in every detail with the human "ginger paralysis."

A pharmacologic study of the action of the phosphoric acid esters of phenol and some of the cresols has shown conclusively that tri-ortho-cresyl phosphate, and in so far as the present evidence goes, it alone, can produce in experimental animals a specific type of motor paralysis of the extremities in every sense comparable with that which occurred recently in human victims who drank of an adulterated fluid extract of Jamaica ginger.

Some of the differences in species susceptibility to tri-ortho-cresyl phosphate previously reported appear now to be due to differences in its absorbability from the alimentary canal. Certain other differences in species susceptibility can not yet be accounted for on the basis of our present limited knowledge of the manner of action of this poison in the animal body.

Pharmacologic evidence has been presented to show conclusively that the adulterated fluid extract of Jamaica ginger used for beverage purposes, resulting in an epidemic of partial paralysis, contained tri-ortho-cresyl phosphate to the extent of about 2 per cent. The chemical evidence we have secured confirms the pharmacologic evidence and fully harmonizes with it.

The etiologic relationship of tri-ortho-cresyl phosphate to the recent epidemic of so-called ginger paralysis is thus definitely established (10).

The present results in chickens with the ether-soluble fractions given in doses equivalent to 120 to 225 milligrams of the ester per kilogram must be considered as conclusive proof of the occurrence of the specific paralyzing ortho cresyl phosphoric ester in some of the adulterated ginger extract distributed recently in Los Angeles and surrounding territory. Since the highest concentration of the specific ester found was not over 0.5 per cent, or only about one-fourth that found last year, the susceptibility of man to this unique poison is of considerably higher order than was hitherto suspected, provided of course that the testimony of some of the recent victims that not more than one 2-ounce bottle was drunk, may be accepted.[3] (11)

Diethyl phthalate.—This is probably the most commonly used plasticizer. It has been used as a denaturant for alcohol so that tests exist for its detection in solution.

[3] If the drinking of 2 ounces of a 0.5 per cent solution of tri-ortho-cresyl phosphate caused paralysis in these cases, then it may be said that approximately 300 milligrams (5 grains) of this substance is capable of producing paralysis in man.

Doses up to 0.5 per cent of body weight, by mouth, cause rats to have pain, weakness, and distress at first, but after 24 hours they are normal. Human beings working in contact with this substance are not poisoned, but in solution in ethyl alcohol it causes irritation of the mucous membranes (2, p. 26).

Colors.—Coal-tar dyes are mainly used as colors; while cases of poisoning from this source among painters are not recorded, some of them are known to be harmful. The physiological effects of a number of them have been investigated with reference to their use in foods (2, p. 17).

Amino compounds of benzene and toluene.—These compounds are influenced in their toxicity by the substitution of methyl or other groups.

Toluidine vapors inhaled continuously over a period of six months produced in mice, rats, and rabbits, hyperemia, bronchitis, peribronchitis, and finally bronchial pneumonia and abscess formation with fatty changes in the kidneys, epithelium, myocardium, and liver.

Toluidine (and naphthylamine) are eliminated through the lungs and urine only when present in large amounts. They are partially changed in the blood into an alkaline salt of *p*-amino phenol sulphuric acid. They destroy the red blood cells and produce methæmoglobin.

Introduction into the nucleus of aromatic compounds like toluene, of NH_2 to form metatoluidine markedly increases the toxicity for frogs. The introduction of NO_2COOH, or chlorine in the meta position increases the toxicity for frogs slightly. All these compounds act on the central nervous system but the NH_2 group causes also cardiac paralysis. Unlike the NH_2 group, the NO_2 and COOH groups in the meta position exhibit strong exciting tendencies.

The action of the aromatic nitro substitution products and amino compounds is primarily upon the blood. By converting oxyhaemoglobin to a methaemoglobin they give rise to symptoms of anæmia. The amino compounds undergo a partial oxidation in the body with the formation of amino phenol or its derivatives. Their toxic action is exerted largely through this substance. In addition to their action upon the blood the nitro substitution products act to some extent upon the central nervous system, causing first stimulation and then paralysis. With the amino compounds this action is much less. In chronic poisoning the liver may become involved from the excessive destruction of the red blood cells and jaundice results.

Aniline does not produce methaemoglobin. It has a direct toxic effect on heart muscle and the conduction mechanism. In the acute stage there is a relative increase in erythrocytes, haemoglobin and oxygen capacity with low blood pressure simulating a condition of shock. In chronic poisoning there is anæmia with emaciation and weakness.

In the studies of tortoise heart, if the toxicity of aniline is one, the toxicity of para and metatoluidine is two and three and of orthotoluidine four (2, p. 17).

Summary

The men employed in spray-coating airplane wings in the dope shop of a naval air station had been drinking milk as a preventive measure against dope poisoning. This procedure was questioned and a search through available literature was instituted to determine the effectiveness of milk drinking as a prophylaxis against possible dope poisoning in coating airplane wings by the spray method. The search was extended to cover, as far as available data permitted, a

consideration of the more or less dangerous materials found in airplane dopes, the manner in which the toxic substances might enter the body, and the manner of controlling health hazards when applying dopes by the spray method.

The subject was extended because it became evident that airplane dopes (or spray lacquers) are composed of numerous substances of varied physical and chemical properties, in many cases toxic in nature, which are used as solvents, plasticizers, diluents, and driers, and of cellulose nitrate or cellulose acetate which are used as film base compounds.

The ingredients of cellulose-derivative spray lacquers in general are in many cases identical with the ingredients of the preparations used in the Navy known as airplane dopes. Due to constant chemical research new organic compounds are continually being developed resulting in frequent changes and interchanges of the lacquer constituents. This, of course, applies not only to spray lacquers in general but also to Navy airplane dopes for which no standard formula is prescribed by specifications.[3]

A brief description of the progress of the search with information pertinent to the subject, obtained from various sources, is presented, followed by a list of organic compounds which may be constituents of airplane dopes either in fact or as a possibility. Information on the toxicity of these compounds as found in the avilable literature follows each item listed. Substances about which no toxicity information was found are not listed.

Conclusions

A study of the data found in available literature applicable to the potential health hazards in applying airplane dopes by the spray method, elicits the following conclusions:

1. *Airplane dopes, nature of.*—(*a*) In the main, airplane dopes consist of cellulose nitrate or cellulose acetate as film base compounds, dissolved principally in organic esters as solvents, mixed with high boiling point esters as plasticizers, and the whole mixed with alcohols and/or hydrocarbons as diluents, with other organic compounds added as dryers.

(*b*) The organic compounds used in airplane dopes that might be classed as the more toxic are : Benzol (benzene), tetrachlorethane, tri-ortho-cresyl phosphate, methyl, butyl, allyl, and amyl alcohols turpentine, benzine, gasoline, toluol (toluene), xylol (xylene), and tetralin.

[3] Except in the case of "airship dope" which is used for application to rubberized airship and balloon fabric.

(*c*) It appears certain that satisfactory coats can be produced without the aid of benzol, tetrachlorethane, and tri-ortho-cresyl phosphate; therefore these substances should be excluded from Navy airplane dopes. Present Navy specifications prohibit the use of tetrachlorethane. Benzol is prohibited in pigmented and semipigmented dopes, which prohibition should be extended to include all Navy airplane dopes in the case of benzol as a solvent. No dopes should be used for spraying which contain over 0.5 per cent of benzol that might be present by accident. Tri-ortho-cresyl phosphate is capable of producing paralysis probably in very small doses. This does not seem to be the case with either tri-para or tri-meta cresyl phosphate. Therefore, if it becomes necessary to use tricresyl phosphate in Navy dopes it should be insisted upon that the para or meta isomer be furnished and the use of the ortho isomer prohibited.[4]

(*d*) With the elimination of the three organic compounds mentioned above it must still be considered that airplane dopes contain substances that are more or less toxic and that applying these dopes by the spray method constitutes a definite health hazard for which precautions are necessary. The worker is subjected to an atmosphere containing toxic substances in vapor form or droplets that may enter the body principally by inhalation, to a limited extent by ingestion, and rarely by skin absorption.

(*e*) Airplane dopes should bear a label that lists the ingredients, with the percentages of each constituent clearly indicated. Dopes bearing trade name labels only should not be accepted for Navy use. In the case of tricresyl phosphate the label should indicate which one of the three isomers of tricresyl phosphate is being furnished. (As stated previously, the ortho isomer should be prohibited.) Further investigations may call for more specific labeling in cases of other compounds now in use or which may in the future be used in dopes.

(*f*) A standard formula for Navy airplane dopes should be prescribed that will exclude the most dangerous toxic substances, that will as far as possible reduce the necessary toxic ingredients to a concentration below that which constitutes a health hazard, and that will furnish a preparation for Navy use that will not be subject to frequent changes in composition, thereby eliminating the danger of spraying unknown poisonous substances. When such a standard preparation has been adopted, no other dope combination should be accepted for Navy use.

2. *Prophylaxis.*—(*a*) Milk drinking as a prophylaxis: The presence of food in the stomach is desired because the influence of proper

[4] Recent investigation indicates that triphenyl phosphate is also a highly toxic compound and constitutes a distinct hazard, but published details regarding it are not available at this writing.

and sufficient food may assist to some extent in warding off increased action of poisons that may be due to the fasting stomach, but no food of any kind is to be eaten in the dope shop where there is a possibility of its contamination with the dope poisons. Milk drinking appears to be of nutritional value only (except possibly in the case of lead), and milk itself may be considered an excellent food as such, but its inclusion in the daily diet is optional because of the fact that the Navy ration of three (hot) meals a day is always available, and therefore at naval stations the fasting stomach would not seem to be an entity.

(*b*) Ventilation: Exhaust ventilation is absolutely essential wherever any type of airplane dope is being sprayed. Exhaust ventilation installations should be designed and constructed with proper duct size and apparatus should be properly adjusted to remove the dope-charged air and fumes by an air movement past the sprayer toward the exhaust at a sufficient rate of speed to keep the concentration of harmful material in the air breathed by the worker below the danger limit. To prevent the exit of the charged air other than through the exhaust the intake of air should not exceed the exhaust.

Spraying should be carried on in a part of the building set aside and equipped exclusively for that purpose, and exhaust ventilation should be continued after spraying is completed to remove the fumes that collect while drying. If the objects sprayed are removed to another apartment for drying this apartment should be properly equipped for exhausting fumes and the apartment used for no other purpose.

(*c*) Spraying machine: The spraying equipment should be provided with automatic shut-off and any other device that will prevent the toxic spray from escaping into the workroom. The air pressure should be the lowest that will provide a satisfactory finished coat. The nozzle of the spray gun should be held at least 1 foot distant from the object sprayed, in order to avoid rebounding of the spray as much as possible.

(*d*) Air masks, helmets, etc.: Dopers should be required to wear an efficient air mask or helmet while spraying dope. A mask or helmet of the hose type should be furnished (not gas masks of the type used in chemical warfare). Uncontaminated air should be supplied into the mask at sufficient pressure to barely exceed that required by the sprayer so that the escaping excess air will prevent the dope charged air of the workroom from entering the mask.

(*e*) Medical examination: In all cases, men who are to be detailed as dope sprayers should be given thorough physical examinations, which should include complete blood examinations, prior to beginning such employment and periodically thereafter (every three months) as long as they continue as sprayers. If at any time it is noted that a

man has been affected by the spraying materials to any extent whatsoever he should be immediately removed from the detail and not allowed to resume his duties as sprayer except on medical advice and under medical supervision. Sprayers should be encouraged to report to a medical officer when feeling sick to any degree.

(*f*) Observance of regulations: It is common knowledge that workers do not like to wear cumbersome safety devices; nevertheless these devices are necessary to reduce the health hazards to a minimum. Regulations and instructions providing for the necessary protection of the worker should be issued and the operators should be required to observe such regulations and instructions.

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PUBLIC HEALTH CONDITIONS IN SOUTH CHINA

A Brief Sanitary Survey of Canton, Wuchow, Hong Kong, and the Delta Area, 1932

By A. C. Surber, Jr., Lieutenant (Junior Grade), Medical Corps, United States Navy

CANTON

General description.—Canton is the capital of the Province of Kwangtung on the south coast of China, and extends for 4 miles along the north bank of the Pearl River, 60 miles from Hong Kong. The island of Shameen constitutes the foreign concession.

Climate.—Ordinarily the temperature range is from 42° F. to 96° F. The hot season lasts from May to October. The rainy season usually lasts from February to May. Snow is rare. Summers are hot and very humid. Typhoons may occur, usually in August. As a rule, the weather from October to February is excellent.

Population.—The estimated population is 1,200,000. No reliable vital statistics are available.

Organization of board of health.—The Canton municipality is organized as a separate entity under the provincial government of Kwantung. The municipality has six departments, one of which is the department of health. This department, headed by a commissioner, is subdivided into divisions of sanitation, prevention, health, education, and vital statistics. In rural districts matters of public health are administered by the district magistrate.

Water.—The water supply comes from three sources: (1) The city water works, (2) public and private wells (contaminated, but extensively used in the suburbs), (3) the river (turbid and badly contaminated with sewage).

In 1905, during the Ching dynasty, the late governor of Kwantung organized the first water works, on the bank of the West River at the western limits of the city, a slow sand-filter type. Operation was begun in August, 1908.

In 1929, due to flagrant financial and sanitary failure of the old water works company (1,506 deaths from typhoid in three years) the municipal government took over full control, and a new rapid-gravity plant was placed in operation the middle of 1931, using the West River as a source, at Tsing Po. During the past year (1931–32) only 112 typhoid cases have been reported, with 21 deaths. It must be remembered, however, that of the 1,200,000 inhabitants of Canton, only about 200,000 use the city water supply (5).

Although bacteriological examination of the city water shows *B. coli* present in 50 cubic centimeters as the smallest determinate volume, it is wise to caution liberty parties that the city water is probably unsafe and to drink only certified bottled beverages.

Shameen water supply.—Shameen has its own water works, small but of similar type, and similar precautions should be observed.

Sewage.—Excreta in houses and public latrines is collected only in the morning, transported to boats, and sold as fertilizer. The sampan population either dispose of excreta directly into the river or sell as fertilizer. Plans have been made to construct 1,139,000 feet of new sewers, but at present all pipes lead to the "six main drains" (42,000 feet) constructed several hundred years ago, many of which are blocked (7).

Soil pollution is common in rural communities; hookworm infection is the natural sequel to the use of human manure. Direct defecation into fish ponds and rivers is responsible for the infection of fish and mollusks with liver fluke and tapeworm. Schistosomiasis is not common in South China.

Consequently, only cooked native vegetables should be eaten. If native lettuce, cabbage, or tomatoes are served raw, they should be previously dipped in boiling water and permanganate solution 1:5,000) and thoroughly washed.

Refuse, garbage, ashes.—A sanitary tax of 30 to 40 cents Canton currency is assessed monthly against first-class houses. This tax is farmed out to a private company by the department of public safety of the municipality. The company provides scavengers to remove garbage and refuse. Streets are swept daily and the avenues sprinkled daily in summer. Refuse collected is sold for fertilizer. Revenues from this monopoly aggregate $500,000 Canton currency annually (3) (7).

Vital statistics.—No reliable vital statistics are available, so only estimates can be made. Legal requirements are not enforced.

The crude death rate in 1921 was estimated at 3.7 per 1,000, obviously an absurdly low figure. Reports of the Bureau of Public Health show statistics of births and deaths in Canton, from January 1 to December 15, 1931, as follows (9):

	Male	Female	Total
Births	5,737	4,834	10,571
Deaths (infants)	2,086	2,787	4,873
Total deaths	6,047	3,392	9,429

Based on these statistics, Canton has a birth rate of 12.08 per 1,000 (official estimated total population of 875,000 (?)).

On the same basis the death rate totals 17.76 per 1,000, which is considered too low by competent observers. (4) (6). According to Doctor Oldt of the Canton Hospital (an American public health specialist), a more accurate and conservative figure should be a death rate of from 25 to 30 per 1,000.

A probable estimate of infant mortality (Doctors Chau and Wright—based on 12,291 case histories at the Canton Hospital) reveals the staggering total of 50 to 70 per cent. Many children die of tetanus infection of the cord. Native midwives are largely responsible (1).

The chief causes of morbidity, in order of importance, are: Tuberculosis, malaria, syphilis, beri beri, typhoid, pneumonia, nephritis, and influenza.

The chief causes of mortality are: Tuberculosis, gastro-enteritis, dysentery, typhoid, pneumonia, malaria, nephritis, influenza, syphilis, and beri beri.

Milk supply.—The only milk from native dairies that can be drunk safely, without boiling, is that from the dairy of the Canton Christian College, although the instructor of animal husbandry at Lingnan University states: "Because of the danger of tuberculosis and typhoid it certainly is not safe to drink milk produced locally unless it is first pasteurized" (10).

The Dairy Farm Co. of Hong Kong (British management) maintains a branch on Shameen, and meat and dairy products obtained from this source may be regarded as quite safe in spite of the recent diphtheria epidemic (January, 1932) attributed to a contaminated milk supply.

Sanitary nuisances.—The following nuisances may be cited: Public cesspools; stagnant water; mosquitoes; flies and rats; defective plumbing, drainage, and sewage disposal; crowding; dust hazard; poor heating facilities; gambling and opium dens; universal hawking and spitting in public places; use of common towels in restaurants; dead animals and defecation into the rivers; sale of decayed meats; unwholesome milk; and raw fish mixtures.

Industrial hygiene.—There is none. Workmen are not protected from trade risks. Women and children perform heavy manual labor, a practice peculiar to South China. Trade unionism is strong, however, and these organizations strike for better hours and wages (2), (3), (7).

The silk industry is the principal one. There are virtually no other factories (except brick and tile kilns) in the entire consular district. The household system prevails generally. These silk filatures are actually "sweat shops," and the general hygienic conditions are very unsatisfactory (8).

Housing.—City dwellings are exceedingly crowded, but whether city or rural, the average houses have stone or dirt floors, without cellars, and are poorly lighted and ventilated. In poorer sections sleeping quarters may be relayed, and many families may occupy the same room. The toilet is usually immediately adjacent to the kitchen in the rural districts; crude mud-brick and thatching are used (3).

Infectious diseases.—Tuberculosis, cholera, smallpox, plague, leprosy, typhoid fever, meningitis, and dysentery are among the important communicable diseases. There are two leprosaria, Shek Lung and Shu Tan.

Notification is required but not enforced. Quarantine is not enforced.

There are sporadic epidemics of cholera (which is constantly *endemic* in the city), meningitis, smallpox, diphtheria, scarlet fever, mumps, and measles.

Venereal diseases of all types prevail universally, are not reported, and are not controlled. In addition to prostitution in public houses in the city, a tremendous additional exposure hazard is provided by the large floating population ("flower boats"). The sampan women constantly solicit enlisted personnel.

As is evident from the annual sanitary report of the U. S. S. *Mindanao*, venereal disease incidence in 1931 showed a particularly gratifying decrease, reflecting vigorously prosecuted preventive measures (see General Order 69A and Ship's Orders). There were only 11 admissions in 1931 from all types of venereal disease, as compared with 35 in 1930 and 31 in 1929. A study of venereal disease incidence conducted in 1930–31 showed that the expectancy rate for the patrol could be placed at 136 and for the *Mindanao* at 33, so that the 1931 admission rate was well below normal expectancy.

Hospital facilities.—Ordinarily, emergency surgical cases, or infectious cases requiring isolation, are referred to Doctor Thomson at the Canton Hospital. Medical (noninfectious) cases requiring special nursing care may be sent to the nursing home (Dr. R. L. Lancaster) on Shameen.

WUCHOW

General description.—Wuchow, the "gateway" city of Kwangsi, is located at the junction of the two great rivers (the West and the Fu) which tap the province with its 8,000,000 people, and is naturally of great commercial importance.

Population.—The estimated population is 100,000.

Board of health.—Until its recent discontinuance due to political unrest, the Chinese Government maintained a large office of public health and a considerable staff, under the local magistrate (6).

There is an attempt at free inoculation every year against smallpox, and many take typhoid and diphtheria prophylaxis (6).

Water.—The water supply comes from two principal sources: (*a*) Public and private wells, and (*b*) the rivers. However, construction of a large waterworks is being rapidly carried forward and will be in operation by the middle of 1932.

Liberty parties in Wuchow should drink bottled beverages of recognized brands only (avoiding Chinese soft drinks). The water is unsafe except at private European homes.

Sewage and refuse.—A modern sewer system has been laid and the main streets are well paved. Drainage is fairly adequate. Refuse is properly disposed of.

Vital statistics.—No reliable statistics are available. Regulations are not enforced. Statistics are required of all hospitals and physicians (that are compulsorily registered), and deaths and births are fairly well reported.

Sanitary nuisances.—Wuchow is much smaller than Canton, and the city proper is much cleaner. Extreme squalor, however, prevails in the outlying districts, particularly the low-lying eastern part.

Stagnant pools; contaminated wells; mosquitoes, flies, and rats; suburban squalor; dust hazard; public spitting; river pollution; and unwholesome foods may be cited as nuisances.

Industrial and housing situation.—Essentially the same as in Canton, but on a smaller scale.

Diseases.—Wuchow is an endemic focus for malaria. Hookworm infestation is common, as well as the common intestinal parasitic diseases, and smallpox, cholera, typhoid fever, dengue, diphtheria, cerebrospinal meningitis, and dysentery are epidemic sporadically. Bladder stone is common.

Opium addiction is not unusual. Venereal diseases are common and uncontrolled.

Hospital facilities—medical practice.—The Stout Memorial Hospital (Baptist), under the able direction of Dr. Geo. W. Leavell, provides over 200 beds.

The Government operates a well-kept hospital.

In the Province itself, Nanning (the capital) has two small hospitals, both under mission boards. A fairly well-equipped Government hospital is maintained at Low Chow. At Kweilin, two mission hospitals are operated.

The crying need is for a central tuberculosis sanitarium and a modern lying-in hospital. There are no such institutions in either of the two Kwang Provinces, with a population totaling 35,000,000 (4), (6).

HONG KONG

This modern English city, with its splendid public health administration, compares favorably with many of our American communities. The city water is perfectly safe. The dairy farm provides products of exceptionally high standard. The Chinese quarter is

well regulated. Quarantine is ably administered. Occasional epidemics occur, such as the recent diphtheria and meningitis outbreaks, but these are unusual.

Hospital facilities.—The best private institution is probably the French hospital in the Wanchai district. There is a Government civil hospital and an infectious hospital, Matilda Hospital, on the Peak, is maintained for charity cases. The Peak Hospital cares for private cases.

United States naval personnel are admitted, by courteous arrangement, to the Royal Naval Hospital, upon prior notification of the commodore aboard the H. M. S. *Tamar*.

In case of infectious diseases among the Chinese personnel, the port medical authority should be notified. He will provide facilities for removal and hospitalization in the Government hospital.

Pratique is automatically granted all United States naval vessels, even if coming from an infected port.

THE DELTA AREA (SI KIANG OR WEST RIVER)

The general precautions cited above regarding water, fresh fruits, and vegetables and native foods, should be strictly observed by all personnel going ashore anywhere in the delta area.

Sanitary nuisances and menaces to health essentially similar to those already listed prevail generally.

Hospital facilities.—Kong Moon (The Port): An excellent general hospital (150 beds) is maintained here by the Canadian Presbyterian Mission under the competent supervision of Doctors McClure and McDonald.

Typhoid fever, malaria, and parasitic infestations are common, and the region is remarkable for its extremely high incidence of bladder stone.

Siulam: A small hospital-dispensary (15 beds) is maintained in this city of canals by the United Brethren Mission in affiliation with the girls' school. The resident physician and nurses are Chinese. Doctor Oldt of the Canton Hospital makes periodic visits.

Takhing: The Reformed Presbyterian Mission supports a small hospital (The Gregg Memorial, 15 beds), under Chinese staff and direction.

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QUESTION AND ANSWER

The following letter was received from a medical officer:

From: Medical Officer.
To: Bureau of Medicine and Surgery, Navy Department, Washington, D. C.
Subject: Information, request for.

1. It is requested that the following information be furnished this activity:
   10 most common injuries in aircraft crashes.
   10 most common suicidal poisonings.

As it is believed that the information sent in reply to the above letter may be of interest to others in the Service, the statistics requested are published in the following tables:

TABLE I.—*Diagnosis in aircraft accidents (including flight and ground accidents)*

	1925	1926	1927	1928	1929	1930
Bruises	30	38	19	25	24	25
Burns, scalds, and sunburns	4	3	11	11	16	13
Concussions	3	0	2	1	1	0
Cuts and lacerations	23	19	25	30	23	29
Punctures	2	2	1	2	1	0
Dislocations	2	5	0	2	3	3
Fractures	29	31	28	30	24	27
Sprains and strains	11	18	16	21	13	15
Drowning	5	4	5	12	2	4
Injuries, multiple, extreme	24	19	19	29	19	23
All others	12	2	4	6	4	6

TABLE II.—*Suicides and attempted suicides by poisoning, 1925–1930, inclusive*

Poison:	Cases
Iodine	18
Bichloride of mercury	16
Illuminating gas	6
Lysol	6
Veronal (barbital)	6
Phenol	4
Potassium cyanide	4
Arsenic	2
Chloral hydrate	2
Formaldehyde	2
Lye or lyewater	2
Nicotine	2
Strychnine	2

	Cases
Strychnine sulphate	2
Unknown substance	2
Button polish	1
Chloroform	1
Cocaine	1
Cocaine hydrochloride	1
Commercial muriatic acid	1
Copper salts	1
Copper sulphates	1
Coryza tablets	1
Digitalis	1
Digitalis folium	1
Methyl salicylate and mercurochrome	1
Mecuric chloride	1
Morphine	1
Nitric acid	1
Opium	1
Potassium ferrocyanide	1
Potassium permanganate	1
Roach paste	1
Sodium cyanide	1
Sloan's liniment	1
Zonite	1

TABLE III.—*Fatal suicidal poisoning, 1925–1930, inclusive*

Poison:	Cases
Illuminating gas	[1] 16
Bichloride of mercury	5
Strychnine	2
Strychnine sulphate	2
Formaldehyde	2
Phenol	2
Potassium cyanide	2
Chloral hydrate	1
Chloroform	1
Cocaine	1
Commercial muriatic acid	1
Lysol	1
Methyl salicylate and mercurochrome	1
Nicotine	1
Nitric acid	1
Opium	1
Sodium cyanide	1
Veronal (barbital)	1

ORPHANS ENJOY SUPERIOR HEALTH ADVANTAGES

One can not avoid the conclusion that the proper place to determine the true value of prophylaxis in communicable diseases is in those localities or institutions having a high percentage of nonimmunes.

[1] Includes 10 deaths recorded as "Asphyxiation, illuminating gas."

The orphanage presents such conditions and thus particular interest attaches to the reports of the experiences of Mooseheart, Soldiers Orphans' Home, and St. Mary's Orphanage. The results obtained in these institutions are summarized in the June 15 copy of the Illinois Health Messenger, the official bulletin of the State Department of Public Health.

The Soldiers Orphans' Home under the medical care of Doctor Peairs has a child population of about 700. Eight years ago diphtheria was banished by the use of toxin-antitoxin and smallpox has not appeared since the beginning of the institution. In 1925, the attack on scarlet fever started and until 1929 the cases varied from 4 to 75 annually. A single case occurred in 1929 and there have been none since. All children are given the Dick test and nonimmunes are immunized with five doses of toxin. At the time of admission to the home about one-third of the children were found to be nonimmunes.

Mooseheart has 1,500 orphan children. Immunization against diphtheria started in 1919. Except for one case in 1920, diphtheria has been absent. Prior to 1926 scarlet fever was present every year. After an extensive epidemic of 67 cases in the fall of 1926, immunization of nonimmunes was started. Since that time eight cases have occurred in the nonimmunized and no cases among the immunized.

At St. Mary's Orphanage, Doctor Earle has had similar success with 1,000 children.

It must be remembered that these striking results were obtained during a period when the State of Illinois had 111,060 cases of diphtheria with 8,872 deaths and 89,814 cases of scarlet fever with 1,530 deaths.

PELLAGRA PROPHYLAXIS

The following note from Georgia's Health, the publication of the Georgia Department of Public Health, states briefly the latest work in the prophylaxis of pellagra:

The Turnip Patch

Sow turnip seed; eat turnip salad; have plenty of greens, is the advice of Doctor Abercrombie, the director of Georgia Department of Public Health.

The season is not too late to sow a turnip patch, and everyone should have plenty of greens; it is important from the standpoint of health.

It has been proven that a liberal supply of greens in the diet will prevent pellagra and quite likely other diseases of like character.

Have a late garden, and in that garden plenty of turnips.

HEALTH OF THE NAVY

The general admission rate, all causes, based on returns for April, May, and June, 1932, was 521 per 1,000 per annum, as compared with

497, the rate for the corresponding months of 1931. The median rate for the second quarter, as indicated by the records of the preceding five years, is 502. The admission rate from disease was 470 per 1,000 per annum, as compared with 457, the 5-year median for the corresponding three months. The admission rate from accidental injuries was 51. The median or expected rate for the corresponding quarter of the previous five years is 48.

Although there were outbreaks of catarrhal fever and influenza during the first quarter of the year, common acute respiratory diseases in general did not exceed expectancy at shore stations in the United States during the second quarter. A total of 612 cases of these diseases were reported, 305 of which were notified in April, 170 in May, and 137 in June. As usual, catarrhal fever predominated.

The United States naval training station, San Diego, Calif., reported 106 cases of catarrhal fever, which was slightly more than 25 per cent of all cases notified by shore stations in the United States. The other three naval training stations were remarkably free from this disease.

Two cases of cerebrospinal fever were notified by the training station, San Diego, Calif., one in May and one in June. In both instances the diagnosis was definitely established after the patients had been transferred to the hospital. One case of scarlet fever was reported by the United States naval training station, Great Lakes, Ill., in April.

An outbreak of German measles comprising 50 cases occurred at the United States naval training station, Norfolk, Va., the first part of June. Apparently effective control measures were promptly instituted, as only one active and two suspicious cases remained in isolation June 30. This station had an outbreak of German measles during the corresponding quarter of last year.

The admission rate, all causes, for forces afloat was 486 per 1,000 per annum for the quarter, as compared with the corresponding median rate for the preceding five years, which is 444. The increase over the expected rate was due to acute respiratory diseases, which have been prevalent among forces afloat since the first of the year. There were 852 cases of catarrhal fever reported from all ships during the quarter. These cases were distributed among a large number of ships and very few outbreaks occurred. The U. S. S. *Hannibal* reported 68 cases; the U. S. S. *Arkansas*, 42 cases; and the U. S. S. *Beaver*, 36 cases. Only 13 cases of influenza were reported during the quarter. One case of cerebrospinal fever was notified in April from the U. S. S. *Canopus*, in Chinese waters. The U. S. S. *Sacramento* reported 1 case of diphtheria in April and the U. S. S. *Rochester* 1 in May. Seven cases of scarlet fever were notified by forces

afloat during the quarter. The U. S. S. *Texas* reported 1 case in April and 2 in June; the U. S. S. *Dobbin*, 1 in April; the U. S. S. *Nevada*, 1 in April and 1 in June; and the U. S. S. *Rochester*, 1 in May.

A fatal case of septicemic plague was reported by the U. S. S. *Palos*, at Kiukiang, China. The patient, 19 years of age, was admitted to the sick list June 22. After three days his only symptom was an elevation of temperature. He died early in the morning of the fourth day. Post-mortem examination indicated death occurred from plague, septicemic in type. A blood smear from the spleen showed *B. pestis.*

The medical officers of the naval hospital, Guam, and forces in China reported an unusual prevalence of catarrhal fever and measles during the months of April, May, and June.

TABLE No. 1.—*Summary of morbidity in the United States Navy and Marine Corps for the quarter ended June 30, 1932*

	Forces afloat	Forces ashore	Marine Corps	Entire Navy
Average strength	70,786	38,966	16,822	109,752
All causes:				
Number of admissions	8,596	5,708	2,444	14,304
Annual rate per 1,000	485.75	585.95	581.14	521.32
Disease only:				
Number of admissions	7,630	5,279	2,237	12,909
Annual rate per 1,000	431.16	541.91	531.92	470.48
Communicable diseases, exclusive of venereal diseases:				
Number of admissions	1,632	1,778	680	3,410
Annual rate per 1,000	92.22	182.52	161.69	124.28
Venereal diseases:				
Number of admissions	2,981	899	593	3,880
Annual rate per 1,000	168.45	92.29	141.01	141.41
Injuries:				
Number of admissions	960	426	163	1,386
Annual rate per 1,000	54.25	43.73	38.76	50.51
Poisoning:				
Number of admissions	6	3	44	9
Annual rate per 1,000	0.34	0.31	10.46	0.33

TABLE No. 2.—*Deaths reported, entire Navy, during the quarter ended June 30, 1932*

		Navy			Marine Corps			
		Officers	Midshipmen	Men	Officers	Men	Nurse Corps	Total
Average strength		9,145	1,826	81,433	1,179	15,660	509	109,752
CAUSE—DISEASE								
Primary	Secondary or contributory							
Abscess, appendical	Embolism, pulmonary			1				1
Abscess, lung	None			1				1
Abscess, subphrenic	Pneumonia, lobar	1						1
Alcoholism, acute	None			2				2
Alcoholism, chronic	Pericarditis, chronic					1		1
Apendicitis, acute	Embolism, cerebral					1		1
Do	Obstruction, intestinal, from spastic or paralytic causes.			1				1
Do	Peritonitis, general, acute			1				1
Do	Septicemia			1				1
Carcinoma, colon	None			2				2
Cellulitis, neck	Abscess, brain			1				1
Embolism, cerebral	None			1				1
Embolism, pulmonary	do			1				1
Encephalitis, lethargic	do			2				2
Endocarditis, subacute	Septicemia	1						1
Influenza	Meningitis, cerebrospinal	1						1
Leukemia	Abscess, ischio-rectal			1				1
Myocarditis, chronic	None	1						1
Do	Dilatation, cardiac, acute			1				1
Do	Pneumonia, broncho						1	1
Pancreatitis, chronic	Cirrhosis, liver	1						1
Pneumonia, broncho	None			1				1
Pneumonia, lobar	do	1		1				2
Pneumothorax	do			1				1
Plague (septicemia)	do			1				1
Pyelonephritis	Myocarditis, acute	1						1
Septic sore throat	Pneumonia, broncho					1		1
Status lymphaticus	None					1		1
Syphilis	Dementia, paralytica					1		1
Do	Thrombosis, coronary artery					1		1
Thrombosis, coronary artery	None					1		1
Do	Gastritis, acute				1			1
Do	Myocarditis	1						1
Tuberculosis, chronic, pulmonary.	None			1		1		2
Tuberculosis, general miliary.	Tuberculosis, meninges			1				1
Tuberculosis, acute, pulmonary, general miliary.	None			1				1
Tumor, malignant, mixed, lymphosarcoma, mediastinum, lung.	None			1				1
Valvular heart disease, mitral insufficiency.	Dilatation, cardiac, acute			1		1		2
Valvular heart disease, mitral stenosis.	Thrombosis, right aurical			1				1
Total for diseases		8		26	1	9	1	45

TABLE No. 2.—*Deaths reported, entire Navy, during the quarter ended June 30, 1932*—Continued

		Navy			Marine Corps			
		Officers	Midshipmen	Men	Officers	Men	Nurse Corps	Total
Average strength		9,145	1,826	81,433	1,179	15,660	509	109,752
CAUSE—INJURIES AND POISONING								
Primary	Secondary or contributory							
Asphyxiation (illuminating gas).	None			1				1
Drowning	do	1		10				11
Fracture, compound, skull	do			8		1		9
Fracture, simple, skull	Hemorrhage, intracranial			1				1
Do	Hemorrhage, subdural			1				1
Do	Intracranial injury			1		1		2
Fracture, simple, spine and pelvis.	None			1				1
Fracture near joint with dislocation, vertebræ, cervical.	do			1				1
Fracture, simple, vertebræ, dorsal.	Pneumonia, broncho					1		1
Injuries, multiple, extreme	None	2		14				16
Intracranial injury	do			1				1
Rupture, traumatic, heart	do			1				1
Rupture, traumatic, rectum	Peritonitis, general, acute			1				1
Strangulation, neck	Dementia præcox			1				1
Wound, gunshot, chest	Hemorrhage, aorta			1		1		2
Wound, gunshot, head, chest, and abdomen.	None					1		1
Wound, gunshot, heart	do					1		1
Wound, gunshot, multiple	do				1	3		4
Wound, lacerated, wrist	Alcoholism, acute	1						1
Poisoning, acute, gasoline fumes.	Pneumonia, broncho					1		1
Poisoning, food, salmonella group, bacterial toxin.	None			1				1
Poisoning, mercury, acute	do			1				1
Total for injuries and poisoning.		4		45	1	10		60
Grand total		12		71	2	19	1	105
Annual death rate per 1,000:								
All causes		5.25		3.49	6.79	4.85	7.86	3.83
Disease only		3.50		1.28	3.39	2.30	7.86	1.64
Drowning		.44		.49				.40
Other injuries		1.31		1.62	3.39	2.30		1.68
Poisoning				.10		.26		.11

ADMISSIONS FOR INJURIES AND POISONING, SECOND QUARTER, 1932

The following table, indicating the frequency of occurrence of accidental injuries and poisoning in the Navy during the second quarter, 1932, is based upon all Form F cards covering admissions in those months which have reached the bureau:

	Admissions, April, May, and June, 1932	Admission rate per 100,000, per annum	Admission rate per 100,000, year, 1931
INJURIES			
Connected with work or drill	639	2,329	2,433
Occurring within command, but not associated with work	378	1,378	1,649
Incurred on leave or liberty or while absent without leave	369	1,345	1,603
All injuries	1,386	5,051	5,685
POISONING			
Industrial poisoning	4	15	17
Occurring within command, but not connected with work	4	15	35
Associated with leave, liberty, or absence without leave	1	4	22
Poisoning, all forms	9	33	74
Total injuries and poisoning	1,395	5,084	5,759

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, 1932	Year, 1931	April, May, and June, 1932	Year, 1931	April, May, and June, 1932	Year, 1931
Per cent of all injuries	46.1	42.8	27.3	29.0	26.6	42.5
Per cent of poisonings	44.4	23.4	44.4	46.8	11.1	29.8
Per cent of total admissions, injury and poisoning titles	46.1	42.5	27.4	29.3	26.5	28.2

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.

The following cases, selected from April, May, and June, 1932, reports, are worthy of notice from the standpoint of accident prevention:

Gasoline hazards.—Familiarity breeds contempt. It is well known that gasoline is a highly explosive and inflammable substance, but the familiarity with which it is handled and the many uses to which it is put to-day, have apparently caused its dangerous qualities to be forgotten and overlooked. A better realization of the need to exercise care and vigilance in using and handling gasoline is plainly a

necessity if life and property are to be properly safeguarded. Some results illustrating the contempt and carelessness with which it is handled are shown in the cases immediately following:

While lighting a galley range the hands, arms, face, and one leg of a seaman, first class, were burned by the ignition of a can of gasoline into which burning gasoline dripped back. Hospitalized 34 days.

A seaman, second class, threw waste gasoline into an incinerator on board ship. The resulting flareback caused a burn of his face and left hand for which he was on the sick list nine days.

Fumes of gasoline from an empty, open, gasoline drum ignited from the lighted cigarette held in the hand of one person, burned the face, lip, and foot of another person. The burned seaman, second class, was on the sick list nine days and the case plainly shows the reason for prohibiting smoking around gasoline and gasoline-drum stowage.

A marine private struck a match while cleaning clothes with gasoline and was burned about his face. While the time lost was but five days, the case is reported to show the possible danger of this practice to life and property.

In addition to gasoline, such substances as cresol, lye, and lye water, are used and handled by naval personnel without even ordinary caution and care. The apparent contempt for the dangers of these familiar substances is shown in the cases given here as examples:

Lye.—A coxswain inspecting the order and cleanliness of a compartment pulled a piece of paper, on which loose lye had carelessly been left, from the top of a locker. As he did so some of the lye fell into one eye causing a chemical burn. Due to the negligence of others he was on the sick list 105 days, 102 of which were in a hospital.

Cresol.—Thinking he was using kerosene, a fireman, third class, washed his arms in cresol to remove paint and was chemically burned. He was on the sick list for 13 days, 7 of which were hospital days, as a result of the negligence of others in failing to stow cresol properly.

Lye water.—Overlooking the probability of the canvas tops of tennis shoes becoming saturated, a fireman, first class, scrubbing decks with lye water while wearing tennis shoes received a chemical burn to one foot for which he was incapacitated 3 days.

STATISTICS RELATIVE TO MENTAL AND PHYSICAL QUALIFICATIONS OF RECRUITS

The following tables were constructed with figures taken from monthly reports submitted by naval training stations:

Cumulative data

	Number	Per cent of recruits received	Per cent of recruits reviewed
JAN. 1 TO DEC. 31, 1931			
All naval training stations:			
Recruits received during the period	7,071		
Recruits appearing before board of medical survey	227	3.21	
Recruits recommended for discharge from the service	122	1.73	53.74
APRIL, MAY, AND JUNE, 1932			
United States naval training station, Hampton Roads, Va.:			
Recruits received during the period	285		
Recruits appearing before board of medical survey	2	0.70	
Recruits recommended for discharge from the service	2	.70	100.00
United States naval training station, Great Lakes, Ill.:			
Recruits received during the period	419		
Recruits appearing before board of medical survey	8	1.91	
Recruits recommended for discharge from the service	8	1.91	100.00
United States naval training station, San Diego, Calif.:			
Recruits received during the period	501		
Recruits appearing before board of medical survey	0	0	
Recruits recommended for discharge from the service	0	0	0
United States naval training station, Newport, R. I.:			
Recruits received during the period	286		
Recruits appearing before board of medical survey	21	7.34	
Recruits recommended for discharge from the service	3	1.05	14.29
Recruits held over pending further observation	18	6.29	85.71

The following table was prepared from reports of medical surveys in which disabilities or diseases causing the surveys were noted as existing prior to enlistment. The time which elapsed from date of enlistment to date of medical survey is noted in each case. With certain diseases, survey followed enlistment so rapidly that it would seem that many might have been eliminated in the recruiting office. The difficulty in establishing a diagnosis in nervous and mental cases is demonstrated by the time interval in the table. An exception in this group is epilepsy which may or may not diagnose itself promptly. Certain groups, of course, present difficulties in diagnosis at the time of enlistment due to lack of equipment:

Cause of survey	Number of surveys	Number of days between enlistment and survey
Bronchitis, chronic	1	32
Bursitis, chronic	1	5
Caries, teeth	1	2
Constitutional psychopathic inferiority, without psychosis	1	126
Do	1	70
Dementia præcox	1	80
Do	1	119
Do	1	55
Enuresis	1	68
Epilepsy	1	38
Flat foot	1	54
Hernia, inguinal	1	8
Hypermetropia	1	9
Malformation, little toe	1	10
Perforated nasal septum	1	4
Tachycardia	1	16
Do	1	8
Do	1	2
Trichophytosis	1	2
Union of fracture, faulty	1	7
Valvular heart disease, combined lesions, aortic and mitral	1	5

VOL. XXXI APRIL, 1933 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
LIEUTENANT COMMANDER JOHN HARPER
MEDICAL CORPS, U. S. NAVY, IN CHARGE

Compiled and published under the authority of Naval Appropriation Act for 1933, Approved June 30, 1932

UNITED STATES
GOVERNMENT PRINTING OFFICE
WASHINGTON : 1933

For sale by the Superintendent of Documents, Washington, D. C. - - - - - See page II for price

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.

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Volume XII, No. 1, January, 1918.
Volume XIII, No. 1, January, 1919.
Volume XIII, No. 4, October, 1919.
Volume XIX, No. 3, September, 1923.
Volume XX, No. 5, May, 1924.

SUBSCRIPTION PRICE OF THE BULLETIN

Subscription should be sent to Superintendent of Documents, Government Printing Office, Washington, D. C.

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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 indorse all views or opinions which may be expressed in the pages of this publication.

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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 shoud 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.

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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. XXXI APRIL, 1933 No. 2

SPECIAL ARTICLES

A SECOND PSYCHOLOGICAL STUDY MADE ON CANDIDATES FOR AVIATION TRAINING

By C. G. DEFONEY, Lieutenant Commander, Medical Corps, United States Navy

In May, 1932, the writer submitted to the Bureau of Medicine and Surgery a report of the findings obtained on a group of 677 individuals who were given psychological ratings for aviation adaptability upon their reporting at the naval air station, Pensacola, Fla., for flight training. This report was a supplement to a previous study made on a group of 628 individuals along the same lines, reported upon in August, 1930, and published in volume 29, No. 2, of the U. S. Naval Medical Bulletin of April, 1931.

The present article contains the essence of the second report, also figures showing the results obtained on both groups studied.

The second report was made upon the writers detachment from duty at the naval air station, Pensacola, Fla., and the study at that time embraced a total of 1,741 individuals. However, only 1,305 had reached the stage where they had proved or disproven the original psychological prediction made before the individuals began flight training.

As in the first group studied, a prediction was made as to the student's ultimate success or failure to qualify as an aviator. The student then proceeded with his training and the outcome was checked against the original prediction. Capt. D. G. Sutton, Medical Corps, United States Navy, and the writer were designated to carry on this study, which was begun in September, 1928. The present group of 677 individuals was examined and studied by the writer.

As stated in the report made on the first group studied, the examiners were able to determine the good and bad types after association and study with all classes of student aviators under stress of training. The theory formulated at that time, that instability of any kind was indicative of poor material, was adhered to

throughout both groups studied. In other words, the detection of the neurotic individual was considered to be the most important function of the psychological examination. For the benefit of those who did not see the first report, it might be well to state that the examiners decided that the factors of stability, aggressiveness, and courage in the individual were the outstanding components that made for success. It was found that the factors of intelligence and concentration apparently had no influence upon an individual's ability to qualify or fail to qualify as an aviator. Reaction time as measured by the domino test was found to be of no value, although the manner in which the test was performed was of decided benefit to the examiner.

The system of grading used was devised solely for the examiners' information. The grade of 2.5 was given to the average type, 2.6 to the slightly above average, 2.7 and above was given to increasingly better types. The grade 2.4 was given to slightly below average, 2.3 and below to poorer types.

From the fact that most students before being sent to Pensacola are selected as adapted for aviation, it will follow that the largest number of individuals will fall in the groups graded 2.3, 2.4, 2.5, and 2.6. Those graded 2.4 were considered doubtful.

A total number of 163 students were given a grade of 2.5 and 2.6. Eighty-four and seven-tenths per cent of these qualified as aviators; 25, or 15.3 per cent, failed to qualify. Of those who qualified, 45 individuals, or 32.6 per cent, required an average of 3.4 hours extra instruction during the course. In this group one man had part of the course at a previous time.

A total number of 289 students were given a grade of 2.3 and 2.4. Of these, 104 individuals qualified as aviators, making a 35.3 per cent error. Fifty-five individuals of this group required an average of 3.76 hours extra instruction during the course, 13 men had part of the course at some previous time, and 3 men had two elimination courses, which meant that extra instruction had been given them. Of the remaining 225 individuals, 115 were graded 2.2 and below. Eighty-nine of these, or 79 per cent, failed to qualify as aviators or pilots, and only one qualified as a combat pilot. One hundred and ten were graded 2.7 and above. Of these, 94 individuals, or 85 per cent, qualified as aviators or pilots, and 38 of these qualified in combat flying.

In a group of 190 individuals given combat training (128 officers and 62 reserves) graded 2.5 and above, 61 individuals, or 34 per cent, qualified as combat pilots. In a group of 117 individuals (93 officers and 24 reserves) graded 2.4 and below, only 16 individuals, or 13.6

per cent, qualified as combat pilots. This seems to bear out the belief of the examiners that the better types as determined by psychological methods would qualify more individuals in that type of flying which requires better than average adaptibility for aviation. In this connection it was interesting to note the relation between the original psychological marks given and the percentage of individuals qualifying in combat flying. The following figures show the original psychological ratings given and the percentages in each group which qualified in combat flying:

Original psychological marks given:	Per cent qualifying in combat flying
2.8 and above	100
2.7	85
2.5 and 2.6	34
2.3 and 2.4	13.4
2.2 and below	00

Two hundred and seventy-eight individuals graded 2.4 and below who failed to complete the course at Pensacola spent a total time of 98 years 2 months and 1 day there before they were found by actual training to be poor aviation material. These figures reveal that the average time taken to discover by actual training that an individual is not adapted for aviation is four months and seven days. It can readily be seen that considerable time and money could be saved if elimination by psychological examination was practiced before the students reported at Pensacola.

The writer had the opportunity, when the last three classes examined by him reported for training at Pensacola, to check his psychological opinion against that formed by two other examiners. These examiners were made familiar with the method used here during a short course given them at Pensacola. One examined students at Hampton Roads, Va., and the other at San Diego, Calif., where the elimination courses are given. The psychological estimates check very accurately in all instances, although the examiner had no previous information as to the original psychological opinion formed. This seems to be fairly conclusive. It demonstrated that several examiners familiar with the method used could arrive at approximately the same results independently.

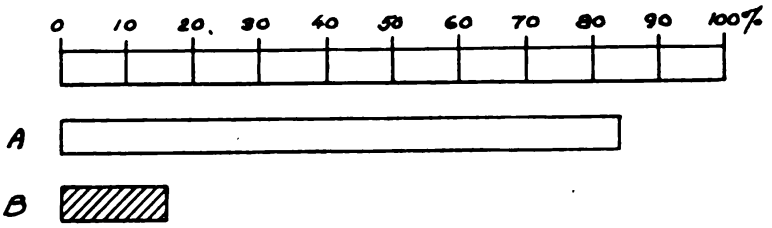
The following charts constitute the expression of psychological opinions compared with the actual results of training.

Figure 1 represents a group of 267 officers and men selected by psychological examination, from a total of 677 individuals, as being good aviation material. Since only 16 per cent of this group failed to qualify, and the predictions were 84 per cent correct, it is evident

that the results obtained were satisfactory. In this, the second group studied, an improvement of 14 per cent in accuracy over the first like group studied is noted.

Figure 2 represents a group of 410 officers and men selected by psychological examination, from a total of 677 individuals, as being poor aviation material. There was an error of 32.5 per cent in this group. This is explained in part by the fact that in the group graded 2.4 and considered a border line group, the greatest per cent of errors occur. While it is considered by the examiners that this

GROUP OF 267 OFFICERS & MEN SELECTED BY PSYCHOLOGICAL EXAMINATION FROM A TOTAL OF 677 INDIVIDUALS AS BEING GOOD AVIATION MATERIAL.



A – 224 individuals of this group (84%) qualified as Aviators or Pilots.

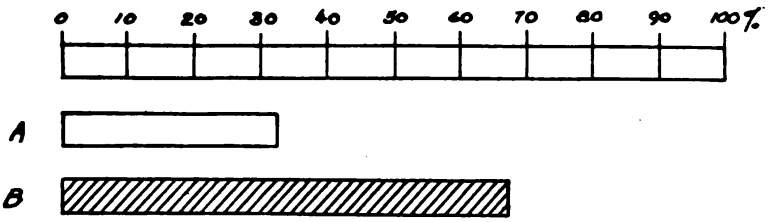
B – 43 individuals of this group (16%) failed to qualify.

FIGURE 1

group was not quite up to the standard demanded of average students, it was expected that some of them would qualify as aviators. A higher percentage of correct predictions would have obtained in those adjudged poor material had not, as the examiner believes, the standards for qualification been lowered at one period during the study. It is the opinion of the examiner that this change in requirements was made in order to meet an urgent demand for more aviators at that time. In consequence, many men were qualified who were not up to the usual standards, as evidenced by the fact that unofficial complaints of inefficiency were made by those in charge of the operating units to which some of these men were sent.

Figure 3 is an analysis of crashes on 640 individuals who have qualified as aviators or pilots during this study. Covering a period

GROUP OF 410 OFFICERS & MEN SELECTED BY PSYCHOLOGICAL EXAMINATION FROM A TOTAL OF 677 INDIVIDUALS AS BEING POOR AVIATION MATERIAL.

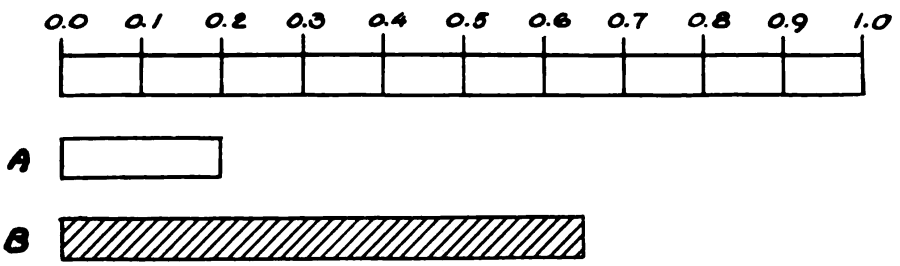


A – 133 individuals of this group (32.5%) qualified as Aviators or Pilots.

B – 277 individuals of this group (67.5%) failed to qualify.

FIGURE 2

ANALYSIS OF CRASHES.



A – 372 Aviators or Pilots who were determined, by psychological examination before starting training, to be good aviation material had a crash rate of 0.2 during the years 1929, 1930 and 1931.

B – During the same time, 268 Aviators and Pilots who were determined, by psychological examination before starting training, to be poor aviation material had a crash rate of 0.65.

FIGURE 3

of almost 4 years, 372 persons, who were adjudged good material by psychological examination, have had in the years 1929, 1930, 1931, 82 crashes due to the fault of the pilot, showing a crash rate of two tenths. In the same length of time 268 aviators or pilots, adjudgd poor material by psychological examination before commencing training, have had 116 crashes due to the fault of the pilot, showing a crash rate of sixty-five hundredths, or more than three times the rate of those in the group determined to be good material by psychological examination.

Figure 4 shows the results of actual training in the second group of 677 individuals studied, as compared with the results had the psychological method of selection been accepted.

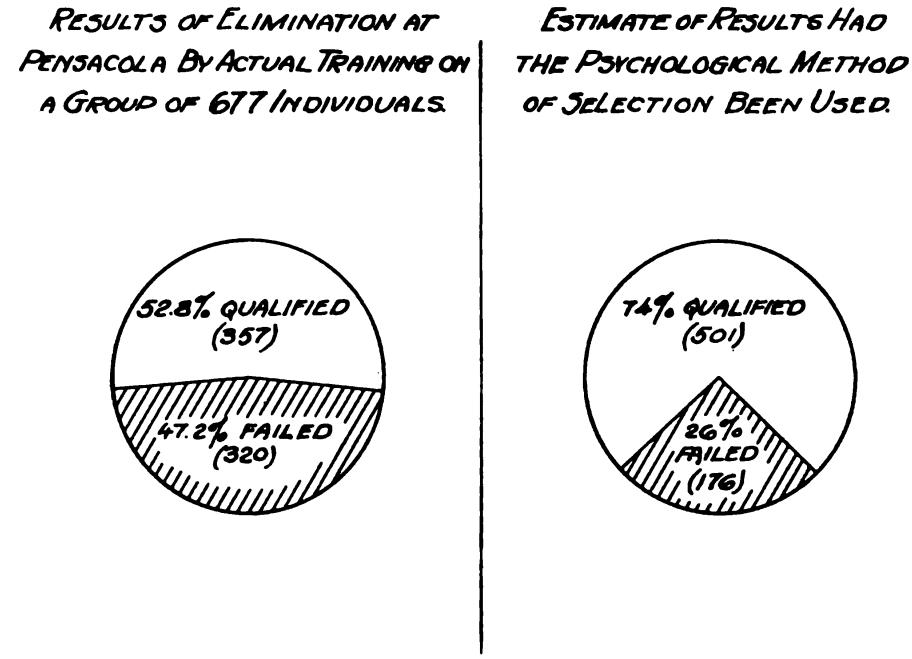


FIGURE 4

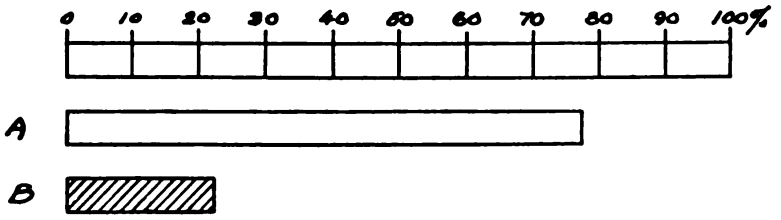
Figures 5 and 6 show the results obtained in the entire number of individuals reported upon.

Figure 5 shows the results as obtained by actual training with 479 officers and men who were selected as being good aviation material by psychological examination from a total of 1,305 individuals. This includes both groups studied.

Figure 6 shows the results obtained in a group of 826 officers and men selected as being poor aviation material by psychological methods from a total of 1,305 individuals.

Figure 7 shows the results of actual training at Pensacola on a group of 1,305 individuals, as compared to the estimate of results had the psychological method of selection been used.

GROUP OF 479 OFFICERS & MEN SELECTED BY PSYCHOLOGICAL EXAMINATION FROM A TOTAL OF 1305 INDIVIDUALS AS BEING GOOD AVIATION MATERIAL.

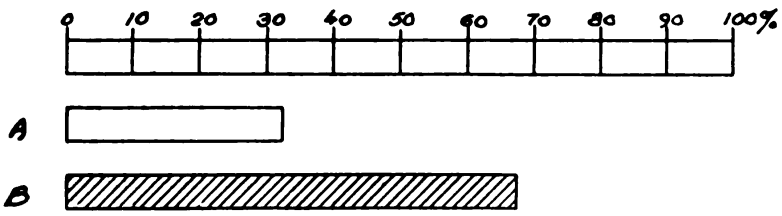


A — 372 individuals of this group (77.6%) qualified as Aviators or Pilots.

B — 107 individuals of this group (22.4%) failed to qualify.

FIGURE 5

GROUP OF 826 OFFICERS & MEN SELECTED BY PSYCHOLOGICAL EXAMINATION FROM A TOTAL OF 1305 INDIVIDUALS AS BEING POOR AVIATION MATERIAL.



A — 268 individuals of this group (32.5%) qualified as Aviators or Pilots

B — 558 individuals of this group (67.5%) failed to qualify.

FIGURE 6

CONCLUSIONS

(*a*) That the psychological method of elimination should be used as a routine measure for the elimination of poor aviation material. This belief is fortified by the fact that a large percentage of those individuals who qualify as aviators or pilots, although the psychological examination has determined them to be poor material, require extra instruction in order to complete the course, and a very small percentage qualify as combat pilots. These individuals also have three times the crash rate of those determined to have been good material, and quite a few have been dropped from aviation as being

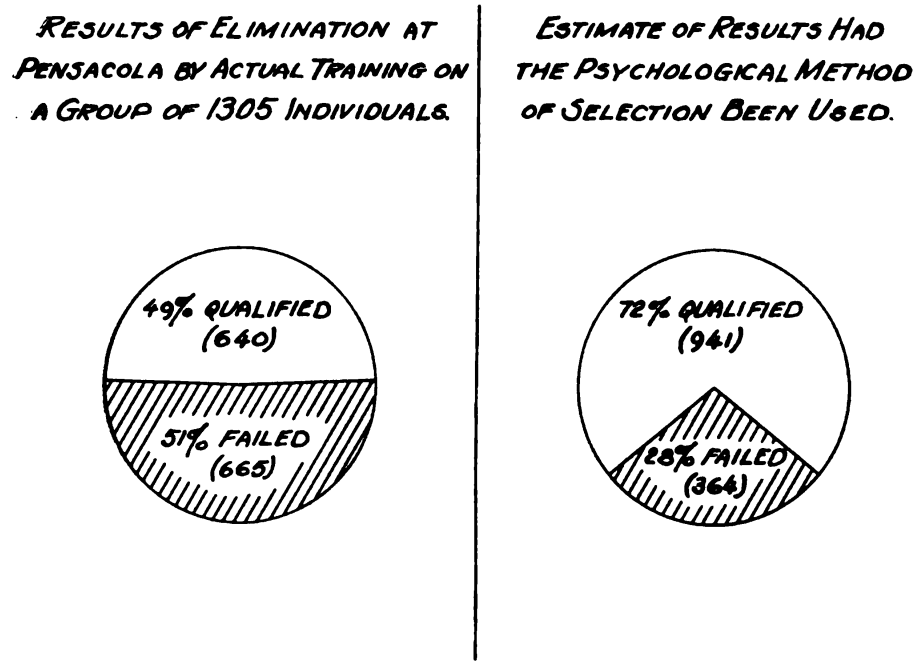


FIGURE 7

inapt or dangerous, some have been killed, some caused the death of other persons when they crashed.

(*b*) Only those individuals, however, who are definitely below the average standard should be eliminated by this method, and then only by trained and experienced observers.

(*c*) Stability is still considered the necessary trait for the individual to possess in order to be considered good aviation material. The psychological examination form was constructed with this belief in mind, and the examiners were 72 per cent correct in their predictions as to the success or failure in a group of 1,305 individuals.

(*d*) The great amount of time wasted by sending poor material to Pensacola for training would be saved by eliminating this type at

the source when applying for training. This would result in a tremendous saving in man power, material, and money.

(*e*) While the data are not complete owing to the fact that many of these officers are still under instruction, it is the opinion of the examiner that there are more early failures among those officers who are sent to Pensacola shortly after graduating from the Naval Academy than there are among those who have had several years experience after graduation. The examiner also believes that at this time the course of instruction has been standardized to a greater degree than has obtained in the past.

(*f*) It is believed that the early elimination of the poor material is to be desired and the practice of giving extra instruction in primary seaplanes to individuals who are not considered to be well adapted should be discontinued, as a large percentage of those so favored fail to qualify in primary landplanes and the few who are designated naval aviators fail to make good in operating units.

Recommendations

(1) It is recommended that the psychological study be continued and the method further perfected.

(2) It is recommended that elimination of the very poor material be made at the source, or at least very early in training at Pensacola, when the student's instructors agree with the psychological opinion. It has been shown in the foregoing report that, while some of this type qualify as aviators or pilots, they do not measure up to the standards expected.

(3) If the practice is instituted of eliminating poor material at the source, more medical officers should be trained in this work.

(4) A system of grading should be devised which would show to the casual observer a greater variation in the marks of those graded good and those graded poor material. It shoud conform to the standard Navy system of marking.

The writer acknowledges the kind assistance and suggestions of Lieut. Commander R. H. White, Medical Corps, United States Navy, in the compilation of the above data.

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A STUDY OF THE CONVULSIVE SEIZURES CAUSED BY BREATHING OXYGEN AT HIGH PRESSURES[1]

By C. W. SHILLING, Lieutenant (Junior Grade), Medical Corps, United States Navy, and B. H. ADAMS, Lieutenant Commander, Medical Corps, United States Navy

INTRODUCTION

The individual escape apparatus or "lung" provided for submarines in the United States Navy consists of a rubberized cloth bag which is filled with oxygen and contains a soda lime canister to absorb carbon dioxide. The bag is fastened across the chest. The subject breathes in and out of it through a rubber mouthpiece such as is used with metabolism apparatus and wears a nose clip.

In escaping from a submarine the compartment in which the men have collected is flooded until the pressure inside and outside is equal. The hatch is then opened, and with escape apparatus adjusted, each man goes through the hatch and up a line to the surface. It is necessary to breathe high concentrations of oxygen at the pressure provided by the depth reached by the submarine. That high tensions of oxygen are dangerous is well known. Both the lungs and nervous system may be affected. The possibilities of pulmonary damage as a result of prolonged exposure to relatively low concentrations of oxygen have been discussed in papers from this laboratory by F. J. C. Smith and his associates (1) (2). The data they presented do not give information as to the tensions of oxygen which cause convulsions nor as to whether lung damage is also found in animals subjected to these acute effects of oxygen.

The necessity for clearing up these points will be quite clear when one realizes that if a submarine lies on the bottom at 200 feet, the oxygen in the escape apparatus will be breathed at a tension of approximately seven atmospheres absolute. What may one expect from such a situation?

Paul Bert (3) found that small birds had convulsions when exposed to three atmospheres absolute of oxygen, and that as the pressure increased the incidence and severity of the convulsions increased and death rapidly ended the picture. The convulsions came on within 5 minutes when the pressure was 3.5 atmospheres of oxygen, and at 4.5 atmospheres death came after 20 minutes in some instances. A few mice and rats were exposed to various pressures, but no convulsions were noted until a pressure of four atmospheres of oxygen was reached. Dogs showed convulsions at 3.8 atmospheres of oxygen but they were breathing from a bag of oxygen and no provision was made for the removal of the carbon dioxide which in most cases rose

[1] From the department of physiology, Harvard School of Public Health. This investigation has received aid from the Miriam Smith Rand Fund of Harvard University.

to a very high concentration and thus distorted the results. He noted marked tetanic convulsions upon rapid decompression from high oxygen pressures when similar decompression from air caused no convulsions. Lung damage was not considered in its relation to oxygen poisoning.

Using 31 mice and 14 small birds, J. L. Smith, (4) studied convulsions and lung damage caused by high oxygen tensions. He found that oxygen tensions of 1.8 atmospheres caused death in mice in 23 hours and with 3.5 atmospheres death came in 5 hours, but that no convulsions occurred in mice until the oxygen tension reached 4.5 atmospheres absolute. Two larks showed convulsions under an oxygen tension of three atmospheres. He studied the lung pathology and found that a pressure of 40 per cent of an atmosphere of oxygen caused no harm in 8 days, 80 per cent killed two mice in 4 days, 125 per cent killed mice in an average time of 64 hours, 180 per cent in 24 hours, and 300 per cent produced inflammation of the lungs in 5 hours. No comparative study was made of lung damage and convulsions, but he states that if the oxygen tension is slowly increased the lung congestion prevents the quick rise of oxygen tension in the blood and so convulsions fail to appear.

Hill (5) says that convulsions seem never to occur when the oxygen tension is below 300 per cent, i. e., three atmospheres absolute, or above 600 per cent, i. e., six atmospheres absolute. Above six atmospheres absolute the animals go to death without convulsions until 60 to 70 atmospheres of oxygen are reached, when they develop instantaneous convulsions, followed immediately by death. He stated: "We may assume that with pressures below 300 per cent atmosphere oxygen the amount of gas in solution is not sufficient to excite; that with pressures above 600 per cent atmospheres oxygen the inflammation of the lung causes the collapse of the animal."

Bornstein and Stroink (6) demonstrated on themselves that breathing pure oxygen under a pressure of three atmospheres absolute for 48 minutes was without any ill effects. Bornstein, however, in a test on himself, found that pedaling a bicycle under the above conditions caused cramps in the legs in 51 minutes, which he attributed to the increased tension of oxygen.

Technique

In the present study over 200 standard white rats were used to determine the oxygen tension at which convulsions developed, the time of exposure necessary at each pressure, and the tissue damage caused by these oxygen tensions. The results of this work with rats were confirmed by the findings of similar studies with guinea pigs, rabbits, and cats.

One of the mail locks of a pressure chamber described by Thomson, Yaglou, and Van Woert (7) was used in this work, the lock being fitted with glass ports to permit observation of the animals. The oxygen pressure in the lock was controlled by admitting oxygen from a high-pressure cylinder up to the desired gauge pressure. By permitting commercially pure oxygen to flow through the lock and apparatus until 40 cubic feet had been used an atmosphere of plus 98 per cent was obtained, and by allowing a small continuous flow throughout the experiment the concentration of carbon dioxide was kept below 1 per cent at all times, while the concentration of oxygen was held at 98 per cent.

The animals not succumbing during the experiment were killed by an intraperitoneal injection of a solution of nembutal and used in the study of the pathological changes produced by high-tension oxygen.

Discussion of the Experimental Data

The time of exposure to oxygen was limited to two hours throughout the experiments. It will be observed from inspection of Table 1 that no convulsions occurred within this period of time until the gauge pressure of oxygen (pressure in excess of one atmosphere) had reached 40 pounds or 3.6 atmospheres absolute. At gauge pressures in excess of 40 pounds there was a progressive increase in the per cent of animals developing convulsions, as is clearly shown in Figure 1, curve A–H, until at 55 pounds gauge or 4.6 atmospheres and above all animals developed convulsions within the 2-hour period. From an inspection of Figure 1, curve 1–6, it is also apparent that with increasing oxygen pressures the convulsion was in general induced by progressively shorter exposures, although there was a marked individual variation in the time of onset, as shown by Table 1.

Table 1.—*Time of development of first convulsion in rats exposed to pure oxygen for two hours at varying gauge pressures*

Exposure time intervals	Animals affected							
	Pressure 15 pounds	Pressure 30 pounds	Pressure 35 pounds	Pressure 40 pounds	Pressure 45 pounds	Pressure 50 pounds	Pressure 55 pounds	Pressure 60 pounds
0–10 minutes							3	8
10–20 minutes				1		5	2	5
20–30 minutes					3	2	5	3
30–40 minutes					1		1	2
40–50 minutes					1			2
50–60 minutes						2	1	1
60–70 minutes						1	3	2
70–80 minutes								
80–90 minutes						1	1	
90–100 minutes				1	2		2	
100–110 minutes				1		1		
110–120 minutes				1	2	2		
Total affected	0	0	0	4	9	14	18	23
Total exposed	22	13	18	18	18	18	18	23
Per cent affected	0	0	0	22	50	78	100	100

There were variations not only in the time of onset but also in the type of the convulsions observed in these experiments. If the pressure was high—that is, 55 to 60 pounds—certain animals would show a typical severe clonic seizure characterized by spasms of extensor rigidity within the first 10 minutes and without any of the usual prodromal signs. Another animal might exhibit a wild convulsion in which without warning it would throw itself violently about the cage for as long as 10 minutes without stopping.

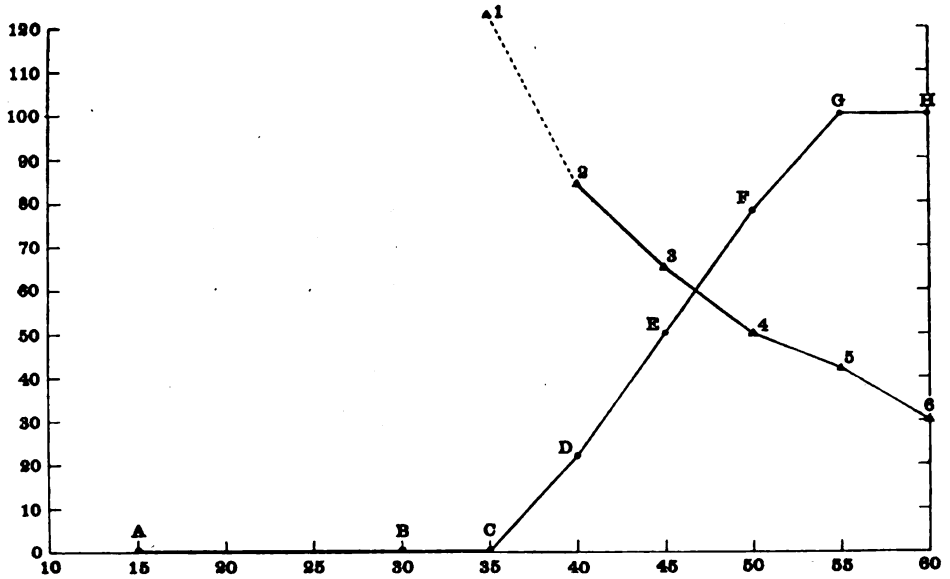

FIGURE 1.—Curve A-H, per cent of rats developing convulsions during a two-hour exposure to varying oxygen tensions; curve 1–6, average time of development of first convulsion in rats exposed for two hours to varying oxygen tensions. Ordinates, per cent and time in minutes; abscissae, pounds pressure gauge of oxygen. A, 22 rats exposed to this pressure with none developing convulsions; B, 13 rats; C, 18 rats; D, 18 rats; E, 18 rats; F, 18 rats; G, 18 rats; H, 23 rats. *1*, probable average time of onset of convulsions if the experiment had been continued over two hours; *2*, average time of first convulsion for the 4 rats developing convulsions out of the 22 exposed; *3*, 9 rats; *4*, 14 rats; *5*, 18 rats; *6*, 23 rats. (Shilling and Adams.)

The most common condition is best described by taking a representative case and following it through an entire experiment. In experiment No. 56 a rat exposed to 50 pounds gauge oxygen pressure began to exhibit dyspnea 9 minutes after full pressure had been reached. This continued in a moderate way for 22 minutes, when it subsided and the rat was quiet for 18 minutes. It then became restless and again experienced dyspnea, which increased in severity until every inspiration was accompanied by a gasp and marked bodily effort. Seven minutes later, or 47 minutes after the start of the experiment, convulsive movements began with twitching and vigorous shaking of the body. This shaking of the entire body is often quite violent and is usually followed by the typical clonic convulsion.

These movements passed over into a mild convulsion after a total of 62 minutes exposure.

The actual seizure was characterized by rapid spasmodic extensor movements of the head associated with rapid flexion and extension of the forelegs. This attack lasted half a minute, and was followed by a second and more severe convulsion in three minutes, which was characterized by spasms of extensor rigidity of the entire body. Another occurred two minutes later, and then convulsions were practically continuous until the animal died after a total exposure of 95 minutes.

As will be noted in Table 2, the results obtained with guinea pigs conformed very closely to those obtained with rats. With these animals, however, in contradistinction to rats, we were able to count the respirations and found that in almost every case the rate was increased during the early minutes of exposure to increased oxygen tensions, but within 15 minutes it gradually became progressively slower until it reached 36 to 20 per minute, at which time convulsions developed. The dyspnea during the time just prior to the convulsion was quite marked in all cases.

TABLE 2.—*Development of convulsions in guinea pigs, rabbits, and cats*

Number of experiment	Animals used—type and number	Oxygen tension used (pounds gauge)	Length of exposure (minutes)	Number developing convulsions	Time of first convulsion in each animal (minutes)	Time of death of each animal (minutes)
40	Guinea pigs, 2	35	120	0		
39	do	40	120	0		
41	do	40	120	0		
42	do	40	120	1	105	
67	Guinea pigs, 3	45	120	1	118	126, 128
68	do	50	105	3	94, 96, 101	99, 100, 104
69	Guinea pigs, 2	55	120	2	116, 118	121
70	do	60	107	2	89, 106	
38	Rabbits, 2	35	120	0		
71	Rabbit, 1	40	109	1	27	108
72	do	45	88	1	14	87
73	do	50	120	1	89	120
74	do	55	28	1	9	27
43	Cat, 1	40	120			
44	do	40	120			
79	do	40	120			
45	do	45	120			
75	do	45	120			
78	do	45	58	1	29	
46	do	50	33	1	24	
76	Cats, 2	50	112	2	47, 48	115, 117
77	Cat, 1	55	75	1	54	
80	do	60	28	1	15	

The work with rabbits served to confirm the results obtained with rats and guinea pigs, but the rabbits were slightly more susceptible than either of the latter, although they did not show convulsions until the gauge pressure of 40 pounds oxygen was reached. Cats in general showed a greater resistance than the other species of animals, none of our cats having convulsions until 45 pounds oxygen pressure

was reached. Otherwise they reacted similarly, the convulsions in these animals being the same in type as those noted with the rats.

Except for the lungs, no pathological effects were noted grossly in any organ or tissue other than mild congestion. The lungs, however, showed the following very definite gross pathological effects: Congestion alone, congestion and edema, severe congestion and edema with areas of hemorrhagic exudation, and almost solid hemorrhage, causing the lungs to sink in water. The number and severity of these gross signs varied with the individual susceptibility, the length of exposure, the pressure of oxygen used, and the number of convulsions the animal experienced. These pathological changes are clearly brought out in Table 3, which records the time of exposure, pressure of oxygen, number of convulsions, time of onset of the first convulsion, and the pathological findings.

TABLE 3.—*Pathological findings in rats exposed to high oxygen tensions*

Number of experiment	Oxygen pressure (gauge pounds)	Designating mark on animal	Time of exposure (minutes)	Time of first convulsion (minutes)	Number of convulsions	Time of death (minutes)	Gross pathology				Microscopic pathology		
							Fluid in pleural cavity	Edema of lung	Congestion of lung	Hemorrhage in lung tissue	Hyperemia	Exudate	Hemorrhage
1	15	B	120	----	----	----	----	----	----	----	+	----	----
8	15	A	120	----	----	----	----	----	+	----	+	----	----
8	15	B	120	----	----	----	----	----	++	----	+	----	----
11	15	B	120	----	----	----	----	----	+	----	+	----	----
2	30	A	120	----	----	----	----	----	+	----	+	+	----
2	30	B	120	----	----	----	----	----	+	----	+	+	----
7	30	White	120	----	----	----	----	----	++	----	+	----	----
12	30	Red	120	----	----	----	----	----	+	----	+	----	----
19	35	White	120	----	----	----	----	----	+++	----	+	+	----
32	35	White	120	----	----	----	----	----	+	----	+	----	----
32	35	Blue tail	120	----	----	----	----	----	+	----	+	----	----
34	35	Red	120	----	----	----	----	----	+	----	+	+	----
14	40	White	120	----	----	----	----	----	+	----	++	+	----
33	40	Blue	120	17	1	----	----	----	+	----	++	----	----
33	40	Brown	120	----	----	----	----	----	+	----	+	----	----
33	40	Red	120	----	----	----	----	----	+	----	+	----	----
13	45	Blue	120	27	1	----	----	----	++	----	+	----	----
13	45	Yellow	120	38	1	----	----	----	+++	----	++	+	+
50	45	Red head	120	119	1	----	----	+++	+++	+	+++	++	+
50	45	Blue head	120	----	----	----	----	+	+	----	++	+	----
49	50	Yellow tail	120	16	Many.	66	+	+++	+++	++	+++	+++	++
55	50	Yellow head	62	27	Many.	56	++	++++	++++	++++	++++	+++	+++
55	50	Blue tail	120	59	Many.	----	----	+++	+++	++	++	+++	++
56	50	Red head	120	----	----	----	----	----	++	----	+	+	----
56	50	Blue tail	120	62	Many.	95	+	++++	++++	+++	+++	++++	+++
52	55	Blue head	36	9	Many.	----	----	+	+	----	+	+	----
52	55	Blue tail	36	10	Many.	30	----	++	++	----	++	++	+
53	55	Red tail	101	98	2	----	++	+++	+++	+	++	++	+
54	55	Blue tail	101	94	1	----	----	++	++	+	+	+	+
53	55	Blue head	101	28	Many.	86	----	++++	++++	+++	+++	+++	++
4	60	White	42	34	Many.	54	----	++	++	++	+++	++	+++
NI	60	Blue	20	14	1	----	----	++	++	----	++	+	+
NI	60	White	20	17	1	----	----	++	++	----	++	+	+
24	60	Red tail	81	60	1	----	----	++	+++	+	++	+	+

Binger, Faulkner, and Moore (8) described these lesions as hemorrhagic edema. Adams (9) referred to the lung changes as irritative pneumonia. J. L. Smith (4) considered the pulmonary picture

as a slowly developing inflammatory effect. Hill (5, p. 136) stated: "High partial pressure of oxygen exercises a marked irritant effect on the lungs, producing at first congestion of the alveolar capillaries, and afterwards hemorrhagic exudation and consolidation. To the naked eye the lungs present in the early stages a suffused redness. Patches of more intense exudation occur in the apices and edges of the lungs. At a later stage the congestion passes into typical hepatization, the lungs sink in water, and are of a dark purple color The pneumonia is patchy if quickly and universal if slowly developed."

Microscopically the pathological picture in general conformed to that indicated by the gross examination. Varying degrees of such acute conditions as hyperemia, transudation, and acute hemorrhage into the alveoli and tissue spaces were found.

Extensive microscopic studies of the brain and nervous tissue of animals subjected to oxygen convulsions have been made by Finley and will be reported in another paper. The findings were essentially negative.

The possible relation between lung damage and convulsions is of prime importance. It was apparent from the results of this study that lung damage was not the cause of the convulsions; that is, the convulsion was not due to asphyxia induced by the functional loss of lung tissue. There was practically no gross lung damage in animals autopsied immediately after the first convulsion occuring early during exposure to high pressures of oxygen. In several instances animals went on to death under high-pressure oxygen with lungs that were not sufficiently damaged to account for convulsions due to asphyxia. This was especially true of the larger animals with death occurring during exposure, and yet at autopsy congestion only being found or an occasional area of hemorrhage. If the convulsions were directly induced by lung damage, the seizures should be progressive and recurring, but in numerous cases the animal had one early convulsion and no more during the entire experiment.

On the other hand, there appears to be no evidence that the convulsions due to high pressures of oxygen in themselves cause lung damage. Rats with fatal convulsions produced by strychnine show no gross lung changes. Rats given an anesthetic (nembutal or veronal) and exposed to high-pressure oxygen do not develop convulsions, yet the lung damage is similar to that in unanesthetized animals with convulsions.

In spite of the evidence that there is no causative relation between lung damage and convulsions, lung damage was found in the majority of the animals that developed convulsions. At the end of a 2-hour exposure the lungs of animals that had died from the con-

vulsive experience were almost solid from hemorrhage. Animals with numerous convulsions but surviving the experience showed, when sacrificed, severe congestion and edema with scattered areas of hemorrhage. Animals that had but one or two convulsions showed congestion and edema alone, and those that had gone through the experiment without any convulsions showed little change in the lungs. This is clearly demonstrated in Table 3.

Bert (3) did not attempt to link lung damage with the convulsions, which he designated as status epilepticus, but concluded that oxygen under high tension was a direct nerve poison and in that manner killed the animal. J. L. Smith (4) considered that the cause of death was lung damage, and referred to the rapidly developing effect on the nervous tissue as functional. Hill (5) states that early lung damage will prevent the quick rise of oxygen tension in the blood and, therefore, convulsions fail to appear.

From the present study it is clear that the convulsions and pulmonary damage are separate, unrelated phenomena, both probably caused by the high tension of oxygen but acting in a different manner. Sometimes the convulsions occur without lung damage, and sometimes lung damage is caused without convulsions, but in the majority of the animals both appear and usually coincidently.

Discussion

We do not know at present how closely man corresponds to animals in susceptibility to oxygen poisoning, but the results of these experiments serve at least as an indication of the threshold of danger. We can say with reasonable certainty that pure oxygen at a gauge pressure of 40 pounds, or 3.6 atmospheres absolute or over, is likely to cause convulsions if breathed for over an hour and is almost certain to cause some lung damage. As the oxygen pressure increases the danger of both convulsions and lung damage is increased, and the safe time limit of exposure is lessened until at 60 pounds gauge pressure of oxygen, or 5 atmospheres absolute, it is almost certain that most men would suffer severe lung damage and convulsions within the first hour of exposure.

It is unlikely that a man wearing the submarine "lung" would be breathing over 70 to 80 per cent oxygen unless he had filled the "lung" more than once while breathing into it. In spite of this, were the submarine at a depth of 200 feet, the pressure of oxygen in the "lung" would be approximately 5.25 atmospheres absolute, or 64 pounds gauge, and grave danger would be encountered in breathing this tension of oxygen even for so short a time as required for an escape from the submarine, particularly if the "lung" is donned some time prior to the actual escape.

From our observations on animals, however, we are of the opinion that men exposed to dangerous tensions of oxygen will have early and ample warning of impending lung damage and convulsive seizures. In all probability there will be early irritation of the nasal and pharyngeal mucous membrane preceding any lung damage and nervous-system manifestations such as general restlessness, irritability, and muscular twitching prior to the actual convulsive seizure. As has already been mentioned, Bornstein and Stroink (6) reported cramps in the legs, but as they were pedalling a bicycle this should not be considered as an early warning sign. Even after a severe convulsion, recovery would probably be rapid and complete if the exposure were immediately terminated.

Further experiments are in progress at this laboratory to determine the cause of both the lung damage and the convulsion. This work will be reported at a later date.

Summary

(*a*) In these experiments we have determined the acute effects of high oxygen pressure on rats, guinea pigs, rabbits, and cats with special reference to the production of convulsions and lung damage.

(*b*) Data are presented showing that no convulsions develop within a 2-hour exposure to oxygen pressures of 35 pounds gauge or below, but as the pressure is increased the number of animals developing convulsions within the 2-hour period increases until all species exposed to 55 or 60 pounds gauge develop convulsions. As the pressures increase the time of exposure required to produce convulsions becomes less.

(*c*) Lung damage which varied from mild congestion to severe hemorrhagic edema and acute hemorrhage was found to be associated with the convulsive seizures, but the two were not interrelated; that is, one might have severe changes in the lungs and no convulsions and vice versa.

(*d*) The experiments constitute a warning against the uncontrolled use of pure oxygen for rapid decompression of divers and in the submarine "lung" when escaping from great depths.

ACKNOWLEDGMENTS

The writers wish to express their thanks and appreciation to Prof. Cecil K. Drinker, to whom we are indebted for the outline of these investigations and for his suggestions and criticisms which made this study possible. We also wish to express our thanks to Lieut. Commander I. B. Polak, Medical Corps, United States Navy, for his assistance in the early experiments, and to Mr. Louis A. Shaw and

Mr. Robert M. Thomson and others of the Harvard School of Public Health for timely suggestions and technical assistance in the performance of the experiments.

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THE TREND IN ANESTHESIA AND ITS RELATION TO NAVAL MEDICAL DEPARTMENT PERSONNEL REQUIREMENTS

By K. C. Melhorn, Captain, Medical Corps, United States Navy

To those whose endeavor it is to maintain balanced staffs in naval Medical Department units the task of supplying personnel qualified to administer inhalation anesthetics has become one of increasing difficulty. That there has been widespread supplanting of general anesthetics by local and spinal agents is known to all. While it is equally well recognized that inhalation anesthesia is still the method of choice for certain types of patients and conditions, it is evident from requests and reports received by the bureau that the opportunities for training personnel in this type of anesthesia are not being taken advantage of in enough instances to meet current requirements of the Navy as a whole. In an effort to secure the opinion of the service at large on this matter an inquiry was circulated recently among district and force medical officers. Exceedingly in-

teresting comments and recommendations having been received, it is the purpose of this article to give the service the benefit of the survey in the words of the reports themselves. Accordingly the inquiry and replies are listed as follows, names and locations being omitted:

THE INQUIRY

To: * * *

Subject: Instruction in methods of administering general anesthetics.

1. In an endeavor to meet the needs of certain Medical Department units with respect to supplying personnel trained in the administration of general anesthetics the bureau is becoming more and more handicapped. Due in great measure to the increasing employment of local and spinal anesthesia, practical instruction of medical officers, selected nurses, and Hospital Corps men in methods of administering general anesthetics is rapidly diminishing. As an instance of the present trend, recent study of data available in the bureau reveals that at one of the major hospitals of the service in 2,866 operations general anesthesia was employed but 21 times. It is obvious that unless the existing state of subject instruction is improved not only will present difficulties in supplying trained personnel be intensified but also a certain specific preparedness for a national emergency will be materially affected, if it is not so already.

2. With the above presentation of facts and bearing in mind anticipated lack of funds for training in nonservice institutions, it is requested that, after consultation with the commanding officer and chiefs of services, your views as to the best method of solving the problem be supplied the bureau.

3. Your assistance in this matter at the earliest convenient date will be much appreciated.

THE REPLIES

(1) The nurse who gives general anesthetics is now on part-time duty, the remaining time in the dressing room. During last fiscal year—ending June 30, 1932—in 1,359 general surgical cases the following anesthetics were given: Spinal, 644; caudal, 311; local, 381; general, 23.

In the eye, ear, nose, and throat department: Cocaine, 493; procaine, 689; local, 37; ether, 14.

Mastoids are given general anesthetics and they are also used at times for plastic work.

With full agreement of all members of the conference, the following is recommended:

(*a*) Junior medical officers and selected Hospital Corps men to be given instruction in general (and other) anesthetics by both lectures and demonstrations.

(*b*) The surgical staff to give more general anesthetics, in selected cases where the risk is excellent, in order that more experience may be given the members of the "anesthetics class."

As noted by Doctor Lundy (Mayo Clinic) (Journal American Medical Association, September 17, 1932), there seems to be an increased danger in giving spinal anesthetics for cases above the umbilicus, and, of course, spinals are not considered for thoracic cases. Work on the legs and arms can usually be best handled by nerve block.

I believe we must accept the fact that spinal and local anesthetics will be used in a large majority of cases in the future. In the field (and aboard ship during action) the determining factor would seem to be whether or not absolute sterilization can be secured—for spinal anesthesia, probably this would not always (or often) be possible—and nothing less than a perfect technique can be used for spinal injections. Chloroform, now seldom used, may be necessary (in the field) and the hospital plans to give at least didactic instruction in its administration.

(2) The discussion of the merits of general anesthesia versus local, regional, and spinal anesthesia is a never-ending one. To make my answer short, concise, and to the point, I will submit facts only as I have interpreted them in conversation with 10 medical officers of all ranks during the past few days.

The staff at * * * hospital is prejudiced in favor of spinal, local, and regional in nearly all operative cases of procedures below the neck, except in children, and is for rectal ether in head cases and mastoid surgery. The staff has no hesitancy in stating that this type, local-spinal, is far superior for general service conditions. There are no failures and no casualties, at least there have not been any at the * * * naval hospital; they have had only two sequelæ in over a year, both having bladder paralysis, one lasting two days and the other several weeks, both ultimately recovering.

In over 600 cases operated upon in the main operating room in the last eight months at the * * * naval hospital there has been but one straight ether, and that was given for a personal idiosyncrasy to novocaine. The officers at the hospital, however, admit that 2 to 3 per cent of these cases are completed with ether; that is, reenforced for the last 15 or 30 minutes by a light ether or gas anesthesia. These hospital medical officers contend that all junior medical officers should be especially instructed in regional, local, and spinal anesthesia and also that they should know at least the rudiments of

general anesthesia prior to entering the Navy. They, the hospital officers, contend that while trained ether and gas anesthetists in naval hospitals are highly desirable, they are not absolutely necessary to present-day functioning of these institutions. One medical officer was serious in likening a trained ether anesthetist in a hospital to a spare tire on a motor car—bad to be without, not absolutely necessary most of the time, but absolutely necessary in an emergency which occurs when least expected. The hospital officers do not take the position, unalterably, that ether anesthesia is to be abandoned, but that continued general instruction in this type should proceed and that medical officers should know both types well—capable of doing either one as the case at hand may demand. Of course, due allowance should be made for hospital officers' prejudice in favor of spinal and regional. They do it every day, have developed a highly trained staff in this specialty, and this must be considered when applied to the service at large, on board small ships, in dispensaries, and in the field. The hospital medical officers see no cause for apprehension in using such a large percentage of regional and spinal anesthetics, for, they say, that in a national emergency they could proceed as they are now doing and obtain the necessary number of skilled general anesthetists from civil life. I quite agree with them on this point and am of the opinion that regional and spinal anesthesia is not used to such an extent in civil hospitals as in naval institutions. However, I do not believe this fact should deter for a moment the continued education in general anesthesia in the Navy during peace times.

One commander of the Medical Corps, just coming from the fleet, told me that he thinks that at least 90 per cent of the emergency operations at sea are done under ether-gas-oxygen or straight ether. Another commander of the corps, a man who has done family practice for years, is by no means convinced that spinal can or ever will supplant ether anesthesia. This medical officer is constantly in touch daily with the * * * and * * * hospitals located in * * *; is associated with many general surgeons and obstetricians, and he stated that ether-gas-oxygen preponderates over spinal anesthesia by 10 to 1 in these hospitals and in even a higher percentage than this. Both of these commanders agree, and I am also of the same opinion, that ether must remain the basic anesthesia in the hands of the medical profession at large and the choice of anesthesia, except where trained regional and spinal experts are developed, and even then, at these places, trained ether anesthetists are highly desirable, for one never knows when an ether anesthetist is needed.

My reaction to this question is that one group will argue for the spinal cause, one group for the ether cause; but when all is said and done, the ether methods, both open and machine methods, must be taught and continue to be taught to doctors, nurses, and chief petty officers, or reliable first-class petty officers.

Medical officers in hospitals give local and spinal anesthesia in such a preponderance of cases for three main reasons:

(*a*) Mortality and morbidity are reduced to a minimum—less than ether; hence less chance of inviting trouble in the military-naval service.

(*b*) Less discomfort after operation to all patients as well as during the operation, except to the highly nervous ones.

(*c*) Less trouble for all concerned during operations and in post operative care of the patient.

From the standpoint of the surgeon, spinal anesthesia is almost ideal, giving excellent anesthesia and relaxation quickly and with a minimum of assistance, as it can be given by the surgeon himself; but the studies of Crile and others have conclusively proved that the damage to the brain cells by pain or even the anticipation of pain is considerable, and it is a question whether or not medical officers have allowed their enthusiasm for spinal anesthesia and local anesthesia to dim their eyes to the mental side of surgery, and it is not the surgeon that will eventually see cases of lame backs, and vague headaches, fancied, it is true, but the internist or the psychiatrist.

The problem of anesthesia peculiar to the Naval Establishment resolves itself into two factors: First, the policy, the wisdom of which it is difficult to dispute, of studiously avoiding giving any general anesthetic in any naval station except naval hospitals; second, there is no doubt that an element in the passing of ether-gas anesthesia in dispensaries and small shore stations—and an element hard to combat—is the constant fear of a fatality with subsequent investigation and court-martial. Hence the shifting of practically all cases needing an anesthetic other than the most trivial and the attendant responsibility to the nearest naval hospital, the result being that medical officers and corps men, outside of naval hospitals, see very little anesthesia of any kind, the cases needing a little ether going to the nearest hospital.

It is easier to criticize than to offer a practical remedy, but one partial solution to the problem might lie in the encouraging of the giving of light narcosis by nitrous oxide or open-drop ether, both of which will show mortalities as low as spinal or local anesthesia, in shore stations and naval hospitals. Intelligent Hospital Corps men are much enthralled at the prospect of participating in any surgical procedure, and it would not be difficult to find sufficient men to train

one or two on each station, other than at hospitals, to give the lighter grades of anesthesia.

(3) Attached memorandum is inclosed so that the bureau may learn at first hand the situation regarding gas anesthesia at the naval hospital * * *. A similar situation presented itself in the * * * naval district somewhat more than a year ago. The only solution found after thoroughly canvassing all likely institutions in * * * was the offer of a special course in gas anesthesia for Hospital Corps men at one of the civil hospitals in * * *. All this information was submitted by me as district medical officer to the bureau at that time.

The situation seems to resolve itself to this, namely, no facilities exist at naval Medical Department activities within this district for training Hospital Corps men in the administration of general anesthesia.

Comments of the medical officer in command of * * * hospital: It is unquestionably true that until recently anesthesia by means of drop ether was the method of choice for major operations on board ship and in the field; and it is the case that for some years the bureau has been concerned about how corps men could be trained in that method now that in hospitals, where if anywhere experience must be gained, ether has been all but displaced by local, caudal, and spinal anesthesia or by other inhalation anesthetics (nitrous oxide, ethylene).

The extent to which ether has passed out is revealed by the fact that ether is employed at this hospital for general surgery in only about 4 per cent of cases, and it is to be noted that it is used so often only because we considered it a requirement that each intern should have had at least 10 ether anesthesias. In other words, when ether is administered nowadays, it is not as the anesthetic of choice, but to give internes stipulated experience.

It being, however, wholly impracticable to give similar experience to corps men, they will continue to go out from our hospitals lacking so much in training. That particular training heretofore has been regarded as an essential element of medical preparedness. As a matter of fact, it is no longer important or even desirable for conditions have come about such that we can with advantage abandon ether, placing dependence hereafter on agents which are topically applied.

I believe we can take the step with assurance * * * The advantages inherent in any of the methods of locally applied anesthesia are obvious: The agents used are less bulky and neither explosive nor inflammable; the use of sterile ampule containers makes handling simple; an anesthetist is not required; after care of patients is less onerous. Hence, whatever question may arise concerning the feas-

ibility of employing such agents turns on the ability of personnel to use them.

Such ability is general. In the first place, every medical officer qualified to perform a major surgical operation is certainly able to do his spinal, caudal, or local anesthesia. Secondly, virtually every medical officer has learned to do a spinal puncture, for purposes of diagnosis, therapeusis, or anesthesia, so that to-day a far larger proportion of officers are capable of doing a spinal anesthesia than formerly were able to give a good ether.

It will occur to you that the course suggested makes no provision for those cases in which a general anesthetic is still commonly employed—mastoids, for example * * * The answer is that local or regional anesthesia, supplemented on occasion with some basal anesthetic, suffices in practically every instance of operation at a level too high for a safe spinal. The cases in which inhalation or rectal anesthesia is obligatory are too few to require consideration in a general program.

A further consideration which should reconcile us to abandoning an old friend is this: It is exceedingly probable that in a short time there will have been developed safe drugs for inducing general anesthesia by the simple technique of intravenous injection.

I have given no consideration to nitrous oxide and ethylene because their use in the field is impracticable.

Summary: Plans for training and supply may hereafter be based on the assumption that injection methods exclusively (local, caudal, spinal, vascular) will be employed on board ship and in the field for the induction of anesthesia.

(4) Practically all of the major operations at this hospital are performed under spinal anesthesia, and it is the opinion of the chief of the surgical service that it is the safest and best anesthetic. A series of 3,000 cases without a death are recorded. It is more economical, costing approximately $0.06 per patient against $1.50 for gas-ether. It permits greater rapidity in handling cases and economy in personnel. The technique of administration is important and the Labat method is considered the most satisfactory. Approximately one-third of the medical officers attached are competent to give spinal anesthesia because of their familiarity with spinal puncture in the medical, urological, and surgical departments. Persistent headache may occur after spinal anesthesia, but this is offset by the post operative effects of general anesthesia. There are a few cases where general anesthesia must still be used and also a certain percentage of cases where spinal anesthesia must be supplemented with general anesthesia, hence the service of a trained anesthetist is required at our hospitals. A good machine is very valuable. The one

at this hospital is not satisfactory. All the methods of local, regional, and spinal anesthesia are in use. The value of avertin is emphasized.

It would appear that the problem presents two aspects: First, that of training Hospital Corps men to give a general anesthetic when necessary on shipboard or on field service, when the medical officer does not desire to give spinal anesthesia. Second, the training of nurses to give anesthetics at hospitals or on the hospital ship. It is believed that the nurses' training render them more suitable for special instructions in anesthesia than Hospital Corps men, for the rated men are needed in the administrative departments.

In solving the problem as presented it is recommended that—

(*a*) Instruction in the use of the drop method of giving ether be continued for Hospital Corps men in view of the necessity of their giving a general anesthetic on shipboard or in the field.

(*b*) It is considered that the small cost of instruction ($100) at a nonservice institution for the extensive experience acquired warrants the continuation of these courses for nurses, if possible. If funds can not be used for this purpose, however, then it is recommended that several of our larger hospitals be designated to give this training and that two nurses be assigned to each hospital for a six months' course of instruction. It is believed that these nurses should have no other duties and not be included in the hospital complement for general duties. They should be instructed by a competent nurse anesthetist. Those three nurses should be in addition to the nurse in charge of the operating rooms.

In view of the belief of many surgeons that ether is the safest anesthetic and that spinal anesthesia, in spite of its advantages and economy, has not yet been fully established as the anesthetic of choice (Journal American Medical Association, September 17, 1932), and also that supplementary general anesthesia may have to be used, it is recommended that, if instruction in anesthesia is given to nurses at naval hospitals, such hospitals be directed to use general anesthesia in a sufficient number of cases to provide the necessary instruction. The use of supplementary anesthesia to spinal anesthesia, it is considered, will not give adequate instruction.

(5) The problem of anesthesia for the Navy admittedly is complex.

The trend in favor of local anesthesia in selected cases is very strong, as indicated by paragraph 1 of reference, by data from naval hospitals, and just-furnished information showing present practice of civil hospitals hereabouts, as follows:

	Percentage of anesthesias	
	Infiltration spinal caudal	Inhalation
* * * Hospital	50.00	50.00
* * * Hospital	38.52	61.48
* * * Hospital (about)	10.00	90.00
* * * Hospital	60.00	39.00
* * * Hospital	20.00	80.00
U. S. Naval Hospital * * * (past 2 years)	97.51	2.49
U. S. Naval Hospital * * * (past 6 months)	95.80	4.20
U. S. Naval Hospital, * * * (Oct. 1, 1931, to Sept. 30, 1932, exclusive of nose and throat), 1,258 major operations:		
Spinal, 405, or 32.20 per cent Caudal, 163, or 13 per cent Infiltration, 531, or 42.20 per cent Avertin, 27, or 2.10 per cent Gas-ether, 132, or 10.50 per cent	87.40	10.50

The foregoing data, plus experience in naval hospitals, and such expression of editorial opinion on the subject as appeared in the conservative London Lancet of August 6, 1932, cause me to modify my attitude toward surgical anesthesia produced otherwise than by inhalation.

Infiltration, spinal, and caudal anesthesia appear to have a widening field of usefulness in selected cases, particularly for the men of the active service. It is believed, e. g., that the percentage of use of local methods of anesthesia would be higher in civil hospitals but for the character of patients served (women, children, and aged).

It would appear that—

(*a*) Personnel trained in anesthesia (inhalation or otherwise) may be found always in our naval hospitals where major surgery is being done.

(*b*) There is a great improbability that a technically trained anesthetist can always be available at all places where emergency operation is indicated.

(*c*) All naval medical officers are, or should be, capable of administering ether in emergency (if they are not trained in administration of local anesthesia or the various inhalation anesthesias), and if a medical officer is the sole medical attendant he can supervise administration of ether by the Hospital Corps men. (The medical officer must in emergency do as the country doctor—render the best medical attention available.)

(*d*) Since the Navy's peace-time requirement seems to be not over 10 per cent of gas or gas-ether anesthesia, it is desirable to embrace every opportunity offered for instruction of personnel in inhalation anesthesia in our hospitals where and when the administration of inhalation anesthesia is desired by the surgeon.

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(*e*) Medical officers trained in administration of the inhalation anesthetics should be detailed at the larger hospitals to instruct personnel when inhalation anesthesias are indicated.

(*f*) In view of the preference and responsibility of the operator it appears of questional propriety to require inhalation anesthesia in cases where it is not definitely indicated.

(*g*) In national emergency the larger hospital units probably would have trained anesthetists from civil life capable of administering the various inhalation anesthetics as indicated, and desired by the operator.

(6) The subject of instruction in methods of administering general anesthetics has been given careful consideration. Medical officers in the naval service and surgeons in civil practice have been consulted regarding the matter. The report of the final conclusions of the commanding officer and the staff at the naval hospital * * * is inclosed.

The findings and conclusions contained in this report, I believe, will express the majority opinion of the younger generation of operating surgeons in the Navy. The Navy offers an unusually fertile field for the employment of spinal, regional, and local anesthetics, because we are here dealing almost entirely with adult males.

In civilian hospitals one finds a higher percentage of general anesthetics. Many of the patients are women and children in whom the psychical factors are important. In the administration of general anesthetics one finds a tendency to specializing to a high degree. (Reference: The article by Dr. Frank H. Lahey, of Boston, in the Journal of the American Medical Association of September 17, 1932.)

In a large percentage of cases in both naval and civilian hospitals general anesthetics are given only to complete the anesthetic effects of other types such as spinal, regional, local, intravenous, rectal, etc., which in themselves are insufficient for operative procedures. These are completed by a small amount of inhalation anesthesia.

Taking into consideration all of the above facts, I am of the opinion that naval operating surgeons should be allowed to select the anesthetic to be given the patient and be held responsible therefore, even though he delegates the duty of administration to another. The operating surgeon should be given consideration regarding suitable assistants of junior medical officers, nurses and Hospital Corps men, but he should be held responsible for training his own personnel in giving anesthetics of his own choice. Personnel thus trained should be continued on this duty as long as the exigencies of the service permit.

In accordance with the trend in civilian hospitals, I am of the opinion that only medical officers should administer anesthetics other

than ether by the open drop method. Other methods of giving anesthetics are too technical or complicated to be administered by one who has not been thoroughly trained. On the other hand, a nurse or preferably a corps man may be instructed in giving ether by the open drop method. This type of instruction would prove most useful in expeditionary duty where only simple, standard outfits are available.

Comments of the commanding officer of * * * naval hospital: In accordance with the bureau's letter of September 21, 1932, the question of instruction in methods of administering general anesthetics was taken up with the chiefs of the medical and surgical services of this hospital. The consensus of opinion seems to be that, whenever practical, general anesthesia is being replaced by other and apparently better methods; that, in the event of war, local, regional or spinal anesthesia would be the anesthetic of choice.

At this hospital from January 1, 1932, to September 30, 1932, 371 major operations were performed. In this series of operations general anesthesia was employed in 78 cases, or 21 per cent, as follows: Gas and oxygen, 30; gas, oxygen, and ether, 41; spinal with gas and oxygen, 3; spinal with gas, oxygen, and ether, 1; and spinal with ether, 1. From these figures it may be seen that there is little opportunity afforded for the training of anyone in the administration of general anesthetics, certainly not the opportunity which existed in all of our hospitals 20 years ago when the employment of general anesthesia was in vogue.

Lieutenant Commander * * *, Medical Corps, United States Navy, chief of the surgical service at this hospital, is of the opinion that spinal, regional and local anesthetics are the anesthesias of choice for the following reasons:

(*a*) The preponderance of the safety factor over that of general anesthesia.

(*b*) The ease of administration.

(*c*) The complete relaxation obtained under spinal anesthesia.

(*d*) The greater post-operative comfort of the patient.

(*e*) Less post-operative care required.

(*f*) Shorter convalescence.

(*g*) No trained anesthetist or complicated apparatus necessary.

When ether was used almost exclusively as a general anesthetic, it was not a matter of great difficulty to train a Hospital Corps man in its administration. The introduction of gas and oxygen alone or combined with ether produced a more difficult problem, which was solved by the training of members of the Nurse Corps in their administration. As the number of medical officers, nurses, and Hospital Corps men attached to this hospital is limited and is barely sufficient to

cover the various details, it does not seem feasible to assign any one person to the surgical department for the purpose of instruction in general anesthesia, particularly in view of the slight use of that method at the present time.

A younger generation of medical officers is now doing the surgical work of the Navy, and it is believed that, with these officers, spinal, regional, and local anesthesia will be used almost exclusively.

In summing up the situation as it applies to this hospital, the facts are as follows:

(*a*) General anesthesia is not the anesthetic of choice.

(*b*) Additional personnel would be required for training.

(*c*) General anesthesia is used only in about 20 per cent of cases.

(*d*) The younger men are being trained to operate without general anesthesia.

The following suggestion is offered for what it is worth: That a certain number of well-chosen nurses and first-class pharmacist's mates be selected each year for training; that they be assigned to one of the two largest naval hospitals with the request that those hospitals furnish the necessary training. This would require longer than a few months because of the few general anesthetics given; but over a longer period, such as a year, it seems that they could acquire the proper technique.

(7) The solution of the problem set out in your letter seems rather difficult. The main trouble appears to be that, as the giving of an anesthetic is an event which does not admit of a divided responsibility, it is probably impossible to dictate to an operating surgeon the nature of the anesthetic he is to use.

From several conferences here I get the impression that general anesthesia is used in about 10 per cent of cases.

About the only suggestion I could offer is to encourage the use of general anesthetics by moral suasion and have groups of medical officers, nurses, and Hospital Corps men receive instruction at naval hospitals where those clothed with discretionary authority are favorably disposed toward the use of a general anesthetic in a worthwhile percentage of cases.

In aviation all students get long instructions in what they call "ground work." This instruction appears to be of great value and makes a student almost a flyer before he sits in a plane or leaves the ground. An analogous system may be of some help in solving your general anesthetic problem. So my suggestion is that the higher ratings of Hospital Corps men be given thorough preliminary and theoretical class instruction in giving of general anesthetics followed by practical instruction at hospitals where the use of ether can be encouraged.

(8) The bureau's statement in reference that according to the records of one of the major hospitals of the service general anesthesia was employed in only 21 out of a total 2,866 operations is quite close to existing conditions at the naval hospital * * *. Here general anesthesia is rarely employed and consequently little opportunity for practical instruction in its administration is presented. These facts point to the more and more prevalent recognition among operators of the value of local anesthetics particularly in emergencies, aboard ship, and following battle. They also indicate so decided a trend away from general anesthesia that even the thoroughly trained anesthesist in the Navy is threatened with impaired technique through lack of practice.

Based on opinions expressed during the consultations at this hospital and on the consensus of opinion prevailing at its last weekly conference, which was devoted almost entirely to anesthesia, the only solution to the problem submitted in reference lies in instituting classes of Hospital Corps men of the higher ratings for instruction by nurses and others who have had training in the administration of general anesthetics. The opinion is also general that such instruction should be confined to the ether drop method, which is considered the only safe and universally available one for partially trained men.

It is proposed to institute such instruction at the naval hospital * * *, and it is believed that if the same policy is adopted at all naval hospitals and aboard such vessels as have facilities the problem will be partially solved, at least.

If the bureau approves this policy and should desire to issue instructions for its general adoption, it is suggested that these instructions require that notation be made in the service records of the men concerned, showing that they have satisfactorily completed the course in general anesthesia by ether drop method, and that lists of such men be periodically forwarded to the bureau for its reference in making assignments. These notations and reports should stress, however, facts as to whether the course in the case of each man was confined to theory and demonstration or included practical administration of general anesthetics.

(9) The undersigned believes that this would be an appropriate time for an authoritative statement of the present status of anesthesia, based upon the recent conclusions and practice of the leading surgical clinics of the country, to be promulgated throughout the service. Such a circular would serve to recall to our surgeons the desirability of bearing in mind that all the methods of anesthesia have advantages and disadvantages which should be evaluated in every case before a choice is determined upon. It would in no way

serve to limit or restrain our surgeons in the exercise of their professional judgment.

The following excerpts may be cited from one authoritative report upon the present status of the anesthesia problem, that of Dr. A. D. Bevan, professor of surgery of Rush Medical College, appearing in the Journal of the American Medical Association, November 21, 1931:

> Ether by the open drop method is the safest general anesthetic known for producing prolonged anesthesia and complete relaxation. It should be to-day the standard anesthetic of the surgical clinic.
>
> I believe that local anesthesia with procaine and epinephrine is a very safe and satisfactory method which should be employed in a large group of cases. I do not believe that the range of local anesthesia should be stretched too far. I am using it in about 30 per cent of my cases.

Of spinal anesthesia he makes the following remarks:

> The method is dangerous, more so than chloroform. There is probably more than 1 death in 500 cases. There were 14 deaths in greater Boston in the year 1928 in 1,900 cases collected and reported by one of the anesthetists of the Lahey Clinic, Dr. Lincoln F. Sise.
>
> Spinal anesthesia is not a comfortable method, especially to an intelligent man who realizes what is being done. Few medical men with their knowledge of anatomy and of anesthesia would choose spinal anesthesia in their own case. The application, therefore, of the golden rule should limit spinal anesthesia to a narrow field.

Quoting Dr. W. W. Keen:

> The ideal anesthetic will abolish pain by the abolishment of consciousness but without danger to life. We must consider the mind as well as the body of the patient.

From the above it is seen that what is considered good practice in some of the naval surgical activities is not in accord with this large clinic at least. In any case, speaking from some experience, it would seem that surgeons will do well to be on their guard against overenthusiasm for any one method of anesthesia. They should also avoid drawing conclusions as to the safety of any one method as employed in a relatively small number of cases. The value of each method should be considered in every case and surgeons should remember the need for instruction material in general anesthesia to provide for future emergencies in so far as possible without detriment to the welfare of the patient.

It is the opinion of all the senior medical officers of this district who were consulted that our naval hospitals can provide the necessary training in anesthesia by instituting courses of instruction for nurses and selected corps men. This implies that the present status of the anesthesia problem still warrants the use of the general anesthetic in sufficient number of cases to provide ample material for the practical side of the undertaking.

In order to include all the opinions of the medical officers who were asked to comment on the bureau's letter, reference (a), there is inclosed their written statements for the bureau's further consideration.

Anesthesia is a subject rating careful consideration, a division of surgery the importance of which is not fully recognized by all of our teaching institutions, both service and nonservice. It is a broad subject and the comprehensive survey to determine methods of instruction should include all types of anesthesia, i. e., local as well as general.

Despite the absence of well-organized service training in anesthesia, advancement has been made and is improving throughout the naval service. Specific preparedness for a national emergency has been markedly affected, definite progress and improvement in the scope of anesthesia being constant with the growth of local anesthesia. The medical officer trained in local anesthesia is versatile and no longer dependent, as in former emergencies, upon selected personnel and equipment. Inhalation anesthesia in the naval service, especially during periods of emergency, is not necessarily a serious problem. A basic indoctrination in the use of inhalation anesthesia is possessed by all medical officers prior to entry into the service. Active field surgery, afloat or ashore, does not increase the scope of general anesthesia, but rather tends to restrict its use to simple open drop administration. All surgical units could readily master this.

From January 1, 1930, to the present date—October 6, 1932—the * * * naval hospital has been designated as a place for postgraduate courses of instruction in surgery. The medical officers who have completed 6-month periods of instruction, five in number, are now qualified local anesthetists, which, in addition to their basic training in general anesthesia, renders them qualified for any surgical unit. In addition to the 5, especially designated for instruction, 10 interns and 6 junior and senior medical officers have been trained in the use of local anesthesia sufficiently to meet routine surgical demands. During this period over 4,200 operations were performed, general anesthesia being employed 30 times. The above is cited not only as an instance of the present trend in anesthesia, but as an example of the training facilities available in naval hospitals. This officer personnel so trained could be diffused throughout the naval service and made teaching nuclei for developing surgical teams wherever anesthesia might be a problem.

The training of nurses and Hospital Corps men in anesthesia should not be confined to general anesthetics. Their training should include basic instructions in the preparation of local and spinal anesthetic agents and attendance upon the patient and surgeon dur-

ing its administration. It is my opinion that this may be best accomplished at our major hospitals. I do not believe that the training in nonservice institutions could be as adequate or as efficient.

It is a serious error to consider local anesthesia a fad, for it has passed the experimental stage and is now generally accepted that its scope may include any operation. Its extensive use in all of our major hospitals has qualified many medical officers in its administration. This number could be consistently increased and the qualified officers assigned to hospitals as members of surgical units to train officers and corps men in all forms of anesthesia.

For the above reasons it is recommend:

(*a*) That instructions and methods of administering anesthesia should include all types of anesthesia.

(*b*) That instructions be carried out at our own major hospitals and the individual surgical service be made responsible for the developed in its own anesthetic teams.

(*c*) That the officers so trained in anesthesia be listed and thus be available to train personnel wherever demands might arise.

(*d*) That the relative value of the various types of anesthesia in the advent of national emergency be determined before concentrating on any one type, keeping in mind the economy, adaptability, safety, availability, and efficiency of local agents.

(10) It is my opinion that local and spinal anesthesia is a very valuable agent in the hospital, aboard ship, and to a less extent in the field. However, there are certain operations which can be more advantageously performed, both to the benefit of patient and surgeon, under general anesthesia.

In every well-organized hospital there should be at least one trained anesthetist, and I feel that the bureau should economize in other ways than in failing to provide anesthetists. To effect this condition it is suggested that certain qualified nurses be given a special course of instruction at a school to be organized at Washington, D. C., in conjunction with the naval hospital, to be somewhat of the same nature as is now being offered to medical and dental officers, but dealing with the subjects related to anesthesia. If it would be more economical as far as travel is concerned, a similar school could be located on the west coast in conjunction with one of the naval hospitals there. This would provide a corps of nurses qualified in anesthesia and trained at Government hospitals at little expense.

It is my belief that some of the older nurses who have had several years experience in anesthesia would make good teachers and would be glad of this opportunity to teach others.

(11) Although nearly all operations in the eye, ear, nose, and throat department are done under local and block anesthesia, the

writer considers it necessary to use general anesthesia on the following types of cases:

(*a*) Children.

(*b*) Males and females difficult to control by mental suasion who require extensive operations.

(*c*) Any protracted operation on head as radical or even selected so-called "simple" mastoid operations, lateral sinus operations, labyrinth and decompression operations.

The writer has had a tolerably extensive experience both in operations under general and local anesthetics, as well as in the administration of general anethesia without fatalities from either form of anesthesia. He has been stationed in rather isolated places where, to his dismay, nobody passably trained in the administration of a general anethetic was at hand and it developed on him both to start and supervise the general anesthetic as well as to operate.

After weighing the subject pro and con he concludes that there is a definite and necessary place for general anesthesia. He is all the more convinced of this from his contact with civilian institutions of learning where they are constantly on the lookout for the improvement in the administration of the old and tried general anesthetics and engaged in the trial of new ones.

(12) Recommend intensive instruction in local anesthesia so that general anesthesia may become even less frequently used.

Local anesthesia is very much more suitable in military surgery than general anesthesia.

Medical officers have already had some practical and theoretical instruction in general anesthesia prior to entry into service. It is believed that the facilities for training individuals in general anesthesia will continue to diminish as the use of local anethesia increases and that the necessary training can best be obtained by special courses of instruction. As there are no funds available at present, these should be postponed until such funds are available.

(13) It is the opinion of the surgical and administrative staff that it is deemed advisable to instruct Hospital Corps men in anesthesia in naval hospitals and that a proportionate number of operations should be designated to be done under general anesthesia for instruction purposes, as no other anesthetic yet developed is suitable for all types of operation.

(14) After having had the pleasure of observing the surgical service of this hospital for a period of two years, it is evident that various methods of local anesthesia have displaced general anesthesia except in certain selected cases. It is also evident that more work is completed with less nursing assistance and that the morale of the patients is very high. The patients are always happy. No longer do

you see a nurse and a corps man guarding every general anesthetic case for hours following the operation and holding the pus basin while the unfortunate patient empties his stomach. If, as we know, under local anesthesia more and just as good surgery can be done with less assistance, less cost, and a higher morale of the patients, it would seem that our chiefs of services and their assistants should be still better trained in methods of local anesthesia so that in case of a national emergency more work could be done with less help and with local anesthesia in place of general.

If the bureau considers it necessary to have surgical assistants trained in general anesthesia in order to meet a national emergency, it would seem that this instruction could be given in the naval hospitals having the larger surgical services for a period of two months each year.

(15) It is believed that the training in administration of general anesthetics for naval personnel—officers, nurses, and enlisted men—can best be met by requiring the chiefs of all surgical units to employ general anesthesia in a selected group of surgical cases to the extent of at least 10 or 15 per cent.

It is believed at the present time the use of local and spinal anesthesia amounts to almost an obsession with many operators. It is further believed that local anesthesia is frequently employed to the detriment of the patient, and it is predicted that the enthusiasm for local anesthesia now being manifested by so many operators will decrease markedly with increasing experience. It is believed that local and spinal anesthesia are most valuable adjuncts to the naval surgeon, but both are methods requiring highly specialized knowledge and great experience in their successful employment.

Ether administered by the "open cone" method is a procedure, in most cases, equally as safe in major surgery as local anesthesia. It has the advantage of being known to all graduates of medicine. An average medical man can give a safe and satisfactory general anesthesia with ether. This does not obtain in the case of local or spinal anesthesia.

For the above reasons the recommendation already set forth is believed to be desirable.

(16) I have delayed answering your letter of September 21 in reference to trained and experienced personnel in the administration of anesthesia, both general and local, in order that I might canvass the ships of the * * * force and give you as accurate a picture of the situation as possible. This canvass includes practically all the major ships of the force, a total of 18 ships.

I find that on these ships there are at present 26 members of the Hospital Corps who have had more or less experience in the administration of general anesthesia, divided as follows:

Chief pharmacist's mate	11
Pharmacist's mate, first class	8
Pharmacist's mate, second class	4
Pharmacist's mate, third class	2
Hospital apprentice, first class	1
Total	26

In addition to the above, there are 20 Hospital Corps men with some experience in local anesthesia, including spinal anesthesia.

In discussing the subject with several ships' medical officers and in correspondence with others I note a general trend in the force toward spinal and local anesthesia when appropriate. Most medical officers who are without an assistant medical officer prefer to administer their own spinal or local anesthesia and then proceed with the operation. The senior medical officer of the * * * states that he has performed in the present calendar year five appendectomies under spinal anesthesia and considers it safe and efficient if given not higher than the second and third lumbar vertebræ.

I find that medical officers of the force are endeavoring to instruct and train their corps men in so far as possible in anesthesia methods, but I feel that this training is inadequate.

In my opinion the most practical system of training in so far as Hospital Corps men are concerned would appear to be in our naval hospitals, and I think that pharmacist's mates, first and second class, on duty in hospitals should be given a course of instruction, following which, if qualified, they be carried on the bureau's records as qualified in anesthesia (stating method in a manner similar to the present practice of noting on the man's record his ability as a technician in laboratory work, dental work, etc.

(17) Your letter of September 21, 1932—reference to the problem of instruction in methods of administering general anesthetics—was received while the ships were in * * *, and the reply has been delayed as I wanted to discuss the question at the first medical conference of this force.

It is believed that all dental technicians—and perhaps all other technicians undergoing instruction in the Naval Medical School—should receive instruction in the methods of administering general anesthetics.

This subject could be more emphasized as one of the requirements for those men taking examination for pharmacist's mate first class, and chief pharmacist's mate.

If the above-mentioned groups qualify as anesthetists, this would supply the approximate number required for national emergencies. Regarding the method of furnishing practical training to such men, the problem does not appear so simple. Our naval hospitals and other medical organizations are in a position to give practical demon-

strations in the preferred methods of administering general anesthetics.

If our technicians and higher ratings are well grounded in the fundamentals, then, in event of a national emergency, the acquiring of practical experience will be automatically solved as the casualties begin to arrive.

(18) Reference letter states that no funds are available for training in nonservice institutions and that general anesthesia is seldom employed in our own hospitals. As a result, training of Hospital Corps men in the administration of general anesthetics is impossible, as this is a brand of medicine which can not be learned from books but only by experience in administering a long series of anesthetics.

There is no opportunity for training in this subject on board ship. Very few cases requiring general anesthesia occur, and the bulk of these cases are transferred to hospitals or hospital ships and the negligible number of cases that are operated on at sea does not provide sufficient material to train competent anesthetists.

During the last four years I have been much impressed by the increasing use of Avertin by my friends who are surgeons in civilian life. There are objections to its use, but here is a drug that can be administered by any corps man who can give an enema, can calculate the patient's body weight, and measure out the required number of millilitres. This form of anesthesia can be given much more safely by untrained or poorly trained personnel than any other form of general anesthesia. In some cases it has to be reinforced with a few whiffs of ether at certain stages of the operation, but in general I think there will be less danger from its administration by untrained personnel than will arise from the use of inhalation anesthetics by partly trained personnel.

DATA COMPILED FROM ANNUAL REPORTS OF THE SURGEON GENERAL, UNITED STATES ARMY

	Per cent
For calendar year 1929:	
Inhalation anesthesia	35
Local anesthesia	65
For calendar year 1930:	
Inhalation anesthesia	36
Local or spinal anesthesia	64
For calendar year 1931:	
Inhalation anesthesia	29
Local anesthesia	62
Spinal anesthesia	9

DATA COMPILED BY DIVISION OF STATISTICS, BUREAU OF MEDICINE AND SURGERY, NAVY DEPARTMENT

Through the active interest of Capt. O. J. Mink, Medical Corps, United States Navy, valued information emphasizing the trend in

anesthesia has been recently compiled. With his permission the following is quoted:

General anesthesia, United States Navy, 1926 to 1931

	1931	1930	1929	1928	1927	1926	1920
Total anesthesias	3,241	3,306	2,851	3,018	3,137	3,281	----------
General	435	1,040	1,405	1,471	1,624	1,788	----------
Per cent general	13.4	31.5	49.3	48.7	51.8	54.5	----------
Total diseases	2,958	3,103	2,697	2,767	2,887	2,858	----------
General	349	920	1,280	1,285	1,464	1,474	----------
Per cent general	11.8	29.6	47.5	46.4	50.7	51.6	----------
Total injuries	283	203	154	251	250	423	----------
General	86	120	125	186	160	314	----------
Per cent general	30.4	59.1	81.2	74.1	64	74.2	----------
Appendectomy:							
Total	1,355	1,505	1,306	1,151	1,244	1,182	1,208
General	205	496	762	758	777	775	1,198
Per cent general	15.1	32.9	58.3	65.9	62.5	65.6	99.2
Herniotomy:							
Total	434	510	445	453	427	434	731
General	24	125	165	149	161	185	718
Per cent general	5.5	24.5	37.1	32.9	37.7	42.6	98.2
Hemorrhoidectomy:							
Total	334	435	422	362	327	436	446
General	2	23	16	22	57	115	384
Per cent general	0.59	5.3	3.8	6.1	17.4	26.4	86.1

It is believed that a study of the trend in the use of general anesthesia for the past six years will prove a matter of interest. That a great decrease has occurred in the use of gases in anesthesia during the past 12 years is well known. In comparing 1920 with 1931, general anesthesia in appendectomy has fallen from 99.2 per cent to 15.1 per cent, in herniotomy from 98.2 per cent to 5.5 per cent, and in hemorrhoidectomy from 86.1 per cent to 0.59 per cent.

In a more careful study of the past six years the rates were studied for the following three groups: Total operations, operations for disease conditions, and operations for injuries.

In the tables submitted it is shown that the general anesthesia rate for 1931 compared with 1926 has dropped as follows: All operations, 54.5 per cent to 13.4 per cent; operations for disease conditions, 51.6 per cent to 11.8 per cent; and operations for conditions due to injury, 74.2 per cent to 30.4 per cent. The reduction is most marked in operations for disease conditions and the trend from general anesthesia is most marked in the past year.

A further study of operations for disease conditions shows that about 70 per cent of the operations in this group are appendectomies, herniotomies, and hemorrhoidectomies. The rates for herniotomy and hermorrhoidectomy under general anesthesia are very low, 5.5 per cent and 0.59 per cent, respectively. Surgeons, however, still select general anesthesia in one-seventh of the appendectomies.

Injury surgery is probably our nearest approach to war-time surgery, and the reduction in general anesthesia here is much less marked than in the disease group.

Mayo Clinic Data

Of timely interest to our subject are the following tables of Dr. John S. Lundy, of the Mayo Clinic staff, in his article, "Choice of anesthetic method for different types of patients and conditions," published in volume 23, Collected Papers of the Mayo Clinic, 1931:

Table I.—*Pharmacologic effects of most commonly used anesthetic agents*

	Probability that desired effect will be obtained							
	Inhalation and Insufflation							
	Drop [1] ether	Gases [1] (with or without ether)	Barbiturates [2]	Avertin [2]	Spinal [1]	Block [3]	Infiltration [3]	Combined balanced anesthesia [3]
Anesthesia	Yes	Yes	No	No	Yes	Yes	No	Yes.
Analgesia	No	Yes	Yes	Yes	Yes	No	Yes	No.
Relaxation	Yes	No	No	No	Yes	Yes	No	Yes.
Quiet respiration	No [4]	No [4]	Yes	Yes	Yes	Yes	No	Yes.
Prompt recovery of reflexes	No	Yes	No	No	Yes	Yes	Yes	Yes.
Untoward results:								
Immediate	No	No	No	No	Yes	No	No	No.
Remote	Yes	No	Yes	Yes	?	No	No	No.

[1] Mild effect from preliminary medication.
[2] Marked effect from preliminary medication; used intravenously or rectally as a basic anesthetic.
[3] Definite effect from preliminary medication.
[4] With intratracheal—Yes.

Table II.—*Relative safety of various anesthetic agents and methods in relation to physical condition of patient and some common diseases for which operation is performed*

	Safety of agents or method							
	Drop ether	Gases [1]	Barbiturates [2]	Avertin [2]	Spinal	Block	Infiltration	Combined or balanced anesthesia
Very young, 4 years or less.								
1 [3]	Yes	Yes	No	No	No	No	Yes	Yes.
2 [3]	No	Yes	No	No	No	No	Yes	Yes.
Very old, 75 years or more.								
1	No	Yes	No	No	No	Yes	Yes	Yes.
2	No	Yes	No	No	No	No	Yes	Yes.
Pulmonary disease.								
1	No	Yes	No	No	Yes	Yes	Yes	Yes.
2	No	Yes	No	No	No	Yes	Yes	Yes.
Marked cardiac disease.								
1	Yes	Yes	Yes	Yes	No	Yes	Yes	Yes.
2	No	Yes	No	No	No	Yes	Yes	Yes.
Diabetes.								
1	No	Yes	Yes	No	Yes	Yes	Yes	Yes.
2	No	Yes	No	No	No	Yes	Yes	Yes.

[1] Either nitrous oxide or ethylene, depending on whether one must avoid an inflammable gas, and with or without ether.
[2] Used intravenously or rectally as a basic anesthetic.
[3] 1, fair or good physical condition; 2, general debility.

TABLE II.—*Relative safety of various anesthetic agents and methods in relation to physical condition of patient and some common diseases for which operation is performed*—Continued.

	Safety of agents or method							
	Drop ether	Gases	Barbiturates	Avertin	Spinal	Block	Infiltration	Combined or balanced anesthesia
Marked vascular disease.								
1	Yes	Yes	Yes	Yes	?	Yes	Yes	Yes.
2	No	Yes	No	No	No	Yes	Yes	Yes.
Colitis.[4]								
1	Yes	Yes	Yes	No	Yes	Yes	Yes	Yes.
2	No	Yes	No	No	No	Yes	Yes	Yes.
Intestinal obstruction.								
1	No	Yes	No	No	Yes	Yes	Yes	Yes.
2	No	Yes	No	No	No	Yes	Yes	Yes.
Operation on kidney.								
1	No	Yes	No	No	Yes	Yes	Yes	Yes.
2	No	Yes	No	No	No	Yes	Yes	Yes.
Operation on genito-urinary tract.								
1	No	Yes	Yes	Yes	Yes	Yes	Yes	Yes.
2	No	Yes	No	No	No	Yes	Yes	Yes.
Operation on stomach.								
1	Yes	Yes	Yes	Yes	No	Yes	Yes	Yes.
2	No	Yes	No	No	No	Yes	Yes	Yes.
Operation on liver, gall bladder, or ducts.								
1	Yes	Yes	No	No	?	Yes	Yes	Yes.
2	Yes	Yes	No	No	No	Yes	Yes	Yes.
Operation for goiter.								
1	No	Yes	Yes	No	No	No	Yes	Yes.
2	No	Yes	No	No	No	No	Yes	Yes.
Operation on appendix.								
1	Yes	Yes	Yes	No	Yes	Yes	Yes	Yes.
2	No	Yes	No	No	No	Yes	Yes	Yes.

[4] The anesthesia for surgical procedures on the intestines are considered in another publication.

CONCLUSIONS

On the evidence of data recorded above it can be stated with assurance that—

(*a*) Despite widespread employment of local and spinal anesthetic agents, inhalation anesthesia is still the method of choice in many instances.

(*b*) Whenever inhalation anesthesia is employed, the interest of all Medical Department units will be served if the occasion is utilized for instruction purposes to the same degree now being employed in training personnel in the administration of local and spinal anesthetic agents.

(*c*) As in the case of other specialties, appropriate entries should be made in pertinent records when the individual concerned is considered to be a qualified anesthetist, the entry revealing the type or types of anesthesia which the individual is qualified to administer.

(*d*) The personnel division of the bureau will appreciate greatly being supplied with copies of the entries just mentioned.

THE PULPLESS TOOTH; A LOCAL AND A CONSTITUTIONAL PROBLEM

By H. R. DELANEY, Lieutenant Commander, Dental Corps, United States Navy

The fact that the teeth are parts of the body and that dental infection can influence health has been rediscovered periodically (1, 2, 3). During the past 30 years the metastatic effects of dental disease have received universal attention, and so much enthusiasm has attended the endeavor to eliminate nidi in the dental area that the teeth often have seemed more important because they can be removed than because of their value as functioning organs (4). Happily, the school demanding " 100 per cent vitality " that has flourished during the orgies of tooth extraction and that has been championed by various groups has suffered defections to the more conservative ranks.

It would be worse than foolish to refuse to view very seriously the dangers of diffusion of infection from within and about the teeth, but, it must be conceded that many teeth have been condemned upon wholly presumptive evidence and that benefits from their extraction have been nil. The paradoxical situation of the problem of dental infection, still more or less evident in a disposition to suspect the teeth of being the focal points of virtually all of the pathologic conditions with which the human race is afflicted (5, 6, 7), may have as its next absurdity a return to the form of nihilism that existed prior to the early years of the current century. Indeed, we understand that even now a number of diseases formerly associated with focal infection are attributed to the etiologic factor of nutritional imbalance (8). In view of the confusion of opinion that exists concerning the diagnosis and treatment of dental disease, particularly with respect to diseases of other parts, and while the pendulum appears to be swinging from the radical toward the conservative point of view, this seems a favorable time to discuss one of our problems, viz, the pulpless tooth, in the light of our present knowledge. Our discussion will have to do mainly with diagnostic considerations favoring or contraindicating retention and conservative treatment.

Despite all that has been written about it, the pulpless tooth is one of the most perplexing problems with which we have to deal. It commands the attention of all who are concerned with the prevention and treatment of disease. In approaching this problem, we must correlate all of our diagnostic methods and findings. Above all, we must take into consideration certain fundamentals, among which that of the state of health of the patient is paramount.

To look upon dental infection in a detached manner—that is, without some thought of it in terms of general health—is as unreasonable as it would be so to regard diseases of the heart, brain, or other highly important organs, although disease of the teeth usually

does not manifest itself so directly by interruption of constitutional function. To prevent the impression that our generally inclusive remarks prelude a discussion of all types of dental pulp disease, it must be said that we mean to refer particularly to the form of infection with which the *S. viridans* group is frequently identified and with which metastases are most often associated.

A tremendous amount of space in the professional journals has been given to the subject of focal infection, with many reports of clinical and laboratory observations upon the effects of dental disease. Whether infected teeth demand attention because bacteria from them elect to localize in certain other parts (9) or whether the infection exerts an allergic influence which favors the development of distant lesions (10) is not of paramount importance. The fact that must be borne in mind is that dental disease, in common with disease of all other parts of the body, is constitutional, even though the defensive mechanism may confine its symptoms to an area of very small limits. Therefore, in considering the prognosis of the pulpless tooth, our first criterion is the state of health of the patient.

The fact of old age, or recognition of general debility, or the knowledge that a patient has in chronic form any one of the so-called degenerative diseases (11) prohibits the treatment of pulpless teeth for the simple reason that the prognosis of the teeth themselves is bad and, therefore, that the teeth would become or continue to be an adverse health factor. It is interesting to note in this connection that certain of our radiographic diagnostic points, to be described later, appear to indicate the unfavorable local reaction of teeth in the presence of the conditions mentioned. With improvement in health, the prognosis of the teeth also may improve. We have seen conditions that apparently contraindicated dental treatment respond favorably following a period of constitutional treatment that improved the health factor.

"Health" is a rather loose relative term. Commonly we regard a person as being in good health when he exhibits no striking objective symptoms of disease and does not complain. In the study of dental disease, and particularly of periapical disease, attempts have been made to ascertain more exactly the state of health of patients (12, 13, 14). These attempts in part have been based upon comparison of findings with normals which were assigned narrow limits of variation. However, the apparently arbitrary normals were derived from a considerable number of examinations of persons in whom no evidence of infection could be found. The methods have been criticized not only because of the exactness of the assumed normals but also because no direct relationship could be shown between the various

indexes and the local conditions, e. g., periapical infection undergoing treatment. It is known that some laboratory findings can be interpreted quite specifically (15). If we view the problem of dental infection as a problem of constitutional health, the evaluation of laboratory findings must be given weight regardless of specific relationship.

The much mentioned medical-dental cooperation perhaps can be promoted most advantageously through a comprehensive study of dental disease from the health angle, and there is some reason to expect that the unnecessary loss of many teeth can be prevented (16). As a rule, the removal of parts of the body that are suspected of being infected is not undertaken so long as there is a chance of cure. Why should teeth be extracted needlessly?

One of the leaders in dental research (17) who had much to do with popularizing the extraction of all pulpless teeth now is satisfied that the pulpless tooth, if correctly treated, is no more a menace to health than are many teeth having vital pulps (18). It is impossible to close all of the avenues through which bacteria can enter the blood stream, such as the intestinal and genito-urinary tracts and the nose and its accessory sinuses. The defensive mechanism must be relied upon to maintain health balance if we can eliminate known areas of infection.

The pulpless tooth is not, necessarily, a dead tooth (19). It usually is surrounded or nearly surrounded by living supporting tissue, closely related to it structurally, and the condition of this peridental tissue has much to do with indicating the prognosis of the tooth. Without going into methods of treating pulpless teeth, let it be said that we can seal pulp canals to prevent reinfection or diffusion of infection from them (20). If the patient is in good health and if immediate extraction is not indicated to relieve acute or chronic symptoms, we seek to determine by clinical (physical), radiographic, and microscopic examination whether or not the pulpless tooth will respond favorably to treatment.

In the clinical examination we consider stability of the tooth as a partial indication of the condition of the investing tissues, we appraise the future usefulness of the tooth and its esthetic desirability, we search carefully for evidence of chronic infection such as fistulae, we examine the relationship of the tooth to others in approximation or occlusion, and we seek to elicit vital reaction by electrolytic and thermal tests, etc.

By means of microscopic examinations of smears and cultures obtained from the pulp canal and apical area (21) (15), we seek to identify the type of infection present in some cases and observe progress under treatment. Generally, three consecutive negative

smears are regarded as satisfactory evidence of sterility before sealing the pulp canal. If smears demonstrate infection, there seems to be no point in culturing unless identification of bacteria is desired. There is, of course, the possibility that some organisms may be brought out in culture when smears appear negative, but, our routine has been to depend upon stained smears. No method of bacteriologic examination of material from teeth can be regarded as absolutely conclusive, due to the impracticability of reaching every organism that may be present.

By radiographs we obtain information concerning the condition of the alveolar bone, the peridental membrane, and the apical cementum of the tooth. The pictures must have good detail and should be taken from more than one angle. If rarefaction has occurred, we study the films with a view to determining whether we are dealing with a condensing or rarefying type of osteitis (22), i. e., whether or not there is a natural limitation of the process. We are interested also in the extent of bone destruction, although this is regarded as being less important than the type.

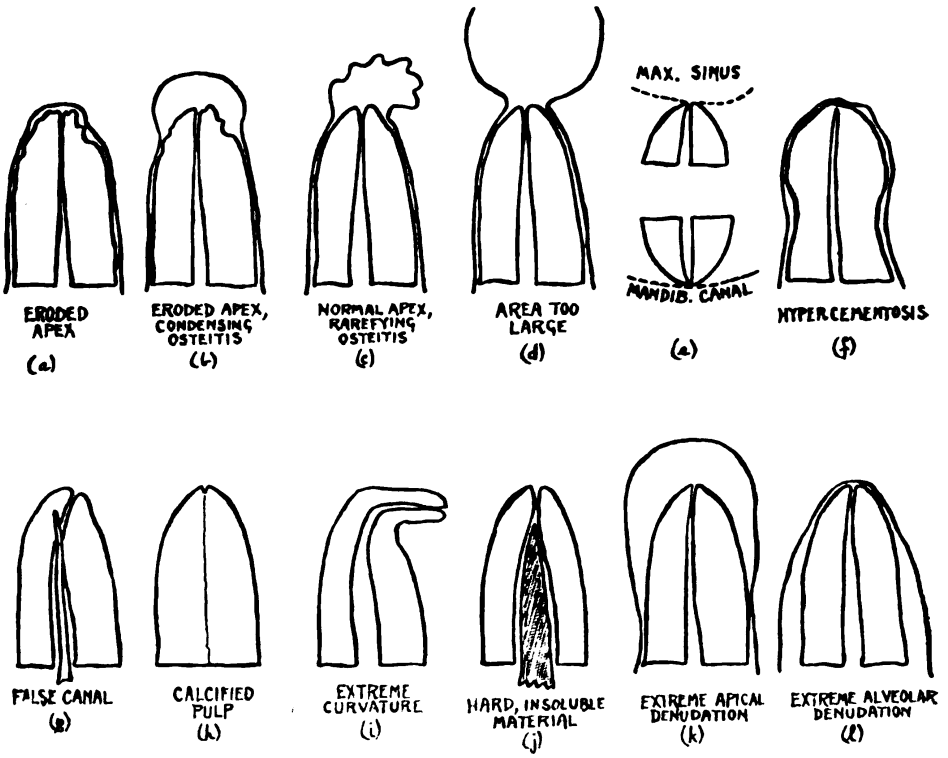
If the apical cementum is eroded (Figs. (a) and (b)), conservative treatment will be hopeless unless apicoectomy is to follow in an attempt to prolong the usefulness of the tooth. If the apical cementum shows no erosion, the prognosis further depends upon the condition of the periapical bone. A rarefying type of osteitis (Fig. (c)) prohibits treatment even though the apex of the tooth be in good condition. This type of area is local evidence of poor health, even though physical symptoms may be lacking. If an area of rarefaction is very large (Fig. (d)), treatment is not indicated unless all other factors are exceptionally favorable and retention of the tooth is especially desired. In this case the condition should be observed frequently for some time following the completion of treatment. The best method of managing large cystic areas is to reflect the overlying gum and periosteum from the necks of the teeth, upward or downward as the case may be, remove the overlying alveolar plate, and enucleate the cyst. Small cysts may be punctured and drained through the pulp canals after preliminary disinfection of the canals.

Close proximity to the maxillary sinus or mandibular canal (Fig. (e)) absolutely contraindicates treatment of pulpless teeth, for, the danger of direct extension of infection into these areas is great. Traumatic injury that may result from treatment of such teeth may lead to chronic sinusitis or to osteomyelitis of the mandible.

Certain local conditions so increase the technical difficulties of treating pulpless teeth that they are, as a rule, contraindications. (Figs. (f) (g) (h) (i) (j).) Among these conditions may be in-

cluded hypercementosis which occludes the apical ends of infected teeth, false canals that may lead to perforation of the root, complete calcification of the pulp, extreme curvature of roots, and obstructions that can be removed only with great difficulty.

Extreme denudation of a root, either from the apical or marginal alveolar end, contraindicates treatment on account of difficulty in disinfecting the rarefied area or because of loss of stability of the tooth. In advanced marginal denudation there is a possibility that infection may extend to the apex.



Diagramatic representation of contraindications against pulp canal treatment. (Delaney)

It should be realized that all radiolucent apical areas are not positive indications of abscess or of nonvitality, as excessive biting stress may cause such appearances without devitalizing the pulp. Conversely, an infected pulp canal may appear normal in the radiogram. Teeth with doubtful pulp conditions must be examined for response to thermal or electrical stimuli. Errors in diagnosis have been made because of superimposition of shadows of the anterior palatine and mental foramina upon the images of the root apices. In such cases, study of the appearance of the pericementum and lamina dura in relation to the root should remove doubt.

To recapitulate briefly, the treatment of pulpless teeth is justified when the health of the patient is good, when the apical cementum

is uneroded, when bone rarefaction (if present) is of favorable type, when there is no danger of extension of infection into or traumatic injury of adjacent structures, when the supporting alveolar process has not been extensively lost, and when the technical difficulties are not unduly great. Many pulpless teeth meet these requirements.

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SPEED OF VISUAL PERCEPTION

By P. RICHMOND, Lieutenant Commander, Medical Corps, United States Navy, and E. C. EBERT, Lieutenant Commander, Medical Corps, United States Navy

Speed of visual perception does not receive the consideration it deserves in our examinations of visual acuity. Our present methods of examination for form and color allow unlimited time in recognizing letter forms and color. Candidates with slight myopia or hyperopic astigmatism frequently disguise these defects by squinting if time is not limited. In order to arrive at some conclusions as to a reasonable time necessary to recognize letters and colors, a series of tests were made at Canacao Naval Hospital with—

1. Flash letters.
2. Projection lantern.
3. Camera box shutters.

Tests were performed on men with 20/20 vision. Ages varied from 19 to 43 years.

The flash letter tests consisted of individual illuminated letters. This system was soon eliminated on account of slight afterglow when flashes were made rapidly.

The projection lantern methods of flashing letters on a silver screen at various speeds proved to be unsatisfactory due to variation in thickness of film emulsion making standard illumination difficult.

The camera box shutters with standard illuminated chart (as illustrated) proved satisfactory on account of uniformity as well as simplicity. This method is so simple and readily performed, at the same time accurately limiting the time a candidate has to recognize letters or colors, that it is suggested that some such method be adopted in the routine naval examinations for visual acuity.

The tests as made at the United States naval hospital, Canacao, P. I., totaled about 300; therefore, only a preliminary report can be made. Before standard time for recognition of letters or form can be recommended, a larger series of tests should be made and the average adopted.

The camera shutter method consists of placing the candidate behind a camera with shutter intact but with the lens removed. The candidate is instructed in setting as well as tripping the shutter, as shown in the illustration (A). The examiner places a standard Snellen's letter behind a reflector with 10-foot candle illumination, as shown in the illustration (B). The candidate reads the letters seen during the time the camera shutter opens. While the shutter is closed, the examiner replaces the card with another letter behind the reflector. Each candidate was tested with 10 letters for each eye before the speed of the camera was changed. The following speeds were utilized: 0.5 second, 0.2 second, 0.1 second, 0.04 second, 0.02 second, 0.01 second.

METHOD OF TESTING SPEED OF VISUAL PERCEPTION. (RICHMOND AND EBERT)

Camera shutters may be placed directly before illuminated letters and tripped by examiner. This method has a decided disadvantage in that candidate is not always at attention when the shutter is tripped. The candidate is prepared when he, himself, trips the shutter. The noise made by the shutter action prevents the candidate from opening it surreptitiously.

Observations Made

I. Per cent letters read with daylight.

At 0.5 second each eye averaged 83.5 per cent, both eyes 99 per cent.
At 0.2 second each eye averaged 43 per cent, both eyes 82 per cent.
At 0.1 second each eye averaged 25 per cent, both eyes 54 per cent.
At 0.04 second each eye averaged 17 per cent, both eyes 39 per cent.
At 0.02 second each eye averaged 8 per cent, both eyes 19 per cent.
At 0.01 second each eye averaged 6 per cent, both eyes 15 per cent.

II. Per cent letters read with electric light.

At 0.5 second each eye averaged 83 per cent.
At 0.2 second each eye averaged 26 per cent.
At 0.1 second each eye averaged 6 per cent.
At 0.04 second each eye averaged 0 per cent.
At 0.02 second each eye averaged 0 per cent.

III. Color perception speed (red, green, blue, yellow, and orange circles, one-half inch in diameter, painted with water colors on white cards.)

At 0.5 second each eye averaged 75 per cent.
At 0.2 second each eye averaged 51 per cent.
At 0.1 second each eye averaged 52 per cent.
At 0.04 second each eye averaged 42 per cent.
At 0.02 second each eye averaged 46 per cent.
At 0.01 second each eye averaged 46 per cent.

Summary and Conclusions

1. A time factor should be incorporated in our visual acuity examinations.

2. 0.5 second observation time is a good standard unit for each eye.

3. Visual speed by daylight is decidedly faster than by artificial illumination.

4. Visual speed ranged from 0.5 second per letter to 0.01 second among candidates examined.

5. A few candidates with slow color perception apparently compensated with rapid form perception.

6. Color perception is not diminished as rapidly as form perception by shortening the period of observation.

7. Form speed of perception for both eyes is twice that of monocular vision.

CLINICAL NOTES

PRIAPISM

With Report of a Case

By P. T. Crosby, Lieutenant Commander, Medical Corps, United States Navy, and R. P. Parsons, Lieutenant Commander, Medical Corps, United States Navy

Priapism is a comparatively rare condition, only about 200 cases having been described in literature. Each case presents a nice, sometimes a quite perplexing, diagnostic problem because of the large number and wide variety of possible causes. In many cases no cause can be assigned.

French (1) lists some 30 different causes, arranged according to age groups, but several of them seem rather questionable as the specific causes, and many others mentioned are not associated with the persistent painful erection of true priapism but with merely the frequently recurring and transitory type of erection which is relieved by intercourse and other simple conservative measures.

A more significant classification appears in Hinman's (2) excellent monograph of 1914. He gives two large divisions: (1) Nervous and (2) local mechanical causes, grouping under the first class: (*a*) Lesions producing ascending peripheral stimuli, (*b*) various lesions of the upper dorsal cord producing direct stimuli, and (*c*) descending cerebral stimuli; under the second class are grouped such local mechanical causes as (*a*) thrombosis, (*b*) hemorrhage and hematoma, (*c*) tumors of the penis, (*d*) inflammatory swellings and œdema of the penis.

Whatever be the cause of the priapism, if the erection has existed for two or more days, it will persist despite any change in the original and underlying cause, since a thrombosis of the corpora will have developed, and this in itself will sustain the erection. So the immediate treatment must be directed at the removal of the thrombi.

McKay and Colston (3), in 1928, reported on a new and rather unique and simple method for this; and in the same year Van Duzen (4) reported a case due to inflammatory swelling of the penis which was treated successfully by intravenous mercurochrome. The method of McKay and Colston will be described presently. Both of these methods were used in the case to be reported. Our case thus offered an opportunity to evaluate these methods and moreover im-

presses us as a case worth reporting because of the diagnostic difficulties it presented at first and also because we were able to observe the patient over a sufficiently long period to form some conclusion as to ultimate prognosis.

Case Report

P. G. B., fireman, third class, aged 18, was admitted on board the U. S. S. *Relief* from the U. S. S. *Utah* on October 10, 1932, because of a painful erection, then of three days' duration, and which was not relieved by intercourse or by other ordinary conservative measures (cold applications, sedatives, etc.).

Patient is an exceptionally well developed and healthy looking youth of exemplary habits who has served 17 months without admission to the sick list. The past medical history contained no items of importance or relevance except for enuresis every night until age of 12; then once every few months for about a year, but no further occurrence of enuresis during the past five years. No history of venereal infection. Heterosexual experiences were desired and indulged in very infrequently, about once or twice a year.

About four months before admission, while on leave, he awoke with painful erection. This persisted about 48 hours, then gradually subsiding. No urinary or other symptoms accompanied this attack and patient felt well and noticed no symptoms for about two weeks.

Early in July, 1932, he again began to be troubled with painful erections which would appear each morning upon awaking and would persist for several hours, sometimes until noon.

These attacks recurred every morning until October 8, when the usual erection, instead of subsiding in a few hours, persisted and caused patient's transfer to the *Relief* on October 10.

The physical and neurological examination was entirely negative except for chronically infected tonsils and the very painful priapism. The prostate was small, soft, nontender. Prostatic fluid could not be obtained because of the priapism. Temperature on admission was 99.6° F. and ranged irregularly during the next 10 days between 99° F. and 102.5° F. The urine showed nothing of note except for a rare pus cell and epithelial cell. The blood picture gave no indication of leukemia and was as follows: Red blood count, 4,710,000; white blood count, 17,500; hemoglobin, 90 per cent; band forms, 4; segmented, 76; lymphocytes, 19; eosinophiles, 1; platelets, 291,000.

X ray showed no radiographic evidence of spinal pathology; and spinal fluid was entirely negative. The blood Kahn was negative.

The day following admission the white count had dropped to 13,700, but as the pain from the priapism was increasing we decided to use the procedure described by McKay and Colston (3).

After anesthetizing the skin and Buck's fascia with 1 per cent novocaine over a point on the lateral surface of the penis about midway of the shaft, a large aspirating needle was inserted into the corpus cavernosum. About 30 cubic centimeters of black bloody fluid (liquified clots) were aspirated with a luer syringe. As this fluid was withdrawn the erection relaxed and reached a point of about semierection. Normal saline solution was then injected but as this was withdrawn with difficulty, only about 10 cubic centimeters were injected. As the saline was injected the penis became more erect, and again became slightly more flaccid as the saline was withdrawn. Local anesthesia was found to be unsatisfactory in this case because of the extreme pain caused by the necessary movement and manipulation of the penis in order to hold it steady

during the aspiration. This and the extreme anxiety and apprehension of the patient, decided us to use spinal anesthesia for any subsequent aspirations. Within two hours after the aspiration the erection became again just as complete and as painful as ever.

On the following day the procedure was repeated, using spinal anesthesia. This was found to be more satisfactory. About 50 cubic centimeters of blackish blood were aspirated and the erection was reduced to a state of about one-third complete. A few hours later it returned to a point of about two-thirds erection, where it remained for the next two days.

By October 15 the white count had dropped to 11,300, but the penis was still two-thirds erect. Intravenous mercurochrome, 10 cubic centimeters of 1 per cent solution, was then given and the same dose repeated the next day, October 16. By the 17th the penis was about one-fourth erect. The same dose of mercurochrome was repeated on the 19th, and on the 20th the penis was pendulous though large and had a fibrous feeling. White count on October 20 was 9,400.

On October 22 the temperature, which had not risen above 100° F. for three days, started to rise and reached 103.4° F. by evening and 104.8° F. by the evening of October 23. This rise was not accompanied by any change in the penis, nor by other symptoms, except malaise and headache. Temperature remained near 104° F. until the 25th, then dropping and reaching normal by the 27th, where it has since remained. The febrile course and leucocyte counts directed our attention to a focal infection which may have been playing at least a remote causative rôle in this picture.

A very gradual softening of the penis then progressed, reaching the normal consistency about December 1, no return of erections having meanwhile occurred.

On November 10 patient had a nocturnal emission without erection and this was repeated on each of the three succeeding nights. This directed our attention to a possible prostatic infection. The prostate was still soft and small but rather tender and the fluid showed numerous pus cells, epithelial cells and considerable débris. Endoscopic examination on November 15 showed congestion and edema throughout the prostatic urethra of a degree which all but obscured the landmarks. Prostatic massage was then given every five days for one month.

On December 5 the tonsils were again noted to harbor an extensive infection of long duration, and tonsillectomy was done on that date. It is of course impossible to say with certainty that the favorable course of events that followed the tonsillectomy was due to that procedure, but within a few days following the operation the patient felt very much better in general, and by December 15 he was considered fit for duty, normal erections having then begun to return (after a period of some eight weeks of impotence.)

Summary and Conclusions

1. There is presented a case of priapism, believed to be due to chronic prostatitis, in turn thought to be the result of chronic tonsillar infection.

2. The aspiration technique described by McKay and Colston is distinctly helpful, and although the relief obtained by that procedure in this particular case was only temporary and partial, and although spinal anesthesia in this case had a decided advantage over

local anesthesia, it is supposed that in other cases more complete and lasting results may be obtained by aspiration and saline washing; also that in other cases local anesthesia may prove quite satisfactory.

3. The use of intravenous mercurochrome may have been of some benefit in this case, and it is thought that whatever value it might have in the treatment of priapism will depend upon its effect on the local infection responsible for the priapism.

REFERENCES

(1) French, H.: Index of Differential Diagnosis; 4th ed., p. 656.

(2) Hinman, F.: Review of literature on 170 cases; Annals of Surgery, 60: 689, December, 1914.

(3) McKay, R. W., and Colston, J. A. C.: Priapism, a new method of treatment; Journal of Urology 20: 121, February, 1928.

(4) Van Duzen, R. E.: Priapism, with report of a case; Journal of Urology 20: 497, October, 1928.

REPEATED FRACTURE OF THE SKULL

By Lucius W. Johnson, Captain, Medical Corps, United States Navy

This case is of interest because of the unusual sequence of events. The patient suffered a fracture of the skull with brain injury which required operative interference. He was eventually returned to duty and, after a few days, suffered a second fracture of the skull in another area.

Case Report

A marine, age 21, was injured in an automobile accident on December 5, 1931. He showed small lacerations over the bridge of the nose and the right cheek and was somewhat dazed. He received emergency treatment at the Los Angeles General Hospital and was then allowed to go to his home. The next morning he had a headache and, during the day, it became progressively worse. Toward evening he became unconscious, so he was placed in an automobile and brought 120 miles to the San Diego Naval Hospital, where he arrived about 30 hours after his injury.

On admission he was drowsy, disoriented, and complained of very severe headache. The pupils were equal and reacted normally; the pulse was 44 to 52. There was clinical evidence of a fracture of the base of the skull in the anterior fossa. He received the usual course of treatment for head injuries and improved greatly but the condition fluctuated. Headache, slow pulse, and drowsiness recurred on several occasions, but could always be controlled by intravenous hypertonic solutions until January 1, when he complained of very severe headache and gradually became comatose. This time, it was not possible to control the symptoms by hypertonic fluids. The coma became more and more profound and was accompanied by slowing pulse and stertorous breathing.

Eye ground examination showed definite papillitis of about 6 diopters in each eye with marked venous engorgement. There were no localizing signs to indicate involvement of any particular cerebral area. The spinal fluid remained clear and with normal pressure.

Operation was done January 5 under local anesthesia. We desired access to both frontal lobes with the least possible scarring, so an incision was made across the vertex from ear to ear. The anterior portion of the scalp was then peeled forward, as far as the supraorbital ridge, giving an excellent exposure of the frontal region. A large trephine opening was made over each frontal lobe. The brain bulged into the opening as soon as the dura was incised, so the anterior horn of each lateral ventricle was tapped and 100 cubic centimeters of 50 per cent glucose was slowly injected intravenously. A careful search was made for hemorrhage, abscess, or other lesion, but none was found. The trephine buttons were replaced and the wound closed.

During the operation the patient became conscious and conversed with us. His mind seemed entirely clear. After operation he was greatly improved. The next day his pulse was 72 and his headache was entirely relieved. Eye ground examination 48 hours after operation showed the papillitis subsided and conditions almost normal. On the seventh postoperative day his headache returned and his pulse dropped to 50. This was relieved by hypertonic glucose and there were no more headaches. He continued to improve and, about seven weeks after operation, he was returned to duty in excellent condition. The scar was invisible, being entirely within the hairy scalp.

Comment.—In passing, it is interesting to note that this was just the type of case in which many authorities say that operation is useless. Progressively increasing intracranial pressure with clear spinal fluid under normal pressure and with no response to intravenous hypertonic fluid gives a most hopeless prognosis. But it is the second case in which I have seen decompression and tapping of the ventricles produce complete relief.

Repeated fracture.—Three days after this man went to duty he was returned to the hospital in an unconscious condition. The health record stated that, during an altercation, he had been knocked down and his head had struck on the concrete floor. There was a contusion over the right parietal and frontal bones. The pupils were dilated and the reflexes generally hyperactive. The spinal fluid was clear and normal in pressure. The X-ray showed a linear fracture of the right parietal and temporal bones about 6 inches long which was not present when he was admitted the first time.

His symptoms were controlled by the usual methods of treatment and he was returned to duty two months later, in excellent mental and physical condition.

THE CANCER SERVICE AT THE UNITED STATES NAVAL HOSPITAL, BROOKLYN, N. Y.

With Data for the Fiscal Year 1932

By C. W. Stelle, Lieutenant (Junior Grade), Medical Corps, United States Navy

On January 29, 1929, a cancer service, as a separate unit coming under the supervision of the surgical service, was established at the United States naval hospital, Brooklyn, N. Y. Prior to this date all cases of malignant disease were assigned to the various services as their diagnosis indicated, for instance, carcinoma of the stomach—to the gastrointestinal wards; carcinoma of the tongue—to the nose and throat department; etc.

Relations were established with the Memorial Hospital, New York, N. Y., where treatment with radium and the Röntgen ray was available. X-ray treatment is also given by the X-ray department at the naval hospital.

Since that time the number of cancer cases treated has steadily increased, due chiefly to two main factors: Firstly, because of the availability of radium therapy at the Memorial Hospital, where, as stated above, a liaison is maintained with this hospital, also the United States Veterans' Administration has elected to send to the Brooklyn naval hospital, malignant cases coming from the Atlantic seaboard from Maine to Florida. Secondly, because the survivors of the Spanish-American and World War are now reaching the so-called cancer age.

TABLE 1

Dates	New admissions	Readmissions	Total
From Jan. 29, 1929, to June 30, 1929	67	26	93
Fiscal year ending June 30, 1930	132	117	249
Fiscal year ending June 30, 1931	178	204	482
Fiscal year ending June 30, 1932	182	352	534
Grand total for 3½ years	559	699	1,258

Table 1 shows that a total of 534 cases was admitted during the fiscal year ending June 30, 1932, making an average of 44.5 patients per month. The number of readmissions was necessarily high because in the successful management of a cancer case, it is imperative that follow-up examinations be made at frequent intervals to check the results of treatment.

TABLE 2

CARCINOMATA (101)

Region:	Cases
Head and neck—	
Nose	4
Lip	16
Tongue	8
Ear	2
Cheek	9
Ethmoid sinus	1
Maxilla	1
Eyelid	4
Parotid gland	1
Tonsil	2
Larynx	4
Pharynx	2
Neck	1
Genito-urinary system—	
Kidney	1
Bladder	8
Prostate	2
Testicle	3
Region—Continued.	
Gastrointestinal system—	
Esophagus	2
Stomach	6
Small intestines	1
Ascending colon	1
Transverse colon	1
Rectum	10
Liver	3
Pancreas	1
Chest—	
Bronchus	1
Mediastinum	1
Lung	3
Miscellaneous—	
Axilla	1
Buttock	1

SARCOMATA (12)

Region:	Cases	Region—Continued.	Cases
Maxilla	3	Abdomen	5
Humerus	2	Vertebra	1
Tibia	1		

MISCELLANEOUS (21)

Tumor:	Cases	Tumor—Continued.	Cases
Papilloma, nose	1	Chondroma, chest	1
Keratosis, lip	3	Osteitis deformans	1
Papilloma, eyelid	1	Leukoplakia	1
Melanoma	3	Chronic mastitis	1
Xanthomatous neurofilroma	1	Thymoma	1
Hodgkins' disease	7	Grand total	134

Table 2 presents data on 134 new admissions during the fiscal year in which the diagnosis was established by biopsy, X-ray findings, or clinical signs and symptoms. In addition, there were 48 cases in which there was no evidence of malignant disease or the diagnosis could not be correctly established. In this latter group biopsy could not be done, autopsy was not permitted, or patient left the hospital before being studied sufficiently.

In checking the age of the patients, it was found that cancer appears to be occurring at younger ages than usually quoted. In the foregoing series, an inoperable neurogenic sarcoma was discovered in a man aged 31, and an adenocarcinoma of the rectum in another patient also aged 31.

The great majority of cases, with exception of those with lesions of the head and neck, were quite far advanced when first seen and therefore the end result of treatment has not been gratifying. Perhaps the increase of cancer in younger persons indicates that our methods of diagnosis are steadily growing better, and that in time this early diagnosis will make for better prognosis.

After complete examination, microscopical diagnosis is made either by aspiration biopsy or ordinary biopsy in all possible cases. Aspiration biopsy is especially useful in metastatic growths where the primary lesion can not be located; for example, bone sarcomas and tumors of the prostate.

In testicular tumors Aschheim-Zondek urine tests are made to determine the teratomatous nature of the growth and also for evidence of metastasis in the event castration has been done prior to admission.

Recently, a complete photographic unit has been established at this hospital, and now all visible lesions are photographed.

Treatment consists of surgery alone; radiation, external and interstitial; or surgery and radiation combined. In addition, Coley's toxins have been used in some cases of sarcoma. In consideration of

treatment, cases are classified as operable, inoperable, early, and far advanced.

Many of the patients are taken to Memorial Hospital, for consultation. This assures confirmation in diagnosis and additional suggestions for treatment, and at the same time affording excellent opportunity for the medical officer in charge to attend clinics and staff conferences there.

Memorial Hospital has always welcomed the naval hospital staff, and much information is to be gained thereby. At present a medical officer is completing a six months' course of study there, and another is scheduled to relieve him.

SUMMARY

1. A short résumé is given of the cancer service at the United States naval hospital, Brooklyn, N. Y.

2. Radium therapy for malignant diseases is available at this hospital due to established relations with Memorial Hospital, New York City.

3. Many cases where Röntgen-ray therapy is indicated are now being treated by the X-ray department at the Brooklyn naval hospital.

4. There were 534 admissions during the fiscal year ending June 30, 1932, an increase of 52 over the previous fiscal year.

5. One hundred and eighty-two patients were seen for the first time. Statistics are given showing the types and locations of 134 proven cases.

6. The remaining 352 admissions represent readmissions for follow-up examination and necessary additional treatment, for patients seen first either in the last or previous fiscal years.

REPORT OF 2,434 SPINAL ANESTHESIAS, WITH SPECIAL REFERENCE TO POSTOPERATIVE PULMONARY COMPLICATIONS IN 1,000 CASES

By F. R. MOORE, Lieutenant, Medical Corps, United States Navy

During the past five years spinal anesthesia has become the anesthesia of choice for all procedures below the diaphragm at the United States naval hospital, Mare Island, Calif. Our figures show that 2,434 spinal anesthesias have been given without an anesthetic death. (Table I.)

The main reasons for the popularity of spinal anesthesia here are: (1) Complete relaxation; (2) low percentage of postoperative complications; (3) ease of administration; (4) low cost.

Postoperative pulmonary complications are said to be more frequent following spinal anesthesia than after inhalation anesthesia.

Brown and Debenham (1) in an unselected group of 812 cases found that in 388 spinal anesthesias 7.4 per cent developed pulmonary complications, while in 472 inhalation anesthesias only 1.3 per cent developed respiratory trouble. Lyons (2) found pulmonary complications developed in only 0.95 per cent following 6,619 inhalation anesthesias.

In reviewing 1,000 unselected, consecutive spinal anesthesias given on the surgical service of the United States naval hospital, Mare Island, Calif., between March, 1931, and September, 1932, 13 cases, or 1.3 per cent, developed postoperative pulmonary complications. None of these complications proved fatal. In 500 cases, during 1931, in which carbon dioxide-oxygen mixture [1] was not administered, respiratory complications developed in 8 cases, or 1.6 per cent. In a series of 500 cases, in which carbon dioxide-oxygen mixture was used routinely, 5, or 1 per cent, had pulmonary complications. (Table II.)

The technique followed at this hospital is similar to that of Labat. The patient receives morphine sulphate, one-fourth grain, and atropine sulphate, one one-hundred-and-fiftieth grain, 15 minutes before going to the surgery, in addition to other routine procedures. Procaine crystals in two and one-half grain amounts are placed in small cotton stoppered ampules and autoclaved. Before introduction of the spinal puncture needle, the procaine crystals are shaken out into a sterile medicine glass. The spinal fluid—4 cubic centimeters for lower abdominal, and 8 cubic centimeters for upper abdominal work—is allowed to drip into the medicine glass. The procaine crystals are rapidly dissolved. The solution is then drawn into a 10 cubic centimeter syringe and the subarachnoid injection is made slowly.

Ephedrine sulphate, three-eighths of a grain, is given subcutaneously, just prior to the spinal tap.

The patient's blood pressure is checked every few minutes and if severe hypotension develops, the head of the table is lowered and the dose of ephedrine sulphate is repeated. Low blood pressure has seldom caused anxiety.

As the skin sutures are being placed, carbon dioxide-oxygen is administered to the patient for five minutes. After the patient reaches his room the nurse instructs him to take several deep breaths every hour. Patients are also turned frequently from side to side. If increased amounts of bronchial secretion are observed following an upper abdominal operation, carbon dioxide-oxygen is administered for five minutes every hour, until the quantity of secretion approaches normal.

[1] Carbon dioxide 5 per cent and oxygen 95 per cent.

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TABLE I

	Spinal anesthesias	Deaths		Spinal anesthesias	Deaths
Upper abdominal	128	0	Kidney and ureter	28	0
Appendectomy	444	0	Testicle, vesicles, and bladder	110	0
Hernia	710	0	Miscellaneous	93	0
Rectal	807	0			
Orthopedic	86	0	Total	2,434	0
Prostate	28	0			

TABLE II

	500 cases with carbon dioxide-oxygen				500 cases without carbon dioxide-oxygen			
	Cases	Pneumonia	Bronchitis	Collapse	Cases	Pneumonia	Bronchitis	Collapse
Upper abdominal	30	0	0	1	34	0	0	1
Appendectomy	80	0	0	1	131	0	1	2
Hernia	159	1	1	0	133	0	1	1
Orthopedic	29	0	0	0	37	0	0	0
Rectal	136	0	1	0	105	0	1	1
Kidney and ureters	12	0	0	0	8	0	0	0
Prostate	9	0	0	0	8	0	0	0
Testicles and bladder	20	0	0	0	29	0	0	0
Miscellaneous	25				15			
Total	500				500			

SUMMARY

1. Report of 2,434 spinal anesthesias without a death attributable to anesthesia.

2. In a series of 1,000 consecutive spinal anesthesias postoperative pulmonary complications developed in but 1.3 per cent of the cases.

3. In 500 instances in which carbon dioxide-oxygen was not used immediately after operation, pulmonary complications developed in 8, or 1.6 per cent, of the cases.

4. In 500 in which carbon dioxide-oxygen was used, only 5, or 1 per cent, of the cases, developed pulmonary complications.

REFERENCES

(1) Brown, A. L., and Debenham, M. W.: J. A. M. A., 99: 209, July 16, 1932.
(2) Lyons, M.: Surg., Gynec., and Obst., 55: 162, August, 1932.

TREATMENT AND PREVENTION OF HEAT CRAMPS

By W. E. EATON, Commander, Medical Corps, United States Navy

Under Notes and Comments in the July, 1932, issue of the Naval Medical Bulletin, pages 378 and 379, there appeared a comment upon an article on this subject by Dr. D. M. Glover.

I wish to state that I have tried on several occasions the administration of salt to men suffering from heat cramps, as well as in those

cases suffering from prolonged exposure to high temperatures, such as is found in the vicinity of the so-called superheaters on boilers, evaporator rooms, etc.

Sometime during 1926 there came to my notice an article by a French (as I recall) medical practitioner, in which was advocated the administration of salt in water, in the strength of 5 grams of sodium chloride to the gallon, to those suffering from heat cramps.

While attached to the U. S. S. *Utah*, in the summer of 1926, several men suffering from heat cramps were given copious draughts of warm salt solution of the above strength, with noticeable benefit. In 1927, while attached to the U. S. S. *Florida*, men frequently applied for treatment for mild cramps. These usually were men who stood duty in the dynamo room, where the steam engines which operated the dynamos were so placed that they were on the opposite side of the compartment from the access hatches, ventilation was practically nil, and a very high degree of heat prevailed. These men perspired profusely, wore as little clothing as possible, and suffered from the heat. These men gained relief by drinking salt water.

On one occasion a number of men were required to work in shifts, upon repairs adjacent to the superheater of one of the boilers. Ten men were overcome during a short period of time and were brought to the sick bay in varying degrees of cramps, the younger men seeming the more affected, and several suffering severely. All of these men were required to drink freely of warm salt water solution, with benefit. Even after the cramps had passed off, these men found the warm salt water solution desirable.

As the boiler rooms and dynamo rooms of the *Florida* were hot, it became our practice to advise, during long hot steaming watches in the Tropics, that salt be added to the drinking water consumed by the men on watch therein, and that the water be warm, rather than cold. Buckets of warm water were brought to the dispensary for the addition of sodium chloride, in the proportion of 5 grams to the gallon, about 15 grains to the tumbler. (It will be recalled Doctor Glover recommended 16 grains to the drink.) Apparently, as the result of this administration of salt in the drinking water, no further cases of cramps appeared.

On the U. S. S. *New York* last year, a man was brought to the sick bay suffering very severely from heat cramps. His abdominal muscles and those of his legs, especially the lower legs, were frequently in a persistent spasmodic contraction, the toes were drawn up in painful flexion, as were occasionally the fingers of the hands. He experienced considerable pain during the contractures. He had been exposed to high temperature. He was requested to drink very freely of warm salt water solution while resting in bed. At the

end of about three hours he experienced no more cramps and the following day complained only of soreness in the tissues affected. He believed that the salt water relieved him and this appeared to be true.

After these experiences and observations, there is no doubt in my mind that the addition of salt to the drinking water consumed by those exposed to extremes of heat, attended by profuse perspiration, has a markedly beneficial effect, not only in preventing cramps, but also in relieving the conditions once they occur. Five grams to the gallon seems adequate and is not unpleasant to drink. Even larger proportions seem to offer no objection, for in the solutions we made up only a rough measure, like a teaspoonful, was used in supplying water for the men on watch, this solution often being made by themselves, I found out later, once the benefits became known to the men.

Doctor Glover's suggested treatment is well worth recognition.

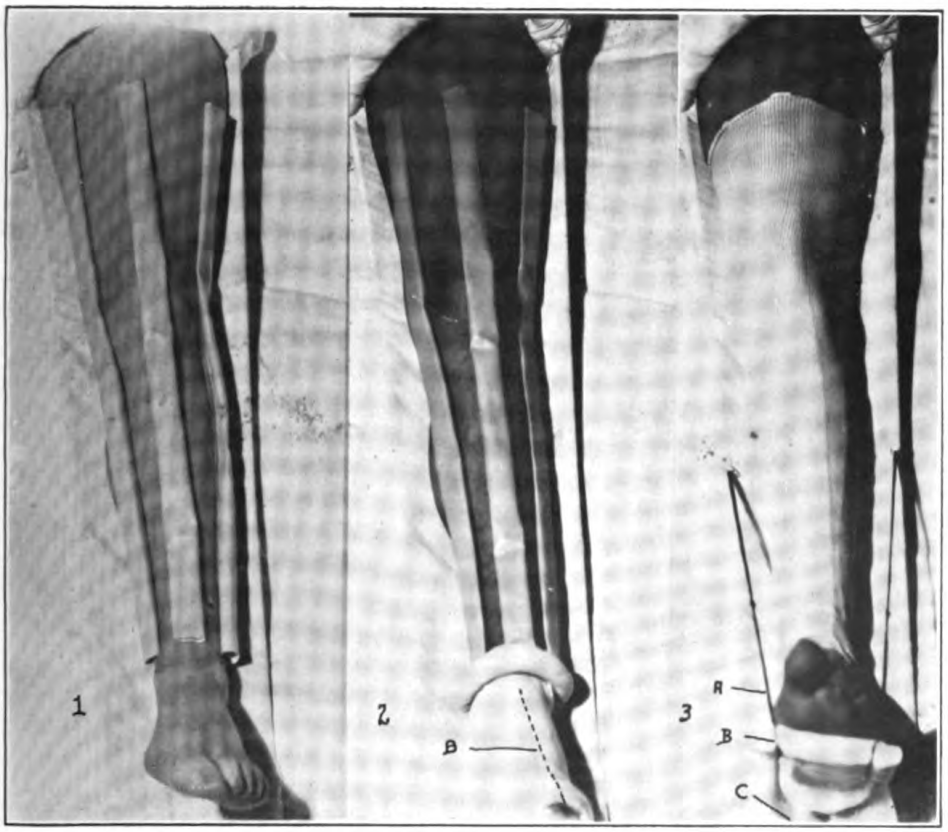
A SIMPLE TRACTION METHOD FOR THE TREATMENT OF UNCOMPLICATED FRACTURES OF THE FEMUR

By J. W. Shumate, Lieutenant (Junior Grade), Medical Corps, United States Navy

In a fracture experience with almost two score fractures of the femur, approximately one-third were so situated and uncomplicated that skin traction was considered the treatment of choice. The fractures treated in this manner were in the middle third or at the junction of the middle and upper third of the femur, and as there was no displacement, good union without deformity, was assured. The method of skin traction selected for the treatment of the first eight such cases was the commonly used one of combining the Thomas hip splint with zinc oxide adhesive plaster. This method, as was expected, gave the desired results as far as union was concerned, but minor complications, such as skin irritation and impaired circulation were encountered so frequently that a search was made for a method of treatment that would remedy these conditions.

The succeeding seven cases in the series were treated in the manner described below. Skin irritation and impaired circulation were done away with so completely that the method used is recommended.

Method.—Three-inch widths of adhesive plaster were cut to such a length that they would extend from a point just above the ankle to the site of the fracture. The strips were then folded into tubes with the adhesive surface outward and placed along the extremity as shown in the accompanying figure, 1 anteriorly, 1 posteriorly, and 1 each laterally. Stockinette, in length sufficient to reach from the toes to the site of the fracture, was rolled up the leg over the adhesive. The lower portion of the stockinette was rolled over the



SIMPLE TRACTION METHOD FOR UNCOMPLICATED FRACTURES OF FEMUR

1, Shows three of the four adhesive tubes in place; 2, shows the method of applying stockinette, (B) shows the anterior line to be cut; 3, shows the method in use, (A) the rubber bands to support foot rest (B), (C) shows position of the spreading block. (Shumate.)

foot, while the ankle was padded with cotton wadding and bandaged. The portion of stockinette covering the foot was cut, anteriorly and posteriorly, to a point just above the ankle and the two ends secured by tying or sewing, thus forming a loop. Into this loop a spreading block was inserted to which traction weights were attached. Countertraction, as in other methods was obtained by the use of the Thomas hip splint. The danger of footdrop was overcome by an apparatus made of padded basswood placed under the ball of the foot and held in place by moderately strong rubber bands secured to the frame of the splint. This apparatus was not only successful in accomplishing the desired result, but proved very comfortable because it allowed some motion of the ankle.

In the opinion of the writer this method of treating uncomplicated fractures of the femur possesses some advantages over the more commonly accepted adhesive method. It is simpler, more comfortable, and it alleviates the complications referred to above.

Stockinette traction is not new, but the writer has found no record of its having been used in the manner described.

NAVAL RESERVE

MEDICAL CORPS

APPOINTMENTS, FOURTH QUARTER, 1932

Name	Rank	Appointed
Capone, Angelo J	Lieutenant (junior grade), MC-V(G)	Aug. 26, 1932
Kelley, Everett E	do	Aug. 17, 1932
McKee, John S	do	Aug. 23, 1932
Brown, James R	do	Sept. 22, 1932
Norfleet, Ashley C	do	Sept. 20, 1932
Anderson, Lang W	Lieutenant, MC-V(S)	Oct. 13, 1932
Bork, Albert L	Lieutenant (junior grade), MC-V(G)	Oct. 21, 1932
Hutson, Thomas W	Lieutenant commander, MC-V(S)	Oct. 3, 1932
Ingrish, George A	Lieutenant (junior grade), MC-V(G)	Oct. 28, 1932
McIntyre, D. R	Lieutenant (junior grade), MC-V(S)	Nov. 8, 1932

PROMOTIONS

Name	From—	To—
Cook, Enos Paul	Lieutenant, MC-F	Lieutenant commander MC-F.
Hammond, Thomas V., jr.	do	Do.
Stedman, Harold E	Lieutenant (junior grade), MC-F	Lieutenant, MC-F.
Cason, William M	Lieutenant (junior grade), MC-V(G)	Lieutenant, MC-V(G).
Koehler, Joseph S	do	Do.
MacKenzie, Pierce	do	Do.
Fonde, George F	do	Do.
Mayne, Roy M	Lieutenant (junior grade), MC-F	Lieutenant, MC-F.
Parish, Benjamin D	Lieutenant, MC-V(G)	Lieutenant commander, MC-V(G).
Robertson, Charles G	Lieutenant (junior grade), MC-V(G)	Lieutenant, MC-V(G).
Ruddisill, Clarence A	do	Do.
Smith, William R	Lieutenant (junior grade), MC-F	Lieutenant, MC-F.
Sybilrud, Hjalmer W	Lieutenant, MC-V(G)	Lieutenant commander, MC-V(G).

TRANSFERS

Name	From—	To—
Dougall, John P	Lieutenant commander, MC-V(G)	Lieutenant commander, MC-F.
Shearer, Beryl C	Lieutenant (junior grade), MC-V(G)	Lieutenant (junior grade), MC-V(S)
Stephens, Doran J	do	Lieutenant (junior grade), MC-F.
Waterman, Julius L	Lieutenant commander, MC-F	Lieutenant commander, MC-V(G).

DENTAL CORPS

APPOINTMENTS, FOURTH QUARTER, 1932

Name	Rank	Appointed
Redden, Richard F	Lieutenant (junior grade), DC-V(G)	Oct. 20, 1932

NOTES AND COMMENTS

POSTGRADUATE INSTRUCTION FOR MEMBERS OF THE MEDICAL DEPARTMENT OF THE NAVY IN THE CALENDAR YEAR 1932[1]

As evidence of the developments in postgraduate instruction of members of the Medical Department of the Navy the following data relative to specialty personnel needs, the number of persons qualified in each subject, and the number under instruction on January 1, 1933, are of interest:

MEDICAL OFFICERS

Specialty	Number required	Number qualified Jan. 1, 1932	Number qualified Jan. 1, 1933	Number under instruction Jan. 1, 1933
Internal medicine	100	78	93	6
Aviation medicine	48	44	43	0
Pediatrics	32	8	6	1
Clinical laboratory	72	34	38	2
Pathology	26	10	17	5
General surgery	80	57	66	2
Orthopedic surgery	20	15	17	3
Urology	40	35	41	1
Gynecology and obstetrics	54	39	44	1
Eye, ear, nose, and throat	55	49	51	3
Bronchoscopy	15	11	15	1
Radiology	40	31	32	2
Neurology and psychiatry	50	29	29	2
Chemical defense	12	18	26	1
Field service	22	10	12	4
Public health	23	12	12	1
Submarine and deep-sea diving	16	13	12	4
Physical therapy	13	8	10	1
Chemistry	7	7	7	0
Supply depot and industrial war college	10	8	9	2
War plans	20	9	8	2

DENTAL OFFICERS

Denture prosthesis	**34**	**9**	**11**	**4**
Minor oral surgery	**28**	**4**	**6**	**2**
Crown and bridge	**6**	**6**	**6**	**0**
Root canal therapy	**8**	**2**	**2**	**0**
Ceramics	**6**	**0**	**0**	**0**
Periodontia	**8**	**1**	**1**	**0**
Light therapy	**4**	**1**	**1**	**0**
Research, nutrition, and diet	**3**	**1**	**1**	**0**

[1] From the division of personnel, Capt. K. C. Melhorn, Medical Corps, United States Navy, in charge.

HOSPITAL CORPS MEN

Specialty	Number required	Number qualified Jan. 1, 1932	Number qualified Jan. 1, 1933	Number under instruction Jan. 1, 1933
Medical aviation	75	67	65	11
Commissary	65	104	89	31
Deep diving for rescue vessel duty	6	1	1	0
Dentistry	190	188	170	30
Electrocardiograph	30	26	31	5
Embalming	40	32	38	8
Laboratory	185	149	173	44
Medical field service	150	23	27	1
Physiological methods for diving and chemical defense	6	2	2	2
Physical therapy	50	22	34	18
Prosthetic dentistry	36	32	33	1
Property and accounting	338	302	275	40
Röntgenology	75	83	84	27

NURSES

Dietitians	35	23	22	0
Instructors	20	19	32	0
Physical therapy	27	26	29	3
Anesthetists	18	18	21	0
Laboratory	15	23	23	0
Röntgenology	10	3	3	0
Metabolism and cardiograph	30	3	4	0

By comparing these data with those published in the Naval Medical Bulletin of January 1, 1932 (p. 33), it will be noted that with few exceptions the specialty requirements for trained personnel are being met in increasing degree. So steady has been the progress in some fields that the anxieties of previous years no longer exist.

The program by which this gratifying situation has been effected was initiated in 1920 by Surgeon General Braisted. Revised and expanded in the succeeding administrations of Surgeon Generals Stitt and Riggs it has brought results that should be a source of pleasure and gratification to its originator and supporters. So well does the system now function that it can be predicted with emphasis that, if the yearly increments of personnel trained for the specialties are maintained at the rates effected in the past two years, complete success for the program is assured. Certain it is that from the standpoint of providing opportunities for postgraduate study the Medical Department of the Navy has every reason to take pride in the position it has gained among the Federal services.

TANNIC ACID TREATMENT OF BURNS

D. M. Glover, writing under the title "Six Years of Tannic Acid Treatment of Burns," in Surgery, Gynecology, and Obstetrics, May, 1932, gives an evaluation of the results in 310 cases treated by this method.

According to the author, the data available showed that with this method of treatment, as compared with other forms of treatment, there was a marked reduction in mortality; the incidence of septic complications was lower; there was no essentially beneficial effect on epithelization; the treatment was comfortable for the patient; and the method was practical and economical.

Stress is laid on the importance of regarding all burns as serious until proved to the contrary and that the patient receive the benefit of prompt and efficient treatment.

The general outline of treatment described by the author (somewhat modified from that originally announced by Davidson in 1925) is essentially as follows:

The patient is immediately put to bed under a lighted cradle with sufficient lights to maintain a temperature of approximately 85° F.

If suffering much pain, morphine is administered hypodermatically.

Any dressings previously applied are removed, and if oil or grease has been applied, remove quickly and gently with ether.

Vesicles that have formed are opened with sterile scissors and the outer layers of epithelium which are loose are peeled off. Any vesicles or bullæ forming in the course of the first day are treated in same manner.

Gross dirt is removed, but thorough scrubbing with an antiseptic is considered unnecessary and too productive of shock.

Just as soon as the burned area is prepared, it is sprayed with an aqueous solution of tannic acid (5 per cent for children and 10 per cent for adults). The solution should be freshly perpared from the dry powder, as tannic acid changes to gallic acid in a few days after exposure to light and air, after which it should not be used. The spray from an ordinary atomizer should be satisfactory. As tannic acid corrodes metal, atomizers made entirely of glass and rubber will be found more satisfactory.

The tannic-acid solution is sprayed over the burned area every hour for the first 24 hours. Very extensive burns may require spraying every 10 or 15 minutes during the first few hours in order to get the earliest possible coagulation. The spraying is discontinued if, at the end of 24 hours, the burned areas are well coagulated and dry, and no further local treatment is necessary for several days. During this time the burned surface is left exposed to the air. Burned skin surfaces are prevented from coming in contact with each other or with the bed by means of sterile towels or sheets.

The tannic-acid spray is not used on the face on account of possible injury to the eye. In this area and in other areas where it is difficult to treat with a spray, a 5 per cent tannic-acid jelly (traga-

canth base about the consistency of ordinary lubricant jelly) is employed. Petrolatum is used about the eyelids, nostrils, and lips, and in burns of the perineum, about the genitalia and anus to prevent them from becoming uncomfortably dry and stiff.

To combat dehydration, a most important feature in the early stage of the burn, an adequate amount of fluid is administered. In case of vomiting or in an extensive burn, physiological salt solution or 5 per cent dextrose solution intravenously, by rectum, or by hypodermoclysis is indicated. Care should be taken not to overload the circulation with fluid and produce edema.

According to Glover, the true toxic stage of a burn is over within 48 hours when efficient tannic-acid treatment is started early. Following this the initial rise in temperature drops to normal or only slightly above. Then a secondary toxic stage may develop at the end of the first week, due to exudate forming beneath the crusts, as evidenced by a rise in temperature, sense of fluctuation beneath the crusts, malaise, and sometimes delirium. For this condition continuous Dakin's solution dressings are recommended to loosen up the coagulum. These dressings are kept continually moist and changed daily until the coagulum is all off and the granulating areas are clean and ready to graft or are healing with epithelial islands not destroyed by the burn. In using these dressings, the normal skin should be protected with petrolatum to prevent the Dakin's solution from causing a dermatitis.

The treatment of chemical burns, after first flushing the affected area with large quantities of water or salt solution to dilute the chemical remaining, is essentially the same as those caused by heat. Some deep burns from concentrated alkalies or acids and small, deep electrical burns are not affected by tannic acid, as the superficial tissues are already coagulated.

CARBARSONE THERAPY IN AMEBIASIS

On the basis of a study of 42 patients, under excellent conditions of control afforded by a protozoölogic survey of new admissions to San Quentin Prison, Calif., Norman David, Herbert Johnstone, and L. L. Stanley, writing in the November, 1932, number of the American Journal of the Medical Sciences, state:

> Our findings generally confirm those reported by Reed (J. Am. Med. Assn., 1932, 98; 189). All but 1 of a group of 42 cases of amebiasis observed for three or more months after oral treatment with carbarsone were successfully cleared of *Entamœba histolytica* (as far as careful stool examinations could reveal), without toxic effect and without hospitalization. It is our opinion that carbarsone offers a relatively nontoxic, satisfactory, and cheap means of inaugurating therapy in amebiasis. It seems to be quite as effective as emetin, and

is to be preferred as an initial therapeutic agent, because of the definite evidence of pathologic damage from emetin in the doses necessary for clinical effectiveness. Acetarsone and chiniofon do not show as satisfactory an effect in amebiasis as we have found for carbarsone. In severe cases of amebiasis the recommended dosage of carbarsone (5 grams in 10 days) may not be enough. Repetition of the course of therapy, supplemented with carbarsone retention enemata, is then indicated. Only after this would one seem to be justified in incurring the risks involved in emetin therapy.

STUDIES ON RINGWORM FUNGI WITH REFERENCE TO PUBLIC-HEALTH PROBLEMS

L. Bonar and A. D. Dreyer, in the American Journal of Public Health for September, 1932, report the laboratory findings in connection with studies on ringworm with reference to public-health problems which have been carried on at the University of California during the past three years. These investigators found that:

Attempts to grow fungus on sound clean wood have yielded only negative results.

Fungus may grow readily on floor material that is covered by a coating of slime or algal growth.

The complete killing of *Trichophyton interdigitale* borne in skin scales, by 1 per cent sodium hypochlorite solution requires a time period of one hour or longer.

Thermal death point studies on spore suspensions, cultures grown in fabrics, and on material imbedded in skin scales show complete killing of the fungus in 10 minutes time at 75° C., or lower in some cases.

The efficiency of the fungicidal action of standard power laundry practice is shown to vary with the nature of the fabric handled and with the temperature applied to the different materials. The standard practice for white cotton fabrics shows a good margin of safety, while that employed for woolens and colored fabrics is doubtful.

The application of standard "dry cleaning" solvents to these fungi, either growing in fabric, or imbedded in skin scales, is shown to have a negligible killing action, in exposures of one to two hours.

IS THE INCREASE OF CANCER REAL OR APPARENT?

Writing under the title above, Dr. M. T. Macklin, in the American Journal of Cancer for September, 1932, summarizes the results of a study of cancer based on the statistics of Canada, as follows:

Cancer is increasing, and it is increasing particularly in the age group over 60. The reason for its increase is not that it is occurring at progressively younger ages, or attacking larger percentages of the younger population. Despite its increase, deaths are fewer from all causes now than they were. We have won more ground from the ravages of infectious disease than we have lost to those disorders which are dependent upon inherent qualities in the chemical and physical make-up of the individuals. Cancer is increasing because, by preventive methods, there has been created a larger population to grow old;

and having grown old, they are kept from dying of those ills from which they formerly suffered. With each increase in the warfare against preventable diseases, there will be an increase in the ravages from cancer, for with each victory there is created a greater available population to die from that disease.

These conclusions are based upon the statistics of Canada. There is strong ground for believing that a similar analysis of the statistics of any other country would lead to the same conclusions. It is true that there may be racial differences in immunity to cancer, but the conclusion here reached will probably prove universal, namely, that excellent public-health measures and high cancer rates are inseparable, at least for the present. Those who point to the low cancer rates existing among primitive peoples, and who state that cancer is a disease of modern civilization, neglect to call attention to the fact that preventive medicine is itself a triumph of modern civilization.

SPINAL MANOMETER READINGS

Spinal manometers use both mercury and water and various authors give the readings in millimeters of mercury or of water. It is frequently desired to compare the two readings, for example, how many millimeters of water are equivalent to 14 millimeters of mercury. To obtain this, multiply the mercury reading by 13.5—the specific gravity of mercury, or in case of a water reading, divide by 13.5.

THE HENRY S. WELLCOME MEDAL AND PRIZE FOR 1933

This year the prize, which consists of a gold medal and $500, will be awarded to the writer of the best paper on one of the following subjects:

1. The Value of Studies in Health and Sanitation in War Planning.

2. Military Medicine as a Specialty. How can a Knowledge of it be Promoted in the Medical Profession in Civil Life and in the Reserves?

The competition is open to all medical officers, former medical officers, acting assistant and contract surgeons of the Army, Navy, Public Health Service, Organized Militia, United States Veterans' Administration, United States Volunteers, and of the Reserves of the United States, and commissioned medical officers of foreign military services.

Each competitor must furnish five copies of his competitive paper and can not write on more than one subject. Papers must not be signed with the true name of the writer, but are to be identified by a nom de plume or distinctive device. They must be forwarded to the secretary of the Association of Military Surgeons of the United States, Army Medical Museum, Washington, D. C., so as to arrive at

a date not later than September 1, 1933, and must be accompanied by a sealed envelope marked on the outside with the fictitious name or device assumed by the writer and inclosing his true name, title, and address. The length of the essays is fixed between a maximum of 10,000 words and a minimum of 3,000 words. The envelope accompanying the winning essay or report will be opened by the president of the association and the name of the successful contestant announced by him.

The winning essay or report becomes the property of the association and will be published in the Military Surgeon. Should the board of award see fit to designate any paper for "first honorable mention" the executive council may award the writer life membership in the Association of Military Surgeons, and his essay will also become the property of the association.

THE DIVISION OF PREVENTIVE MEDICINE

O. J. MINK, Captain, Medical Corps, United States Navy, in charge

TOXIC EFFECTS OF ARSENICAL COMPOUNDS EMPLOYED IN THE TREATMENT OF SYPHILIS IN THE UNITED STATES NAVY—1931 [1]

By O. J. MINK, Captain, 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 numbers of doses of arsphenamine and neoarsphenamine, etc., administered. A separate account of every case in which ill effects are noted is also required.

Previous articles dealing with the information obtained from these reports were published in the September, 1925, January, 1927, January, 1929, July, 1930, October, 1931, and October, 1932, numbers of the United States Naval Medical Bulletin and covered the subject up to and including the calendar year 1930. The present article supplements these and is in the interest of continuous published statistical information relative to the ill effects experienced from the administration of arsenicals in the United States Navy. This installment takes into consideration the doses of arsenical compounds administered in the treatment of syphilis during the calendar year 1931 and includes some comparative figures from the experience of previous years.

It is the desired intention to present to Navy's arsenical experience by years, and we have endeavored to continue the classification of reactions in the manner adopted by Commander J. R. Phelps, Medical Corps, United States Navy, in his fourth contribution, which appears in the Division of Preventive Medicine section of the Naval Medical Bulletin of July, 1930.

The "reactions" or ill effects encountered during the calendar year 1931 are as follows:

Encephalitis hemorrhagica		0
Vasomotor phenomena (shocklike, nitritoid, anaphylactoid, etc.)		30
Arsenical [2] dermatitis and its complications		28
Exfoliative	20	
Erythematous	3	
Eczematous	2	
Erythemato-macular	1	
Erythemato-papular	1	
Papulo-vesicular	1	

[1] Case histories of cases herein described are given as reported by medical officers having cognizance of the case.

[2] The term "arsenical" has been substituted for the term "exfoliative."

Acute yellow atrophy of the liver	0
Jaundice	1
Acute renal damage	0
Ulcerative enteritis	0
Polyneuritis	0
Aplastic anemia	1
Herxheimer reactions	0
Jarisch-Herxheimer	1
Reactions of minor importance, including those in which there were insufficient data for classification	1
Total	62

The following table shows the number of treatments with the various arsenical compounds administered by naval medical officers in the treatment of syphilis during the 6-year period 1925–1930, the calendar year 1931, and the totals for the seven years 1925–1931, with the number and ratio of fatal and nonfatal toxic effects and of deaths to the number of doses administered:

	Number of doses administered	Mild reactions	Severe reactions	Fatal reactions	Total reactions	Ratio of reactions to doses—1 reaction to—	Ratio of deaths to doses—1 death to—
6-year period, 1925–1930:							
Arsphenamine	29,036	25	13	1	39	745	29,036
Neoarsphenamine	399,870	235	82	22	339	1,180	18,176
Sulpharsphenamine	7,963	3	3	0	6	1,327	0
Tryparsamide	13,798	1	0	0	1	13,798	0
Miscellaneous	58	0	0	0	0	0	0
Total	450,725	264	98	23	385	1,171	19,597
1931:							
Arsphenamine	1,353	0	0	0	0	0	0
Neoarsphenamine	95,442	34	28	0	62	1,539	0
Silver arsphenamine [1]	32	0	0	0	0	0	0
Sulpharsphenamine	1,157	0	0	0	0	0	0
Tryparsamide	5,927	0	0	0	0	0	0
Total	103,911	34	28	0	62	1,676	0
7-year period, 1925–1931:							
Arsphenamine	30,389	25	13	1	39	779	30,389
Neoarsphenamine	495,312	269	110	22	401	1,235	22,514
Silver arsphenamine [1]	32	0	0	0	0	0	0
Sulpharsphenamine	9,120	3	3	0	6	1,520	0
Tryparsamide	19,725	1	0	0	1	19,725	0
Miscellaneous	58	0	0	0	0	0	0
Total	554,636	298	126	23	447	1,241	24,115

[1] First administrations during the year 1931.

Referring to the above table, the ratios of reactions and deaths to doses administered for 1931 are: Reactions, 1 to 1,676, and deaths, 0 to 103,911. The averages for the 6-year period 1925–1930 are: Reactions, 1 to 1,171, and deaths, 1 to 19,597. The averages for the 7-year period 1925–1931 are: Reactions, 1 to 1,241, and deaths, 1 to 24,115.

The following table shows the number of fatal and severe reactions which followed the administration of 495,312 doses of neoars-

phenamine during the seven years 1925–1931 and the ratios of deaths and severe reactions to the total number of doses administered:

Complication	Number of fatal reactions	Ratio of deaths to total number of doses administered	Number of severe reactions with recovery	Ratio of severe reactions to total number of doses administered	Total	Ratio of combined fatal and severe reactions to total number of doses administered
Hemorrhagic encephalitis	10	1 to 49,531	0	0 to 495,312	10	1 to 49,531
Vasomotor phenomena	4	1 to 123,828	28	1 to 17,690	32	1 to 15,479
Arsenical dermatitis	4	1 to 123,828	75	1 to 6,604	79	1 to 6,270
Acute yellow atrophy of the liver	1	1 to 495,312	0	0 to 495,312	1	1 to 495,312
Jaundice	0	0 to 495,312	6	1 to 82,552	6	1 to 82,552
Acute renal damage	0	0 to 495,312	0	0 to 495,312	0	0 to 495,312
Ulcerative enteritis	0	0 to 495,312	0	0 to 495,312	0	0 to 495,312
Polyneuritis	0	0 to 495,312	1	1 to 495,312	1	1 to 495,312
Aplastic anemia	3	1 to 165,104	[1] 3	1 to 165,104	6	1 to 82,552
Herxheimer	0	0 to 495,312	0	0 to 495,312	0	0 to 495,312
Total	22	1 to 22,514	[2] 113	1 to 4,383	135	1 to 3,669

[1] Includes 1 case diagnosed as "agranulocytosis" which was not included in previous reports.
[2] Includes 2 border-line reactions carried in previous reports as "mild" but which can be classed as "severe."

Deaths charged to the administration of arsenical compounds in the treatment of syphilis during the past 13 years were recorded as follows:

Year	Arsphenamine	Neoarsphenamine	Kind not specified	Total	Year	Arsphenamine	Neoarsphenamine	Kind not specified	Total
1919	2	0	1	3	1927	1	4	0	5
1920	1	1	0	2	1928	0	6	0	6
1921	3	1	0	4	1929	0	3	0	3
1922	0	4	0	4	1930	0	3	0	3
1923	0	1	0	1	1931	0	0	0	0
1924	1	2	0	3					
1925	0	2	0	2	Total	8	31	1	40
1926	0	4	0	4					

During the 7-year period 1925–1931 there were 16.30 times more neoarsphenamine than arsphenamine injections administered. Many factors must be considered in an attempt to determine the advantages or disadvantages of arsphenamine as opposed to neoarsphenamine, and sufficient data for a comprehensive comparison are not available. However, the following figures from the Navy's experience with 30,389 doses of arsphenamine and 495,312 doses of neoarsphenamine are of interest, because they may show a possible reaction expectancy regardless of whether the ill effects are due to the drug, technique, idiosyncrasy, or some other cause.

Total number of doses of arsphenamine	30,389
Total reactions, arsphenamine	39
Total number of doses of neoarsphenamine	495,312
Total reactions, neoarsphenamine	401

Possible number of reactions from arsphenamine as compared with the number of doses of neoarsphenamine administered	635
Possible ratio of arsphenamine reactions to neoarsphenamine reactions (using the possible figure 635)	1.59 to 1
Possible ratio of neoarsphenamine reactions to arsphenamine reactions (using the possible figure 635)	0.63 to 1

Hemorrhagic Encephalitis

No cases of hemorrhagic encephalitis chargeable to the effects of arsenicals occurred during the year 1931.

Cases suggesting border-line relationship between acute hemorrhagic encephalitis and the other forms of acute poisoning by arsenical compounds used in the treatment of syphilis.—During the year 1931 no reactions occurred which would seem to fall under this heading.

Proportion of deaths from hemorrhagic encephalitis to the number of patients treated for syphilis and to the numbers of doses of arsenical compounds administered.—During the year 1931 there were 1,353 doses of arsphenamine and 95,442 doses of neoarsphenamine administered with no deaths due to hemorrhagic encephalitis. During the 7-year period, 1925–1931, there were 30,389 doses of arsphenamine administered with 1 death due to hemorrhagic encephalitis; during the same period of time there were 495,312 doses of neoarsphenamine administered with 10 deaths due to hemorrhagic encephalitis, giving a ratio of 1 death to 49,531 doses administered.

During the 7-year period 1925–1931 there were 9,120 doses of sulpharsphenamine and 19,725 doses of tryparsamide administered, with no deaths from any cause referable to the arsenical compound.

For past years the actual number of persons treated for syphilis is unknown for many reasons, the most important of which is the fact that each year there are many persons treated who began their treatment in previous years. These persons are not necessarily readmitted to the sick list because of their current treatment.

In order to obtain information as to the number of persons in the naval service who had syphilitic histories, the Bureau of Medicine and Surgery revised N. M. S. Form A and included an annual section which directed that a census be taken of all health records of service personnel on December 31, 1931, to determine how many persons had a history of syphilis; how many persons were treated for syphilis during the year with an arsenical compound, heavy metal, or other mixed treatment; and how many persons were treated during the year with an arsenical compound for diseases other than syphilis. Included in the instructions were directions to make a similar census regarding treatment accorded during the

year to persons other than active-service personnel (Veterans' Administration beneficiaries, supernumeraries, etc.).

As a result of this census the following tables show a recapitulation of data obtained. The first table shows a recapitulation of number of persons treated for syphilis and the number of persons treated for diseases other than syphilis; the second table separates the diseases other than syphilis and shows the number of persons treated for each disease:

	United States Navy and Marine Corps	All others	Total
Average strength, calendar year 1931	112,767		112,767
Average strength, December, 1931	102,099		102,099
Syphilis census (persons)	12,004		12,004
Number of persons treated for syphilis:			
Silver arsphenamine	0	4	4
Arsphenamine	382	0	382
Neoarsphenamine	7,819	4,701	12,520
Sulpharsphenamine	149	104	253
Tryparsamide	45	2,559	2,604
Mixed courses	280	192	472
Total persons treated for syphilis	8,675	7,560	16,235
Number of persons treated for diseases other than syphilis:			
Neoarsphenamine	57	1,031	1,088
Sulpharsphenamine	14	52	66
Total persons treated for diseases other than syphilis	71	1,083	1,154
Grand total	8,746	8,643	17,389

Disease	Number of persons treated for diseases other than syphilis		
	United States Navy and Marine Corps	All others	Total
NEOARSPHENAMINE			
Provocative Kahn	21	11	32
Chancroid (provocative)	9	0	9
Acne	10	0	10
Yaws	7	1,018	1,025
Gonococcus infection	3	0	3
Malaria	2	0	2
Psoriasis	2	0	2
Furunculosis	2	0	2
Arthritis, gonococcus	1	1	2
Gangosa	0	1	1
Total	57	1,031	1,088
SULPHARSPHENAMINE			
Gonococcus infection, epididymis	14	0	14
Yaws	0	51	51
Vincent's angina	0	1	1
Total	14	52	66
Grand total	71	1,083	1,154
NOTE.—Neoarsphenamine—most probably locally applied for Vincent's infection (not included in the above figures)	343	7	350

Vasomotor Phenomena

In previous articles on this subject covering the years up to and including the year 1930, 168 cases reported by medical officers have been grouped as examples of vasomotor phenomena following the intravenous injection of arsenical compounds. Of the 30 cases reported during the year 1931 and which have been grouped under this heading, 27 were mild reactions and 3 were severe reactions, No reaction resulted in death.

Deaths.—As previously mentioned, there were no deaths (vasomotor phenomena) during the year 1931 which followed the intravenous injection of an arsenical compound. The ratio of deaths (vasomotor phenomena) to the number of doses of neoarsphenamine administered during the 7-year period 1925–1931 is 1 death to 128,828 doses administered. The ratio of deaths (vasomotor phenomena) to the number of doses of arsphenamine administered during the 7-year period 1925–1931 is 1 death to 30,389 doses administered.

Nonfatal Cases of Arsphenamine and Neoarsphenamine Poisoning Classed as Vasomotor Phenomena (1931)

Arsphenamine.—No reactions were reported as having resulted from the intravenous injection of 1,353 doses of arsphenamine.

Neoarsphenamine.—The 30 reactions reported during the year 1931 occurred at different times and on different ships and stations. These 30 reactions are presented below with comparative data given in tabular form:

Key	Course of treatment	Injection	Dose in grams, neoarsphenamine	Onset in hours after injection	Duration or reported time of recovery	Apparent first symptoms
12	First	First	0.3	7	About 12 hours	Chill, nausea, vomiting, headache, followed by fever.
16	do	do	.3	15½	24 hours	Fever, headache, general malaise.
17	do	do	.3	6	About 16 hours	Headache, pains in joints, malaise, fever.
19	do	do	.3	9	About 48 hours	Headache, pains in abdomen, vomiting, fever.
5	do	Second	.45	(1)	About 4 hours	Fainted, body cold and clammy, heart action regular but weak, followed by nausea, vomiting, headache.
13	do	do	.45	6	3 days	Slight headache, fever. Later, fever only.
4	do	Third	.6	2½	7 days	Headache, generalized pain and aching of body, face flushed, eyes injected, skin hot and dry, rash over chest, abdomen, and arms, fever.
9	do	do	.45	4	5 days	Fever, chills and rigor, headache, macular rash.
15	do	do	.6	2	8 hours	Fever, vomited once, herpes about lower lip appeared 2 days later.
29	do	do	.3	8	7 days	Fever, chills, followed by anorexia, nausea, perspiration, and fine tremors. (Under treatment for psoriasis.)
1	do	Fourth	.45	½	About 24 hours	Violent abdominal cramps and severe chills, vomiting intermittently and purged in the next 3 hours, considerable bleeding from the gums.

[1] Immediate.

Key	Course of treatment	Injection	Dose in grams, neoarsphenamine	Onset in hours after injection	Duration or reported time of recovery	Apparent first symptoms
[2] 20	First	Fifth	0.6	Several.	3 hours	Chills, headache, fever.
[2] 21	do	Sixth	.3	Several.	do	Do.
[2] 22	do	Seventh	.45	Several.	5 hours	Chills, headache, fever (pronounced).
14	do	Eighth	.75	½	do	Chills, lips and face became quite cyanotic, weak pulse, muscular tremblings.
2	Second	First	.2	6½	(?)	Severe urticaria rash over face, trunk, and arms.
18	do	Third	.6	([1])	3 hours	Dizzy, nauseated, very weak, increasing pulse rate.
6	Third	First	.45	([1])	4 hours	Weak, pale, pulse weak and rapid, slight reflexes, when assisted from table he fainted.
10	do	do	.45	([1])	2 days	Severe headache, pains in joints and muscles, chill, fever, rapid pulse.
7	do	Second	.3	11	24 hours	Headache, nausea, vomiting, restlessness.
3	do	Third	.6	¼	9½ hours	Cramps in abdomen, headache, joint pains, face flushed.
11	do	Sixth	.6	24	8 days	Pain in upper abdomen with definite tender area above and to the left of the umbilicus.
8	do	Seventh	.45	6	1 hour	Fever, severe cramps in abdomen, severe headache.
23	Fourth	First	.45	½2	½ hour	Itching of skin over right antecubital fossa, later urticaria on flexor surfaces of both arms, accompanied by dizziness and faintness.
[3] 24	do	Second	.45	½2	do	Do.
[3] 25	do	Third	.45	½2	do	Slight itching around site of injection, no urticarial or constitutional symptoms.
[4] 26	do	Fourth	.6	1	([5])	Flushing of body, felt warm, noted wheals on arms and body, slight blurring of vision.
[4] 27	do	Fifth	.6	½	1 hour	Eyes, face, and body flushed, profuse sweating, urticaria soon noted over body and extremities, pulse weak and rapid, complained of weakness and a blurred vision, nausea.
30	do	do	.6	¼	([6])	Difficulty in breathing, fainted, very cyanotic, cold and clammy, pulseless in peripheral arteries, heart sounds weak and very rapid, many moist, bubbling râles in both sides of chest. (Under treatment for yaws.)
28	do	Sixth	.15	([1])	2 hours	Eyes injected, flushing of skin, urticaria quickly appeared, eyelids became edematous, pulse imperceptible, vision blurred, weak and dizzy.

[1] Immediate.
[2] These reactions are on the same individual and followed the fifth, sixth, and seventh injections.
[3] These reactions are on the same individual and followed the first, second, and third injections.
[4] These reactions are on the same individual and followed the fourth, fifth, and sixth injections of the fourth course of treatment but the first course of treatment after a new infection.
[5] Recovery from the first reaction is unknown as the individual did not report his condition to the medical officer.
[6] The patient was apparently fully recovered on the following day but examination revealed crackling rales over right upper lobe giving an impression of tuberculosis, pulmonary, active, chronic.

Temperature.—In the 30 reactions grouped above, the elevation of temperature (100° or over) was reported in 15 cases, slight rise in temperature (less than 100°) was reported in 2 cases, while in 13 cases the temperature was not reported.

Symptoms.—The various manifestations encountered in the 30 reactions follow in order of frequency:

Fever	17	Nausea	6
Headache	15	Vomiting	6
Chills	8	Increased pulse	6
Urticaria	7	Weakness	5

Pains in joints	4	Muscular trembling	2
Pains in abdomen	4	Cramps in abdomen	2
Weak heart action	4	Faintness	2
Dizziness	4	Respiration slow and difficult	2
Delayed arsenic elimination	3	Albumin in urine	1
Increased respiration	3	Skin hot and dry	1
General malaise	3	Herpes of lips	1
Faintness	3	Anorexia	1
Eyes infected	3	Sweating	1
Face flushed	3	Sweating, profusely	1
Body flushed	3	Purged	1
Itching of skin	3	Bleeding from gums	1
Blurred vision	3	Hemolysis of the blood cells	1
Lowered blood pressure (taken)	2	Paleness	1
Body cold and clammy	2	Slight reflexes	1
Rash	2	Restlessness	1
Slight edema about the eyes	2	Moist râles in chest	1
Cyanosis	2		

Nonfatal cases—(case histories).—(1–1931.) A patient (V. A. P.) whose date of initial syphilitic infection was not stated in the report, received antiluetic treatment as follows: From June 15 to July 7, 1931, eight injections of bismuth salicylate; daily inunctions of mercury; potassium iodide (sat. sol.) in ascending doses; and neoarsphenamine 0.3 gram June 19; 0.4 gram June 25; 0.45 gram July 2; and 0.45 gram July 9, 1931. The report states that the dilution of the neoarsphenamine was 1 decigram to 2 mils of water and that the rate of injection was 2 decigrams per minute. About one-half hour after the injection given on July 9, 1931, or the fourth injection of neoarsphenamine, the patient was seized with violent abdominal cramps and a severe chill. He vomited intermittently and purged in the next three hours. The vomitus was bloody but the stools were not. There was considerable bleeding from the gums. The laboratory reports showed that the urine was strongly positive for arsenic in the 3-hour specimens and negative for albumin and casts. There was a beginning of hemolysis of the blood cells; fragility, 34 per cent. Blood count report showed hemoglobin, 70 per cent; white blood count, 10,600; red blood count, 3,080,000; differential count: Polymorphonuclears, 72 per cent, lymphocytes, 18 per cent, mononuclears, 9 per cent, eosinophilis, 1 per cent. The patient was treated with adrenalin (m. VII) and morphine sulphate (grain ⅛) hypodermically, sodium thiosulphate (1 gram) intravenously, and ice and ferric chloride in glycerine controlled the bleeding from the gums. The patient recovered satisfactorily and was able to take solid food the next day. Arsenical therapy was discontinued in this case and bismuth and mercury treatment was resumed.[8]

[8] The reporting medical officer states that there were no other similar reactions from the same drug that date and considered that the patient suffered a severe reaction of the gastrointestinal type.

(2–1931.) A patient with a history of previous ill effects following arsenical treatment was again administered an injection of neoarsphenamine for the purpose of determining if arsenical could be continued with special precautions. The patient had received 12 injections of neoarsphenamine (size of dose not stated) and 11 injections of bismuth salicylate from August 19 to October 25, 1930. In January, 1931, he was given three injections of bismuth salicylate and an unknown number of mercurial inunctions. On February 2, 1931, he was given 1 gram of sodium thiosulphate intravenously following which the urine was negative for arsenic by the Dicken's test in both the 3-hour and 6-hour specimens. On February 10 the patient was given sodium bicarbonate (grains XXX) following each meal on that date and that evening he was given a purgative. The next morning (February 11) preparation was continued with sodium bicarbonate (grains XXX) internally and the omission of breakfast. At 9.30 a. m. he was given $\frac{1}{100}$ grain of atropine followed by an intravenous injection of 0.20 gram of neoarsphenamine. The dilution of the neoarsphenamine was 0.2 gram in 4 mills of water and the rate of injection was stated as one minute. No immediate reaction was noted. The Dickens' test on the urine was positive in both the 3-hour and 6-hour specimens. At 4 p. m. the patient developed a severe urticarial rash over face, trunk, and arms with few wheals on legs. He was given 1 gram of sodium thiosulphate intravenously, 15 minims of $\frac{1}{1000}$ epinephrine subcutaneously, 1 ounce of magnesium sulphate orally, liberal water and liquid diet, and kept in bed. In attempting to go to the toilet (without permission) he fainted but this was considered by the reporting medical officer to be due to the epinephrine. At 9 p. m. he was given 10 minims of epinephrine and 800 cubic centimeters of a 2 per cent solution of sodium carbonate was given and retained per rectum. On the morning of February 12, the patient felt much better, the rash was practically gone but there was some itching still remaining. This date he was given two intravenous injections, each 1 gram, of sodium thiosulphate; 1 ounce of castor oil was also given because of the failure of the previous dose of magnesium sulphate to move the bowels.[4]

(3–1931.) A patient who was originally admitted with a diagnosis of syphilis May 26, 1930, received his first course of arsenical treatment from June 3 to July 30, 1930, consisting of 8 injections of

[4] The reporting medical officer concluded as follows: "This report is considered to be an untoward individual reaction and not due to fault in the arsenical compound. Whereas the patient might be able to continue to take neoarsphenamine with precautions and suffer no more than an urticarial rash, nevertheless arsenicals will be discontinued in his case on account of the danger of provoking exfoliative dermatitis and because the patient tolerates mercury and bismuth."

neoarsphenamine, 0.6 gram each, and 8 injections of mercury succinimide, 2/3 grain each, and his second course from October 28, 1930, to January 12, 1931, consisting of 8 injections of neoarsphenamine, 0.6 gram each, and 16 injections of mercury bichloride, 30 minims of a 1 per cent solution, and from January 19 to April 6, 1931, he received 20 additional mercury bichloride injections as above. His third course was begun on July 16, 1931, with an 0.3-gram injection of neoarsphenamine and 0.45 gram was given on July 23, and 0.6 gram on July 30. On July 20 and 25 he received 1 cubic centimeter (each) of a bismuth preparation. Fifteen minutes after the third dose of his third course, the 0.6 gram given on July 30, the patient complained of cramps in abdomen, headaches, and joint pains. His face became quite flushed, pulse and temperature, however, at this time were normal. The acute phase passed off within five minutes. The patient was given 5 minims of adrenalin intradermally and 1 gram of sodium thiosulphate intravenously. This was about 10 a. m. At 1 p. m. the temperature was 101° F., the headache and joint pains continuing, but the patient was fairly comfortable. At 7.30 p. m. the temperature was normal, no complaints, and the patient was restored to duty. Dickens' test was strongly positive at the end of three hours, but through error the 6-hour specimen was not obtained.

(4–1931.) A patient who was exposed in May, 1931, developed a lesion on the inferior surface of the shaft of penis which was repeatedly negative for *Treponema pallidum* by dark-field examination. Kahn tests were also negative June 24 and June 26, 1931, and the case was diagnosed as chancroid. By July 20 the lesion was entirely healed but on August 11 and 17, 1931, the Kahn tests were found to be 4 plus, consequently the diagnosis was changed to syphilis and antiluetic treatment instituted. His first course of treatment was begun on August 25, 1931, with an intravenous injection of 0.3 gram of neoarsphenamine, and 0.6-gram injections were given on September 1 and September 8, 1931. About two and one-half hours after the third injection of his first course, that given on September 8, the patient complained of headache and generalized pain and aching of body. His face was flushed, his eyes were injected, and his skin was hot and dry. There was a rash over the chest, abdomen, and arms, a diffuse mottling with erythema, and his temperature was 105° F. Treatment consisted of 1 gram of sodium thiosulphate three times a day, catharsis, and forced fluids. By September 10 the reaction was disappearing; September 11, the urine was negative for albumin, with leukocytes 8 per field and temperature 100° F. in the afternoon; September 13, the temperature was normal and the patient was up and about; September 15, he was discharged to duty as well. Caution as to the size of dose was

recommended in future treatment in this case and on September 22 and 29 he was given 0.3 gram (each) of neoarsphenamine without further ill effect.

(5–1931.) A patient who was exposed in Washington, D. C., on November 1, 1931, was diagnosed syphilis by a positive dark-field examination of serum from the initial lesion. First course of treatment was started December 1, 1931, with an injection of neoarsphenanime (dose not stated) and on December 8, 1931, he was given an injection of 0.45 gram. This second injection consisted of 0.45 gram of neoarsphenamine in 10 cubic centimeters of triple distilled water and the rate of injection was reported as three minutes. Immediately following this injection the patient fainted, became cold and clammy with heart action regular but very weak. His pupils were equal and regular. These symptoms were followed by nausea, vomiting, and headache. Laboratory reports showed blood counts and urinalysis normal and Kahn tests 4 plus. Dickens' tests not made. The patient was given 1 cubic centimeter adrenalin hypodermically, 1 gram sodium thiosulphate intravenously, and general treatment for shock. Recovery was complete in about four hours.

(6–1931.) A patient was exposed on July 26, 1931, at Norfolk, Va. Diagnosis of syphilis was made by a dark-field examination of serum from the primary lesion. Wassermann test at this time was negative, secondary manifestations noted by glandular enlargement. Two courses of treatment, consisting of 14 injections, were given between the dates of July 29 and October 8, 1931, size of doses not stated. No mercury, iodine, or bismuth given as concurrent treatment. This third course started on December 3, 1931, with 0.45 gram of neoarsphenamine diluted in 10 cubic centimeters of water. Two cubic centimeters of this solution had been injected when the patient stated he felt weak. Patient then became pale, with pulse rapid and weak, reflexes, slight response. He fainted when assisted from the table. Patient was given adrenalin, minims X, by subcutaneous hypodermic injection and then placed in bed. Patient then went to sleep and was discharged to duty well after four hours.

(7–1931.) A patient exposed in Cassville, N. J., September 26, 1930, developed a generalized secondary rash and gave a 4 plus blood Kahn test on October 21, 1930. From October 22, 1930, to April 29, 1931, he received two courses of neoarsphenamine consisting of a total of 20 injections. The first dose of his third course consisted of 0.3 gram of neoarsphenamine given on July 8, 1931, and his second dose, given 14 days later on July 22, was also 0.3 gram. The patient had received six intramuscular injections of bismuth potassium sodium tartrate in 0.1-gram doses from January 7 to February 18, 1931, and six intramuscular injections from May 6

to July 1, 1931. The second dose of the third course of neoarsphenamine, that given on July 22, consisted of 0.9 gram of neoarsphenamine in 20 cubic centimeters sterile triple distilled water and the rate of injection was reported as four minutes for the 0.3-gram dose. Eleven hours after this injection there were headache, nausea, vomiting, and restlessness. The patient was put to bed with ice cap to head, 1 gram of sodium thiosulphate administered intravenously, and recovery was complete within 24 hours.

(8–1931.) A patient, on April 9, 1930, had a lesion appear on his lower lip which had the symptoms of being luetic. Diagnosis of syphilis was made by a 4 plus Kahn test. His first course of treatment was 10 injections of neoarsphenamine given between the dates of April 9 and July 18, 1930, for a total of 4.55 grams. He was given mercury rubs; bismuth salicylate, 6 injections, ½ grain each (intramuscular); and mercury succinimide, 10 injections, ⅕ grain each (intramuscular). The second course of treatment consisted of six injections of neoarsphenamine given between the dates of July 24 and October 16, 1930, total amounts given not stated. The third course was started on April 29, 1931, during which the patient received a total of 4.95 grams of neoarsphenamine in seven injections, the last injection being given on June 10, 1931. Six hours after he received this injection he had an elevating of temperature, severe cramps in abdomen, severe headache, some increase of pulse rate but full pulse, some respiratory upset, irregular respiration but no cyanosis. He was given 1 gram of sodium thiosulphate and an enema. Prompt amelioration of symptoms followed with recovery in about one hour.[5]

(9–1931.) A patient exposed in Shanghai, China, June 13, 1931, later developed an initial lesion on sulcus, generalized adenopathy and had a strongly positive blood Kahn. On August 24, 1931, he was given an intravenous injection of 0.3 gram of neoarsphenamine and 0.45 gram doses on August 27 and September 3, 1931. Between August 24 and September 3 the patient received four 2-grain doses of bismuth salicylate. The third dose of the first course of neoarsphenamine, that given on September 3, consisted of 0.45 gram of neoarsphenamine dissolved in 10 cubic centimeters of normal saline solution and the rate of injection was one and one-half minutes. The patient had been prepared in the usual manner—a saline purge the night before, no breakfast, and a routine examination. Four hours after this injection the patient had a temperature of 105.6° F., chills and rigor, headache, and a macular rash.

[5] The reporting medical officer states that this reaction resulted from the patient eating breakfast, although patient denies eating.

The urine showed no arsenic elimination and a plus albuminuria. He was put in bed, given sodium bicarbonate (grains XX) three times a day and forced fluids of orange and lemon juice. Sodium thiosulphate, 0.5 gram, was given intravenously on September 4, and 1 gram was given on September 5 with a saline purge. September 6, the rash was disappearing and the temperature was 100° F. September 7, the temperature was normal, the forced liquids were continued, and by September 8 the rash had disappeared. No further manifestations developed in this case.

(10–1931.) A patient who was exposed at Newport, R. I., on August 15, 1926, later developed a primary lesion on penis and on September 29, 1926, gave a 3 plus blood Kahn test. From October 5, 1926, to November 9, 1926, he received 6 intravenous injections of neoarsphenamine (amount not stated) and 4 injections of mercury, and from January 18 to February 15, 1927, he received 5 injections of neoarsphenamine totaling 4.2 grams. The first dose of his third course was given February 1, 1931, consisting of 0.45 gram of neoarsphenamine. Six injections of bismuth was reported as concurrent treatment. The patient was given the intravenous injection of neoarsphenamine on board another ship at 10 a. m. on February 1, 1931, and he returned to his ship at 11 a. m. At that time he made no complaint about his condition although he stated later that he felt badly when he returned. At 12.30 p. m. the pharmacist's mate was advised that the patient was ill and in his bunk. When seen he complained of severe headache and pains in joints and muscles. He had a slight chill of about three minutes' duration. His temperature was 101.5° F. and his pulse was rapid. By 1 p. m. his temperature had elevated to 103° F. and the pain in the head and muscles was more severe. He was unable to lie with limbs extended. He was transferred to a naval dispensary for further treatment, where he was given 0.6 gram of sodium thiosulphate. The following morning he had no complaints and on February 3 he was discharged to duty as well.

(11–1931.) A patient was exposed in Nicaragua on May 8, 1930. Diagnosis for syphilis was made by a positive dark-field examination of serum from lesion. First course of arsenicals was administered between May 12 and June 24, 1930, total amounts not stated. Second course of arsenicals was administered between January 1 and February 12, 1931, total amounts not stated. No concurrent treatment given. During his third course of treatment, six injections of neoarsphenamine for a total amount of 3.3 grams, he had symptoms of a reaction. The dose causing reaction consisted of 0.9 gram of neoarsphenamine dissolved in 18 cubic centimeters of distilled water, 0.6 gram dose being administered. The rate of injection was

stated as two minutes. The following morning the patient had pain in upper abdomen, a temperature of 98° F., a white-blood count 5,500, and a very definite tender area above and to the left of umbilicus. Patient was transferred to the hospital and because of the lack of any further evidence of reaction he was carried as "no disease" and was discharged to duty in eight days.

(12–1931.) A patient who denied recent sexual exposure and claimed no knowledge as to how infection was acquired was admitted to the sick list November 14, 1931, with a diagnosis of lymphadenitis, inguinal, right. At this time examination revealed two painless sores, one on the upper portion of the scrotum, right side, and the other on the pubis. The lesions were somewhat indurated and the right inguinal and femoral glands were enlarged. Dark-field examination was positive for *Treponema pallidum* and a blood Kahn reaction was 4 plus. A diagnosis of syphilis was established on November 16, 1921, and on November 17 the patient was given 0.3 gram of neoarsphenamine intravenously, diluted 0.3 gram to 7 cubic centimeters of freshly distilled sterile water with about one minute being required for the injection. About seven hours after this injection the patient had a chill accompanied by headache, nausea, and vomiting. This was followed by a rise in temperature to 103.8° F., with pulse 110 and respiration 18. One gram of sodium thiosulphate was given immediately by vein, 10 minims of adrenalin was administered subcutaneously, and Fischer's solution was given by protoclysis. The symptoms gradually subsided and the next morning the temperature, pulse, and respiration were normal. Van den Bergh, icterus index, and bromsulphthalein were all within normal limits. Twenty-one injections of the same preparation were given on the same date, using the same technique, without any untoward effects whatever, and routine antiluetic treatment including neoarsphenamine (0.45 gram) was given this patient on November 24 without any untoward effects.

(13–1931.) A patient was admitted to the sick list with a diagnosis of tonsillitis, chronic, on September 15, 1931. He gave a history of sore throat for the past two months. Examination revealed enlarged and chronically diseased tonsils. The uvula and left tonsil presented patches of white exudate suggestive of mucous patches. Smear negative for Vincent's organisms. History of syphilis and evidence of initial sore lacking but three Kahn tests were each 4 plus. Some adenopathy was present. Tonsillectomy postponed. A diagnosis of syphilis was made on October 1, 1931. Heart not enlarged, sounds normal and blood pressure 122/84. Urine negative for albumin and sugar. On October 2 and 9 he was given 0.1 gram of bismuth intramuscularly. On October 6 he was given

0.3 gram of neoarsphenamine and on October 13 he was given 0.45 gram of neoarsphenamine which was administered in 10.5 cubic centimeters of freshly distilled sterile water injected slowly (two minutes) into the vein. About six hours after this injection the patient had some headache and fever but he did not report his condition until sick call the next morning (10–14–31). At sick call on October 14 his temperature was 101.4° F., pulse 118, and respiration 19. He complained only of fever, no nausea or vomiting. Examination revealed conjunctivæ slightly injected, no icterus of scleræ, heart sounds normal, blood pressure 115/70, superficial lymph glands were markedly enlarged, examination otherwise negative. Dickens' test was slightly positive for arsenic. Patient was given 1 gram of sodium thiosulphate intravenously and put to bed with further treatment of saline catharsis and forced liquids. At about 4 p. m. this afternoon the patient was given another intravenous injection of sodium thiosulphate, 1 gram. The next day, October 15, the temperature rose to 104° F., pulse 102, and respiration 18. His only complaint was fever, slight headache, and weakness. Dickens' test was negative for arsenic. Sodium thiosulphate 1 gram was given intravenously. On October 16 the temperature was gradually reducing and he was given another intravenous injection of 1 gram of sodium thiosulphate. The patient showed much improvement on October 17 with adenopathy subsiding. This date a macular rash appeared over his body. Sodium thiosulphate, 1 gram, was given intravenously. On October 19 the patient had no further complaints, his temperature was normal, and body rash cleared up entirely. Apparent recovery completed this date. Weekly injections of bismuth, 0.1 gram, intramuscularly continued.[6]

(14–1931.) A patient who was exposed at Swatow, China, on May 17, 1931, developed a lesion on penis on May 29, 1931, which was positive for *Treponema pallidum* by dark-field examination July 24. The blood Kahn serum reaction was 3 plus September 21, 1931. From July 24 to August 16, 1931, the patient received five intravenous injections of neoarsphenamine, 0.45 gram each, and 0.45 gram on September 15; 0.6 gram on September 22; and 0.75 gram on September 29, 1931. No other concurrent treatment was reported. The last (or eighth) dose of neoarsphenamine consisted of a 0.9 to 20 solution and the rate of injection was stated to be two and one-half minutes. About one-half hour following the injection the patient began to have a chill with a feeling of impending danger. The lips and face became quite cyanotic, radial pulse was very weak, and there were universal muscular tremblings. The blood pressure was 126/84. He

[6] Medical officer reported that 20 injections were given at the same time, using same material and technique without any untoward effects whatever.

was given 1 cubic centimeter of adrenalin hypodermically, and one-half hour later, 1 gram of sodium thiosulphate intravenously. The pulse slowed to 120–130, and the temperature was 100° F. at this time. Fluids were forced and the patient was feeling quite well five hours after the injection.

(15–1931.) A patient was exposed on July 19, 1931, at Manila, P. I. Diagnosis of syphilis was made on August 20, 1931, as the result of clinical observation. On August 29, 1931, the diagnosis of syphilis was vertified by a 3 plus Kahn blood test. Patient received 0.3 gram and 0.6 gram doses of neoarsphenamine on August 12 and 19, respectively. No concurrent treatment was given. On August 28 the patient received 0.6 gram dose of neoarsphenamine dissolved in 10 cubic centimeters of sterile distilled water with the rate of injection at approximately 5 cubic centimeters per minute. Total amount of neoarsphenamine administered to date was 1.5 grams. About two hours after the last injection the patient complained of being ill. His temperature was 104° F. and he vomited once. In about eight hours after the first rise of temperature the temperature regained normalcy and the patient apparently recovered from the reaction except for marked herpes about the lower lip, chin, and left cheek, which appeared two days later.

(16–1931.) A patient who was exposed at Guantanamo City, Cuba, on January 26, 1931, developed a primary lesion on the shaft of penis February 18, 1931. *Treponema pallidum* was discovered in the morning of February 20, and at 3 p. m. that afternoon 0.3 gram of neoarsphenamine was administered intravenously. As a matter of routine, the drug is always carefully inspected in the tube before use and while dissolving, this procedure having been observed when the man was given the injection. The drug was administered slowly and all aseptic precautions were observed. The patient demonstrated no ill effects from the medication that evening but the Dickens' test indicated that he was retaining the arsenic. He went to sleep early that night and reported to the sick bay at 6.30 a. m. the following day complaining of fever, headache, and general malaise. His temperature on admission was 102° F., pulse 99, and respiration 20. Sodium thiosulphate, 0.4 gram, was administered intravenously and the patient stated that he felt better immediately. The urine showed a faint trace of arsenic at this time, 16 hours after the administration of the neoarsphenamine. He improved rapidly hereafter but was kept in the sick bay for observation and study. Another injection of 0.3 gram of the same neoarsphenamine was given 10 days later and was easily tolerated, the patient showing absolutely no ill effects and the Dickens' test indicating that he was eliminating arsenic in normal time—the urine being positive in one hour.

(17–1931.) A patient who denied any place and time of infection, and for whom the diagnosis of syphilis was not made but strongly suspected, was given 0.3 gram of neoarsphenamine on January 29, 1931, at about 9 a. m. The dilution of this injection was 1–20 and the rate of injection was 30 seconds. About six hours later the patient complained of headache, pain in joints, and general malaise. Temperature was 102° and continued to rise to 104.2° F. but then gradually returned to normal by the next morning. Patient was given 1 gram of sodium thiosulphate at about 3 p. m. and apparent recovery was completed in about 16 hours.

(18–1931.) A patient was infected on September 20, 1930, at Tsingtao, China. Diagnosis was made by two positive Kahn blood tests. Patient had received first course of treatment, eight injections of neoarsphenamine with a total dosage of 4.2 grams, between the dates of November 20, 1930, and January 8, 1931. Between the dates of November 21, 1930, and March 5, 1931, the patient received sixteen 1 cubic centimeter intramuscular injections of bismuth salicylate suspension. No mercury, iodine, or other previous or concurrent treatment was administered. From February 24 to March 10, 1931, the patient had received three injections of neoarsphenamine, total dosage 1.2. A few minutes after he had received the 0.6-gram injection on March 10, dilution of which was 0.1 gram to 3.3 cubic centimeters sterile distilled water and the rate of injection was 10 cubic centimeters of this solution per minute, the patient became dizzy, nauseated, and very weak with the pulse rate increasing to 100 per minute. The patient was immediately given an injection of 1 cubic centimeter epinephrine, and a similar injection was administered one-half hour later. An injection of 1 gram of sodium thiosulphate was given within one hour of onset of symptoms. Recovery was apparent after three hours. This patient had 5 injections of neoarsphenamine and 12 injections of bismuth administered since his reaction, with no ill effects noted.

(19–1931.) A patient exposed in November, 1931, at Peiping, China, later developed a secondary rash and a diagnosis of syphilis was established by a 4 plus blood Kahn serum reaction. On December 28, 1931, he was given an intravenous injection of 0.3 gram of neoarsphenamine dissolved in 7 cubic centimeters of water, about one minute being required for the administration of the dose. About nine hours later the patient complained of headache, pain in the abdomen and vomiting, and his temperature was 104.2° F. He was given 1 gram of sodium thiosulphate intravenously, 5 minims of adrenalin subcutaneously, 0.025 gram of ephedrin, sodium bicarbonate enema, and 1 tablespoonful of sodium bicarbonate in hot water by mouth. The Dickens' test was positive for arsenic 22

hours after the injection. The patient was symptom-free December 30, or in about 48 hours.

(20–21–22–1931.) Three reactions from arsenical administration occurred in the same man during the month of June, 1931. The patient was given a diagnosis following 3-plus Kahn precipitation tests on May 11 and 12, 1931, and was given intravenous neoarsphenamine as follows: May 12, 0.3 gram; May 19, 0.45 gram; May 26, 0.6 gram; June 2, 0.6 gram; June 9, 0.6 gram. Several hours following the injection on June 9 a reaction occurred, the patient complaining of chills and headache, temperature of 100° F., pulse 80, and respiration 18; this reaction lasted three hours. On June 16, 0.3 gram was administered and a similar reaction occurred with temperature 101° F., pulse 90, and respiration 18; this reaction also lasted three hours. On June 23 the patient was given 7½ grains of sodium thiosulphate intravenously, followed by 0.45 gram of neoarsphenamine, and a more pronounced reaction occurred with temperature 103° F., pulse 100, and respiration 20; this reaction lasted for five hours. Antileutic treatment was subsequently changed to a bismuth preparation given intramuscularly.

(23–24–25–1931.) Three reactions from arsenical administration were recorded against the same man during the month of March, 1931. The patient was infected March 15, 1930, at Savannah, Ga. Diagnosis was established by clinical findings and positive Kahn blood tests. The patient had received three courses of arsenical treatment as follows: 3 injections of neoarsphenamine between dates of April 8 and April 23, 1930; 12 injections of neoarsphenamine between July 15 and September 29, 1930; 12 injections of neoarsphenamine between November 18, 1930, and February 10, 1931. Amounts of dosage not stated. Patient received 1 cubic centimeter bismosol, intragluteally, on February 28, March 6, and March 24, 1931. His fourth course of treatment was begun on March 10, 1931, with 0.45 gram injections of neoarsphenamine being given on March 10, 17, and 24. Each dilution was neoarsphenamine 1 to water 22 and the rate of each injection was four minutes. About five minutes after the injection on the 10th and 17th the patient had an itching of skin over right antecubital fossa, and to a lesser extent a similar condition on the left arm. Later there was moderate urticaria of the flexor surface of both arms, from the middle of the forearm to the middle of the upper arm, which was accompanied by dizziness and faintness. Following the neoarsphenamine administration on the 24th, there was slight itching around the site of injection but no urticaria or constitutional symptoms. The patient on each of the three occasions mentioned above was placed in bed with head low, hot-water bottles and blankets applied, epinephrin (5 minims) administered subcutaneously, and the same dosage given very slowly

intravenously. The time of apparent complete recovery was approximately 30 minutes from the onset of symptoms in each instance.

(26–27–28–1931.) Three reactions from arsenical administration occurred in the same man during the month of October, 1931. The patient had an initial lesion (first infection) May 13, 1930, and a secondary rash and 4 plus Kahn blood test on June 12, 1930. For his first course of treatment, from June 12 to July 29, 1930, he received one 0.3 gram and seven 0.6 gram intravenous injections of neoarsphenamine and 10 intramuscular injections of mercury succinimide. For his second course, from September 10 to November 5, 1930, he received one 0.3 gram and seven 0.6 gram intravenous injections of neoarsphenamine and 10 intramuscular injections of mercury succinimide. For his third course, from January 7 to March 9, 1931, he received one 0.3 gram and seven 0.6 gram intravenous injections of neoarsphenamine and 10 intramuscular injections of bismuthotartrate. The patient had a new infection August 2, 1931, with a 4 plus Kahn blood test on September 16 and mascular-papular rash and mucous lesions on September 18, 1931; rash greatly exaggerated by first injection of neoarsphenamine. For his fourth course, or the first course after his reinfection, he received intravenous neoarsphenamine, 0.3 gram, September 18; 0.6 gram, September 23, 29, October 6, 13; and 0.15 gram on October 27, 1931. About one hour after the injection given on October 6 the patient noted a flushing of body, felt warm, and soon noted wheals on arms and body, with a slight blurring of vision. He did not report the reaction. About 30 minutes after the injection given on October 13 he had a fairly marked reaction. He reported to the sick bay with eyes, face, and body flushed, except for pallor about nose and mouth. Profuse sweating and urticaria were soon noted over body and extremities. The pulse was weak and rapid. He complained of weakness and blurred vision, nausea, and feeling of warmth. He was given 1 cubic centimeter of adrenalin, after which he soon vomited and felt better. He was fully recovered after one hour. Before the injection of 0.15 gram of neoarsphenamine given on October 27, he was given three 3/8-grain capsules of ephedrine hydrochloride. About two minutes after the neoarsphenamine was administered, his eyes became injected, followed by flushing of skin, except about nose and mouth, where there was a noticeable pallor. Urticaria quickly appeared on the body and limbs and the eyelids became œdematous. The radial pulse was imperceptible, the vision blurred, and the patient was very weak and dizzy. His respiration became very difficult and slow. He was given 1 cubic centimeter of adrenalin, and felt better after several minutes. The pulse became weak again, and 1 cubic centimeter of adrenalin was repeated. He felt all right after about

two hours. The report does not state that any other antiluetic treatment was substituted.

(29–1931.) A patient who did not have syphilis but who was treated for "psoriasis" had received neoarsphenamine treatment on September 17, September 22, and September 24, 1931, receiving 0.3 gram at each administration. The patient developed fever following a chill about eight hours following the administration on September 24, the dilution of which was 0.9 gram of neoarsphenamine to 30 cubic centimeters of sterile distilled water, the rate of injection being five minutes for the injection of 10 cubic centimeters of this solution. These symptoms were accompanied by anorexia, nausea, perspiration, and fine tremors. By the following day he felt better. On September 26, two days after the last injection, the patient reported to the medical officer of the day with a peculiar network of urticarial wheals over the entire body surface. On September 28 the patient was turned in at the dispensary ward with a skin eruption of the character described, a small petechial hemorrhage of the right bulbar conjunctiva, a slight œdema under the eyes and complaining of joint pains, temperature 99.5° F., pulse 88, and respiration 20. The patient was placed in bed, was given 1 ounce of magnesium sulphate, and 1 gram of sodium thiosulphate was administered intravenously. Complete recovery was made on October 1, 1931, with the rash entirely faded.

(30–1931.) A reaction occurred in a patient who was being treated with neoarsphenamine for "yaws." The patient, a native of Guam, had previously presented the skin lesions of yaws and a positive blood Kahn, and had received three complete courses of neoarsphenamine. For the fourth course the patient received intravenous neoarsphenamine, 0.3 gram, on May 6 and 0.6 gram on May 13, 20, 27, and June 3, 1931. The last injection, which was the fifth dose of the fourth course, consisted of 0.6 gram of neoarsphenamine in 12 cubic centimeters of sterile water injected in two minutes. Fifteen minutes later there was difficulty in breathing and the patient fainted. Examination showed the patient to be fighting for breath, very cyanotic, cold and clammy, pulseless in peripheral arteries, heart sounds weak and very rapid, and many moist bubbling râles in both sides of the chest. The patient was given one-half cubic centimeter of adrenalin by hypodermic and 10 minutes later, 1 gram of sodium thiosulphate and 2 cubic centimeters of adrenalin intravenously. The patient was apparently fully recovered on the following day but examination revealed crackling râles over right upper lobe giving an impression of tuberculosis, pulmonary, active, chronic.

Arsenical Dermatitis

Deaths.—No fatal cases of arsenical dermatitis occurred during the year 1931.

During the 7-year period 1925–1931 there were four deaths due to arsenical dermatitis following the administration of neoarsphenamine. During this period there were 495,312 doses of neoarsphenamine administered, giving a ratio of 1 death to 123,828 doses administered.

Using the figures of a previous article, which were based on the 4-year period 1927–1930, the case fatality rate for neoarsphenamine cases for the 5-year period 1927–1931 is 4.94 per cent. This calculation is based on 81 cases with 4 deaths following the administration of neoarsphenamine. The calculations thus derived reduce the case fatality rate from 7.55 per cent for the 4-year period 1927–1930 to 4.94 per cent, for the 5-year period 1927–1931, or a reduction of 2.61 per cent.

Three cases with no deaths followed the administration of sulpharsphenamine.

Nonfatal cases of arsenical dermatitis.—During the year 1931 there were 28 cases which followed the use of neoarsphenamine. These cases were classified into 23 cases with severe reactions and 5 cases with mild reactions. Some of the available data for these cases are presented below in tabular form:

NEOARSPHENAMINE

Key	Previous course of treatment	Current course of treatment							
		Course	Dose followed by arsenical dermatitis	Total grams	Number of days in course	Average milligrams per day	Concurrent treatment	Onset of arsenical dermatitis after last dose	Duration of arsenical dermatitis (days)
43	None	First	Second	0.65	7	93	None	3 days	40
47	do	do	do	.9	7	129	Daily rubs of mercury for 2 weeks; 2 injections of bismuth.	do[1]	8
51	do	do	do	.9	7	129	None	2 days	5
52	do	do	Third	1.20	14	86	do	1 day	8
48	do	do	Fifth[2]	2.7	35	77	6 injections bismuth	(?)	18+
50A	do	do	do	(?)	40	(?)	13 injections bismuth	Not stated	41
57	do[3]	do	do	2.25	(?)	(?)	None	11 days	(?)
50	do	do	Sixth	3.6	35	103	6 injections bismuth; 6 doses mercury succinimide.	5 days	3
55	do[3]	do	do	2.85	(?)	(?)	None	14 days	(?)
33	do	do	Seventh	3.15	42	75	Daily mercury rubs first 2 weeks and last 2 weeks of treatment.	2 days	32+
44	do	do	do	4.2	42	100	None	1 day	93
46	do	do	do	3.9	42	93	7 injections bismuth	5 days	101
53	do	do	do	3.9	48	81	5 injections bismuth	28 days	20
54	do[3]	do	do	4.2	(?)	(?)	None	1 day	45+
34	do	do	Eighth	4.65	47	99	8 injections bismuth [4]	7 days	142
39	do	do	do	3.45	50	69	do	3 days	60
42	do	do	do	3.6	49	73	9 injections bismuth	7 days	57
56	do[3]	do	do	4.05	(?)	(?)	None	13 days	22+
38	do	do	Tenth	5.8	52	112	do	3 days	77
37	do	do	Fifteenth	[5] 7.6	[5] 114	[5] 67	15 injections bismuth	1 day	57+
35	Started first course March, 1929, discontinued after fourth injection because of a reaction.	Second	First	.18	1		None	5 hours	32
36	Jan. 21, 1931 to Apr. 14, 1931 (following second injection on Jan. 27, 1931 he had a reaction, symptoms subsided and treatment was resumed), 5.55 grams, neoarsphenamine and 42 inunctions of mercury. Starting June 24, 1931 he received 16 biweekly injections of bismuth.	do	Second	1.8	7	257	do	3 days	153+
32	Apr. 16, 1932 completed first course of 12 injections neoarsphenamine, 5.4 grams; 8 injections of bismuth.	do	Tenth	5.4	77	70	20 injections bismuth	Less than 7 days [6]	29+
41	1918 received 9 injections salvarsan. 1931 received eleven 1/6-grain injections of bichloride of mercury.	do	do	5.4	30	180	None	3 days	108

45	Jan. 8, 1931 to Feb. 19, 1931, 4.7 grams neoarsphenamine; Jan. 2, 1931 to Mar. 13, 1931, 21 injections bismuth.	---do----	Eleventh..	5.05	73	69	18 injections bismuth.....	Less than 6 days [6]	70
40	Oct. 18, 1930 to Dec. 5, 1930 (following first injection he had a slight reaction), 2.55 grams neoarsphenamine and 6 injections of bismuth; Dec. 23, 1930 to May 13, 1931, 24 injections bismuth; Jan. 19, 1931 given an injection of neoarsphenamine, dose (?) (followed by a reaction); Feb. 3, 1931 given an injection of neoarsphenamine, dose (?); Feb. 7, 1931 to Mar. 7, 1931, specific mixture, amount (?).	Third [7]	Third.....	1.15	14	82	4 injections bismuth.....	15 minutes.....	42
49	1919 received treatment a short time, amount (?); Aug. 26, 1930 to Oct. 7, 1930, 3.9 grams, neoarsphenamine.	---do----	Fourth....	2.1	49	43	11 injections of mercury bichloride.	(8).....	20+
31	Jan. 13, 1930 to Feb. 27, 1930, 5.25 grams neoarsphenamine; June 19, 1930 to Aug. 7, 1930, 4.5 gram neoarsphenamine and ten 1-grain injections of bismuth, and 5 mercury rubs.	---do----	Seventh...	4.0	43	93	None.....	1 day.....	14

[1] Shortly after the last injection the patient had a slight headache and chill but was not admitted to the sick list until 3 days later.
[2] 1 week after receiving his fourth injection he complained of "sickness" and the injection for that week was skipped. A week later he received his fifth injection.
[3] Natives of Guam treated for "yaws."
[4] Patient received 8 injections of bismuth while in the hospital.
[5] This man received the following treatment on his first course: Jan. 27, 1931 to Mar. 19, 1931, 3.4 grams neoarsphenamine in 51 days, average milligrams per day, 66.6. Rest interval (reason not stated) of 21 days. Apr. 9, 1931 to May 21, 1931, 4.2 grams neoarsphenamine in 42 days, average milligrams per day, 100. Total of 7.6 grams neoarsphenamine in 114 days, average milligrams per day, 66.6.
[6] These days have been determined as the maximum time of symptoms or date nearest to diagnosis.
[7] This has been considered the third course because of the rest interval between the injection given on February 3, 1931, and the first injection given on June 9, 1931.
[8] Patient developed an eczematous condition on the date of administration of his fourth injection of third course.

In all but one case there is a history of recent injections of from 2 to 15 doses of neoarsphenamine in periods ranging from 7 to 114 days, with varying amounts of the drug averaging from about 43 to 257 milligrams per day over the period comprising the current course of treatment. Some of the patients had also received mercury or bismuth, and in some cases no concurrent treatment was recorded.

A glance at the above table shows that in only 32 per cent of the cases of arsenical dermatitis were the patients receiving dosage in excess of that recommended by paragraph 10 of Bureau of Medicine and Surgery circular letter, Serial No. 492–1929.

The following table is a ready guide for a comparison of the given dosage and the dosage recommended by the above mentioned reference:

Number of injection	Number of days in course	Recommended amount in grams	Total amount in grams to date	Recommended milligrams per day	Number of injection	Number of days in course	Recommended amount in grams	Total amount in grams to date	Recommended milligrams per day
First	1	0.45	0.45	------	Sixth	35	0.45	2.70	77
Second	7	.45	.90	129	Seventh	42	.45	3.15	75
Third	14	.45	1.35	96	Eighth	49	.45	3.60	73
Fourth	21	.45	1.80	86	Ninth	56	.45	4.05	72
Fifth	28	.45	2.25	80	Tenth	63	.45	4.50	71

Primary clinical symptoms in cases of arsenical dermatitis and primary and later clinical symptoms noted throughout the duration of dermatitis—Neoarsphenamine.—The following clinical symptoms ("primary" and "primary and later") were noted in reactions resulting from the use of neoarsphenamine:

Primary symptoms

General erythema	9	Skin eruption on flexor surface of the arms	1
Generalized severe exfoliating dermatitis	2	Rash on extensor surface of forearms and peroneal region	1
Severe exfoliation of face, neck, and arms	2	Dry, itching, exfoliating skin condition	1
Dermatitis of hands	1	Fever	11
Itching erythematous rash	1	Chills	5
Itching and burning erythematous rash	1	Headache, slight	3
Erythematous-papular rash on forearms, arms, and trunk	1	Edema facial	2
Itching eczematous rash	1	High pulse rate	2
Mild eczematous condition of the face	1	Conjunctivæ, injected	2
Mild eczematous condition of the hands and wrists	1	Swollen ankles	1
Skin eruption on flexor surface of the elbows	1	Swelling in both cubital fossæ with itching	1
		Pains in joints	1
		General malaise	1
		General weakness	1

Inability to work	1
Lassitude	1
Dry and cracked lips	1
Itching and burning about face and ears	1
Pharyngeal and buccal mucosa injected	1
Sore throat	1
Nausea	1
Headache, severe	1
Tightness in chest	1
Face flushed	1
Faintness	1
Pallor	1
Vertigo	1

Primary and later symptoms

General erythema	15
Itching and burning erythematous rash	2
Generalized severe exfoliating dermatitis	2
Severe exfoliation of face, neck and arms	2
Dermatitis of hands	1
Dermatitis of both axillæ, back of neck, eyes, hands, legs, and feet	1
Dry, itching, exfoliating skin condition	1
Itching erythematous rash	1
Itching eczematous rash	1
Mild eczematous condition of the face	1
Mild eczematous condition of the hands and wrists	1
Fever	13
Edema, facial	6
Chills	5
Headache, slight	4
High pulse rate	4
Swollen eyelids	3
Conjunctivæ injected	2
Throat injected and red	2
Headache, severe	2
General aching over body	2
Papillæ of tongue prominent	2
Restlessness	2
Stiffness in joints	1
Pain in joints	1
Generalized edema	1
Swollen ankles	1
Edema of hands	1
Swelling in both cubital fossæ with itching	1
Itching and burning about face and ears	1
Skin warm and feverish	1
Dry and cracked lips	1
Pharyngeal and buccal mucosa injected	1
Desquamation of the skin, hair, and nails	1
Loss of hair on face and scalp	1
Painful adenopathy	1
High leukocyte count	1
Tightness in chest	1
Nausea	1
Flushed face	1
General weakness	1
Inability to work	1
Listlessness	1
Apathy	1
Lassitude	1
Vertigo	1
Faintness	1
Pallor	1

Arsphenamine.—No cases of arsenical dermatitis attributable to arsphenamine were reported in 1931.

Neoarsphenamine (case histories).—(31–1931.) A patient who was exposed in Peiping, China, December 26, 1929, developed a secondary rash and adenopathy, gave a 4 plus blood Kahn reaction, and a gland puncture was positive for *Treponema pallidum.* His first course of treatment consisted of intravenous neoarsphenamine given as follows: January 13, 1930, 0.45 gram; January 16, 20, and 23, 0.6 gram each; January 27, 0.9 gram; January 30, 0.6 gram;

February 3, 0.9 gram; February 20 and 27, 0.3 gram each, a total of 5.25 grams given over a period of 45 days, or an average of 117 milligrams per day for the first course. For the second course, between June 19 and August 7, 1930, he received ten 1-grain intramuscular injections of bismuth, 5 mercury inunctions, and intravenous neoarsphenamine, 0.3 gram on June 19, and 0.6 gram doses on June 26, July 3, 10, 17, 22, 31, and August 7, 1930, a total of 4.5 grams given over a period of 49 days or an average of 92 milligrams per day. The third course consisted of intravenous neoarsphenamine (only) with 0.4 gram given on January 22 and 0.6 gram doses given on January 29, February 5, 12, 19, 26, and March 6, 1931, a total of 4 grams given over a period of 43 days, or an average of 93 milligrams per day for the third course. The last dose of the third course consisted of 0.6 gram of neoarsphenamine in 15 cubic centimeters of water and the rate of injection was about three minutes. Following the third course, exfoliative dermatitis developed with the approximate onset March 7, or one day after the last dose of neoarsphenamine. The onset was gradual, afebrile, with itching eczematous rash in groins, slowly spreading over entire body. Lesions later became very scaly, eyelids swollen, and there was a painful adenopathy. Sodium thiosulphate was given in 1-gram doses, intravenously, daily for four days and 1 gram was given by mouth three times daily for 10 days. The skin was treated locally with colloid and bicarbonate baths. On March 21, 1931, or 14 days after onset, the patient was discharged to duty though there was still a mild desquamation at this time. Dickens' test at intervals varied during all courses. In the order of frequency the results were as follows:

2 hours	4 hours	6 hours
Plus.	Plus.	Minus.
Plus.	Minus.	Minus.
Plus.	Plus-minus.	Minus.

At no time were all three specimens negative. After the onset of the dermatitis, Dickens' test failed to show the presence of arsenic. The white blood count was 8,500.

(32–1931.) A patient was infected at San Francisco, Calif., December 25, 1930. Diagnosis was made by dark-field examination of serum from lesion. Patient had received 12 injections of neoarsphenamine, 0.45 grams each injection, and 8 injections of bismuth salicylate, 1 cubic centimeter each injection, and had completed this first course on April 16, 1931. Patient was given 20 intramuscular injections of bismuth salicylate, 1-grain doses, between the dates of

August 20 and November 5, 1931, 2 injections weekly, as concurrent treatment of second course of neoarsphenamine. Patient started second course of neoarsphenamine treatment on August 20, 1931, and had received 10 injections, 0.45 grams each, by November 5, 1931. The last injection on November 5, the tenth injection, consisted of 0.45 gram of neoarsphenamine dissolved in 10 cubic centimeters of normal saline solution, the rate of injection being one and one-half minutes. At the time of the injection there was a small skin eruption about the flexor surface of each elbow which developed into exfoliative dermatitis involving both axillæ, back of neck, eyes, hands, legs, and feet. Urine was 4 plus for albuminuria. Patient was placed in bed, fluids forced, and skin kept soft with olive oil. From November 8 to November 20, the patient was given daily intravenous injections of sodium thiosulphate, 1 gram dose each injection. On November 21 the sodium thiosulphate treatment was discontinued for one week; November 28 the intravenous injection of sodium thiosulphate, 1 gram each injection, was continued and was administered daily until December 3, 1931. On December 1, 1931, the patient had no complaints and on December 4, 1931, he was discharged to duty under treatment.

(33–1931.) A patient exposed in Shanghai, China, November 10, 1930, developed a small lesion on shaft of penis, dark-field examination of which was negative for *Treponema pallidum*. The lesion healed before a second dark field could be made. On December 6, 1930, the patient complained of continuous headaches, stiffness of joints, and a sore and infected throat. Examination revealed an infected throat, with mucous patches, enlargement of glands, and Kahn precipitation test was 2 plus. On this date he was given an intravenous injection of 0.45 gram of neoarsphenamine, which was followed by a very slight reaction (severe headache), but no apparent reactions followed the 0.45 gram injections given on December 13, 20, 27, January 3, 10, and 17, 1931. Up to this time the patient had received a total of 3.15 grams of neoarsphenamine and daily mercurial inunctions for the first two weeks and the last two weeks of the course of treatment. The neoarsphenamine was given over a period of 42 days, making an average of 75 milligrams per day. On the evening of January 19, or about two days after the seventh injection of neoarsphenamine, a rash made its appearance with much itching and burning and the patient was admitted to the sick list the next morning with a diagnosis of dermatitis, exfoliative, acute (following use of neoarsphenamine). The chief complaints were itching and burning erythematous dermatitis of both forearms with some tendency to vesiculation associated with a fever of 99° F. at 9 a. m. The patient was put to bed and soothing hypertonic wet

dressings were applied to both arms and forearms, and gastrointestinal inflammation with magnesium sulphate and forced fluids were instituted. By January 21 the areas of involvement had extended to the neck and thorax and the afternoon temperature had risen to 102.6° F., pulse 106, following which he complained of intense itching and burning with stiffness of the joints and restlessness. By January 22 the involved areas extended to the legs and face while those on the arms were crusting over with the dried weeping serum to which oil of petrolatum was applied, and on this date he was given one ampule of sodium thiosulphate, intravenously.

The patient passed a restless night, and at 9 a. m. on January 23 the temperature was 100° F., pulse 110, and he appeared listless, apathetic, and exceedingly uncomfortable. His face was swollen and his shoulders and neck continuously exuded liquid which tended to crust as had occurred on the arms. At 8 p. m. his temperature rose to 103.2° F., pulse 120, and respiration 22. The urine had daily been negative for albumin, bile, and sugar; liver apparently normal in size, no nausea, and fair appetite. The progress continued with little change, improving at one location only to become exaggerated in another. Itching at times was most intense, and it was found necessary to bandage the hands to prevent damage to the skin while asleep. The afternoon temperature continued high, with some stabilization of the pulse rate. Because of the unsatisfactory facilities for constant and proper nursing and the discomfort caused by frequent overheating and cooling of the sick bay, the patient was transferred to a hospital for further treatment. By February 1 there was some slight general and local improvement; the temperature had subsided and the crusts were slowly being shed. There was a tendency, however, toward a renewal of the skin exudation, but each time less severe in degree. His appetite remained good. There was a desire to scratch the skin that was almost irresistible, necessitating rebandaging of the hands. Soothing lotions were used but without marked effectiveness even with phenol as an ingredient. On February 3 the white blood count was 8,500; polymorphonuclears, 70 per cent; lymphocytes, 26 per cent; transitionals, 2 per cent; eosinophiles, 2 per cent. The urine was negative, routine and microscopic; also negative for bile. By February 10 the patient was up in a chair for short intervals, with skin condition slowly improving. He was weak and had lost much weight. On February 20 the patient was returned to his ship with the skin condition rapidly improving. At this time he was able to go about with the help of a cane. His appetite was excellent at this time. He complained of numbness of the toes, his knee jerks were exaggerated, there was no Babinski nor ankle clonus. Eye reactions were normal. At

times he would fall down without warning. The tympanic membranes and canals were exfoliating and required irrigation. On February 21 the patient was discharged to light duty, under treatment, after 32 sick days.

(34–1931.) A patient was admitted on July 20, 1931, with an initial lesion. Dark field positive for *Treponema pallidum*. On July 22, 1931, his Kahn blood test was 4 plus. The patient was started on his first course of neoarsphenamine treatment on July 23, 1931, and completed the course of eight injections, total of 4.65 grams, on September 8, 1931. During this period he received eight 2-grain intramuscular injections of bismuth salicylate as concurrent treatment. About one week after completing this course of treatment the patient reported with a rash on the extensor surface of the forearms and in the peroneal regions which rapidly spread over his entire body. Patient was transferred to a hospital for observation and treatment. Patient states that there were a few small erythematous papules on wrists on September 8, accompanied with much itching. On September 13 same type of eruption over thighs and extensor surface of forearms, associated with much itching. At this time he had severe headaches, general aching over body, skin warm and feverish. The eruption spread generally over body. The eruption was a fine papulo-erythematous type on an erythematous base. Skin was slightly infiltrated, itched, and was stiff and hot to the touch. Throat was red and injected and papillæ of tongue were prominent. On September 16 the patient was given 1 gram of sodium thiosulphate intravenously daily for eight days. Patient states he felt much improved after the first two or three injections. Desquamations of skin, hair, and nails commenced about two weeks after appearance of eruption. In early part of eruptions there was much oozing of serum from different areas. During the interval from September 16 to December 12 there were repeated negative Dickens' tests. Urine was negative for albumin, no record of red or white blood counts. On November 23 his spinal fluid was normal. A gradual desquamation of skin has continued with no periods where condition appeared to be repeating itself. On November 16 bismuth salicylate was commenced, 2 grains being given intramuscularly biweekly for eight doses. No apparent reactions occurred. On January 19, 1932, there was a fine desquamation over an erythematous base with thickening of skin in the flexures and small fissures which are dry. Hair and nail loss is gradually being replaced. At this time patient is not acutely ill. Urinalysis repeatedly negative. Blood Kahn negative. Red blood count 4,220,000, hemoglobin 85, leukocytes 6,600, eosinophiles 2, lymphocytes 30, polymorphonuclears

61, young forms 7, Dickens' test on February 2, 1932, was 1 plus. Patient is convalescing.[7]

(35–1931.) A patient was infected at Shanghai, China, March, 1929. Diagnosis was made by a positive dark-field examination of serum from lesion. Clinical record of the patient shows that he had started his first course of arsenical treatment in March, 1929, but his treatment was discontinued because of an arsenical reaction, following his fourth injection (dosage and total not stated). On May 5, 1931, the patient started his second course of neoarsphenamine treatment after having been admitted to a hospital with diagnosis of syphilis. Laboratory findings upon admission were a 4 plus blood Kahn and negative urinalysis. Patient's 1929 intolerance was noted and he was given preliminary preparation of eight intravenous injections of sodium thiosulphate. Initial injection of neoarsphenamine was 0.18 gram of neoarsphenamine in a solution of 0.9 gram to 20 cubic centimeters of distilled water. Solution was injected as soon as prepared. About five hours after this injection the patient developed a chill, temperature 103° F., pulse 115, and a general erythema. Patient was given sodium thiosulphate with very little relief and his condition developed into an exfoliating dermatitis more marked over both hands, feet, scrotum, and back. Laboratory findings were: White blood count 15,400, neutrophiles 79, lymphocytes 18, eosinophiles 3. Patient improved and was considered recovered from the arsenical reaction on June 6, 1931.[8]

(36–1931.) A patient when given a physical examination on January 21, 1931, was found to be suffering from syphilis. His condition was diagnosed by clinical findings of scar on dorsum of penis at the corona, frontal headaches, general lymphodenopathy, huskiness of voice and a 4 plus blood Kahn test. The man admitted having had a chancroid in May, 1930, following exposure in Seattle, Wash. Treatment was immediately instituted and he received 0.45 grams of neoarsphenamin on that day, January 21, 1931. Six days later, January 27, he received his second injection of 0.45 gram of neoarsphenamin (total 0.9 gram). Three days later he was transferred to hospital complaining of pain in abdomen, gas on stomach, vertigo, insomnia, dry cough, husky voice, and tinnitus. Examination at the hospital revealed slight tenderness in mid epigastrium, hyperactive reflexes, and slightly positive Rhomberg. Urinalysis negative. On February 5 laboratory tests showed white

[7] Patient states that immediately after each injection of neoarsphenamine he would develop a deep boring type of headache, which would disappear in about two hours if he lay down. No other symptoms were noted.

[8] The medical officer states that the water used for the arsenical preparation was freshly distilled, filtered through sterilized paper, and autoclaved the morning of administration. It was also stated that 62 other injections of neoarsphenamine had been given which had the same lot number and none had caused a reaction.

blood count, 8,400; polymorphonuclears, 58; and blood Kahn 4 plus. February 15, histamine negative. February 20, gastrointestinal series negative for pathology. February 21, all symptoms subsided and antiluetic treatment reinstituted. Patient was given inunctions of mercury and five neoarsphenamin injections (total 2.55 grams) between February 21 and March 24, 1931. He was transferred to duty under treatment for syphilis on March 27. After returning to duty under treatment the patient continued neoarsphenamin treatment and he received three more injections (total 2.1 grams) which completed his first course on April 14, 1931. This first course of treatment consisted of 10 injections of neoarsphenamine, total dosage 5.55 grams, and 42 inunctions of mercury. Beginning June 24, 1931, the patient was administered 16 biweekly intramuscular injections of bismuth in 0.19 gram doses (total 3.04 grams) as intercurrent treatment.

The patient began his second course of neoarsphenamine treatment with a 0.9-gram dose on July 6, 1931, receiving a second 0.9-gram injection on July 13. Three days after the last injection he was transferred to hospital with a skin disease. Dickens' tests showed a normal elimination of arsenicals. Upon admission to the hospital the following information was obtained from questions and examinations: Admitted with complaint of rash over body and swollen ankles. About five weeks prior to admission to hospital and while he was receiving bismuth injections, the patient first noticed that his left arm was covered with multiple red areas. The following day these areas became raised and later his entire body became covered with these reddened raised areas. He had been placed on excused duty, but his condition did not improve. With the rash present the patient was started on his second course of neoarsphenamine treatment. After each injection the patient felt nauseated and lost his appetite for about two days. Following the second injection of his second course the patient's ankles became swollen and if on his feet the swelling would extend up his legs to the knees. The eyelids became puffy and swollen. Further examination disclosed the entire surface of the body covered with a dull red irregular eruption covered with irregular-sized thin to thick whitish scales. The skin was indurated, ankles swollen, and eyelids puffy. On July 20 laboratory-examination reports showed urine negative for albumin and casts; red-blood count, 4,570,000; hemoglobin, 85 per cent; white-blood count, 12,400; eosinophiles, 2 per cent; blood, Kahn negative. Seven grains of sodium thiosulphate were given intravenously on July 21 following which the urine examination showed definite traces of arsenic. Swelling of ankles subsided. Patient is confined to bed. On July 24 the diagnosis was

established as poisoning, neoarsphenamine. Condition cleared slightly on August 3, when 1 gram of sodium thiosulphate was given intravenously. August 10, skin exfoliating, no further edema of ankles, slight edema of eyelids. Sodium thiosulphate, 1 gram, given intravenously three times per week. August 18, skin continues to exfoliate and crack in flexures, hair on face and scalp falling out, patient fairly comfortable. August 25, further exfoliation of skin and loss of hair, no further edema, appetite fair, bowels normal, sodium thiosulphate by mouth (dose not stated). September 8, patient feels much improved, entire loss of hair on scalp and face, skin has ceased to exfoliate. September 15, improving steadily, sodium thiosulphate treatment has been discontinued, boric acid ointment applied to skin. September 29, two small meibomian gland cysts on lower eyelids, hordeolum right eye lanced. October 18, hair on face and scalp now reappearing; skin is assuming a more normal appearance, though still indurated. October 26, patient was surveyed for further hospitalization. November 1, hair on scalp and face coming in well. November 15, condition good. December 1, skin still has dark hue but texture better. December 16, to duty much improved.

(37–1931.) A patient was admitted with a diagnosis of syphilis on January 20, 1931, because of a generalized body rash and a 4 plus Kahn blood test. The patient began his first course of neoarsphenamine treatment on January 27, 1931, and this course continued at weekly intervals until March 19, 1931, when it was discontinued for reasons not stated. On April 9, 1931, the course was reinstituted at weekly intervals and treatments were given until May 21, 1931. The injection given on this date consisted of 0.9 gram neoarsphenamine dissolved in 30 cubic centimeters of water with a 0.6-gram dose administered. The rate of injection was about 20 cubic centimeters per minute. The total amount of neoarsphenamine administered between January 27, 1931, and March 19, 1931, was 3.4 grams and the total amount of neoarsphenamine administered between April 9, 1931, and May 21, 1931, was 4.2 grams, or a grand total of 7.6 grams for the first course. The patient received 15 injections of bismuth as concurrent treatment. About April 17, 1931, the patient reported to the sick bay with dry and cracked lips which were treated by applications of boric acid ointment and which condition cleared up to some extent. About three weeks later, May 10, 1931, his lips again became dry and cracked and the skin of his entire body became dry and blotched. By May 22, 1931, the skin rash covered his entire body and consisted of papules and scales on an erythematous base, fissured on flexor surface of elbows. His lips were fissured and crusted. He did not have any other symptoms. Patient was administered 1-gram

doses of sodium thiosulphate intravenously every other day for a period not stated. Patient recovered on July 17, 1931, and was apparently well.

(38–1931.) A patient exposed at Colon, Republic of Panama, June 18, 1931, developed penile lesions three days later which were repeatedly negative by daily dark-field examinations until July 11, when a large number of *Treponema pallidum* were found and the Kahn blood test on this date showed 3 plus. His first course of intravenous neoarsphenamine was administered over a period of 52 days, as follows: 0.45 gram (each) on July 11 and 14; 0.6 gram (each) on July 17, 21, 28, and August 5; 0.8 gram August 12; 0.6 gram August 19; 0.7 gram September 1; a total of 5.80 grams or an average of 112 milligrams per day for the course. After the diagnosis of syphilis was made on July 11 the course was started with the first four biweekly injections listed above. Dickens' test was done on all urine passed during the first 24 hours after each injection and showed elimination of arsenic within 30 minutes, strongly positive within 3 hours, and negative after 6 hours. An examination of the urine was done for albumin, sugar, casts, and bile before each injection. Kahn blood test on July 21 was 4 plus, after which the course was continued with weekly injections. A complete urinalysis was done before each injection and Dickens' test done on all urine passed 24 hours after injection. At no time was there any trace of albumin, casts, sugar, or bile found in the urine, and up to and including the ninth dose the Dickens' test showed prompt elimination of arsenic, no trace being found six hours after the injections. On September 1 he was given 0.4 gram, the tenth and last dose of the course. Following this injection routine Dickens' test failed to show any arsenic elimination in the first specimen passed after the injection. Three other patients who received the same size dose showed good elimination.

This patient, however, showed only a slight trace of arsenic in the urine for the first six hours. He complained of itching, but thought it was due to prickly heat, from which he always suffered when in the Tropics. He said that he felt well, and his temperature, pulse, and blood count were normal. Due to the fact that Dickens' test failed to show proper elimination of arsenic, he was given 1 gram of sodium thiosulphate intravenously. This was about eight hours after the neoarsphenamine was given. Two hours later he stated that he could "taste arsenic." The urine passed for several hours after that was strongly positive for arsenic, as shown by the Dickens' test. He was given 1 gram of sodium thiosulphate intravenously at 9 a. m. and 1 gram by mouth at 4 p. m. each day and urine carefully checked for arsenic, albumin, and bile. There were

traces of arsenic found in the urine occasionally for two days. On September 4 there was a slight eruption of the skin on the flexor surface of the arms resembling prickly heat. On September 5 the rash on his body still resembled prickly heat, but there was some redness of the hypothenar eminences of the hands which greatly resembled an arsenic rash. The patient felt well except for the itching, but sodium thiosulphate therapy was continued and he was given injections on this date and the following day. On September 7 he stated that he did not feel well; that he was nervous and had not slept well the night before. Examination showed the temperature to be normal and the pulse rate to be 110 per minute. There was some scaliness of the skin on both ears and a rash on the body and limbs which still had the appearance of prickly heat.

It may be mentioned that on three former occasions when in the Tropics this patient had severe attacks of prickly heat (miliaria), which were characterized by the formation of small red papules and vesicles with intense itching and burning, and in no way seemed different from the present eruption except the scaliness about the ears and redness of the hypothenar eminences of both hands. As his ship was scheduled to sail on an extended cruise, he was transferred to a hospital for further observation and treatment. The condition gradually progressed, and on September 9 the diagnosis was established as an acute neoarsphenamine poisoning. The skin became œdematous and thickened. There was a severe papular rash over the entire body; some of the papules were discrete and some confluent. The entire skin about the face, back, chest, upper abdomen, upper arms, and legs were very erythematous. Over this region the confluent papules predominated. The forearms, thighs, buttocks, and lower abdomen were not so reddened but were studded with papules. Here the discrete papules predominated. There were some desquamation, especially about the face. A few pustules appeared on the back, probably secondary to the original eruption. There did not seem to be any localized subcutaneous swelling and the œdema was generalized. Laboratory findings: Blood—hemoglobin, 75 per cent; red-blood count, 4,600,000; white-blood count ranged from 9,600 to 15,600. Differential: Neutrophiles from 71 per cent to 89 per cent; lymphocytes, 11 per cent to 28 per cent; and smear negative for malaria. Urine: Positive for pus and epithelial cells. Feces: Negative for parasites, vermes, and ova. X-ray findings—Chest: Negative for parenchymatous infiltration. Patient had two treatments of general air-cooled quartz-light ultra-violet radiations on September 8 and 9. As the patient was considered to have received the maximum amount of sodium thiosulphate deemed advisable, this drug was used at the hospital only as an external agent,

compresses and wash. In addition, the patient was given tincture of digitalis and ephedrine for weakened myocardium and lowered blood pressure, and an occasional sedative for restlessness and insomnia. By November 15, he was much improved and on November 17 he was discharged to duty, 77 days after the last injection of neoarsphenamine.

(39–1931.) A patient, who gave a negative venereal history and had no evidence of a primary lesion, was given a diagnosis of syphilis February 26, 1931, based on three positive blood Kahn reactions and palpable superficial lymph glands. His first course of treatment consisted of alternate doses of intramuscular bismuth and intravenous neoarsphenamine, given as follows:

Feb. 27, 1931	Bismuth, 0.1 gram.
Mar. 3, 1931	Neoarsphenamine, 0.3 gram.
Mar. 6, 1931	Bismuth, 0.1 gram.
Mar. 10, 1931	Neoarsphenamine, 0.45 gram.
Mar. 13, 1931	Bismuth, 0.1 gram.
Mar. 17, 1931	Neoarsphenamine, 0.45 gram.
Mar. 20, 1931	Bismuth, 0.1 gram.
Mar. 24, 1931	Neoarsphenamine, 0.45 gram.
Mar. 27, 1931	Bismuth, 0.1 gram.
Mar. 31, 1931	Neoarsphenamine, 0.45 gram.
Apr. 3, 1931	Bismuth, 0.1 gram.
Apr. 7, 1931	Neoarsphenamine, 0.45 gram.
Apr. 10, 1931	Bismuth, 0.1 gram.
Apr. 14, 1931	Neoarsphenamine, 0.45 gram.
Apr. 17, 1931	Bismuth, 0.1 gram.
Apr. 22, 1931	Neoarsphenamine, 0.45 gram.

The dilution of the neoarsphenamine was 0.9 gram to 21 cubic centimeters of freshly distilled sterile water and the rate of injection was 0.3 gram of the drug per minute. During the course of treatment no untoward reactions occurred and the patient was returned to his ship on April 22, 1931, apparently in good condition. About three days after the last injection of neoarsphenamine the patient developed a dry itching, exfoliating skin condition, both arms and the backs of legs being involved. Urinalysis on May 4 was negative for albumin and sugar with a very occasional pus cell present. May 6: Icterus index, 6; van den Bergh, less than 0.3 milligram of bilirubin per 100 cubic centimeters of blood serum; Kahn, negative. The patient was given a course of six intravenous injections of sodium thiosulphate, 1 gram each injection, between May 5 and May 18, 1931, together with bland oil to involved skin areas. The condition improved somewhat, but dryness, with some exfoliation, persisted when the patient was again transferred to a hospital for further treatment, where he recovered and was discharged to duty on June 24, 1931.

(40–1931.) A patient exposed in Los Angeles, Calif., August 30, 1930, developed a primary penile lesion which was found by dark-field examination to be positive for *Treponema pallidum* on October 11, 1930. Two days later a general adenopathy developed and on October 20 a blood Kahn reaction was 4 plus. On October 18, 1930, the patient was given his first injection of neoarsphenamine, the size of dose being 0.3 gram. In about eight hours he developed a headache, nausea, chills, pallor, and a cold perspiration. He was given 1 gram of sodium thiosulphate intravenously and suppurative treatment. Because of a potential danger of arsenical reaction he was transferred to a hospital ship for further treatment. At this time examination revealed an indurated painless lesion on the left side of penis proximal to the corona, a general adenopathy, and a suggestive macular rash over the body; a 4 plus blood Kahn reaction; negative urine. October 28: Van den Bergh qualitative was negative, quantitative less than 0.1 per 100 cubic centimeters; icterus index 4. Including the original injection the patient received one 0.3 gram and five 0.45 gram injections of neoarsphenamine and six intramuscular injections of bismuth between the dates of October 18 and December 5, 1930. Treatment was continued with bismuth and from December 23, 1930, to May 13, 1931, he received 24 intramuscular injections. On January 19, 1931, he was given an intravenous injection of neoarsphenamine which was followed by a slight reaction and another dose was given on February 3, 1931, following which no reaction was recorded. From February 7 to March 7, 1931, he received an unknown amount of specific mixture. On June 9 he was given 0.3 gram of intravenous neoarsphenamine, on June 16, 0.4 gram, and on June 23, 0.45 gram, and from June 30 to July 21, 1931, he received four doses of intramuscular bismuth.

On July 25, 1931, the patient was admitted to the sick list with a generalized eruption resistant to treatment and of almost four weeks duration (onset about June 27). The eruption covered the entire body and there was considerable itching. The report states that about 15 minutes after the last dose of neoarsphenamine (given on June 23) a swelling developed in both cubital fossæ with itching. There was also pain in the joints. Several attacks of a papulo-vesicular eruption followed the taking of a hot bath which was considered to be caused by elimination of arsenic through the skin. The skin presented the generalized typical eruption of dermatitis, exfoliative in the papulo-vesicular stage, with some desquamation. July 25, 1931, the urine was negative and the blood Kahn was negative. July 27, white blood count, 1,300. The patient was given 2 grams of sodium thiosulphate intravenously on July 27; 2 grams, July 28; and 1 gram each on July 29, 30, and 31. He was discharged to duty on August 4, 1931, 42 days after the last dose of neoarsphenamine.

(41–1932.) A patient who contracted syphilis in 1918 had received nine injections of salvarsan at that time. Patient has had no treatment since. On April 6, 1931, he was admitted to a hospital for mental observation with complaints of slight defect in hearing, forgetfulness, and unsteadiness of gait. Examination revealed small contracted pupils, very sluggish reaction to light and accommodation; slight speech defect noted, suggestive Rhomberg, knee jerks diminished on both sides. Patient has a blank facies. Laboratory findings were: Blood Kahn 2 plus; spinal fluid globulin positive; cell count 142; spinal Kahn 1 plus; gold cure negative. Patient was given eleven 1/6-grain injections of bichloride of mercury. Date of institution of second course of arsenical treatment was not stated but on May 7, 1931, the Dickens' test showed a heavy trace of arsenic in a 3-hour specimen and negative in a 6-hour specimen. This test was given after the patient had received his second 0.3 gram injection of neoarsphenamine. Patient completed course of 10 injections of neoarsphenamine for a total dosage of 5.4 grams. Date of completion of this course was not stated but it is assumed, from the report, that this course was administered over the period of May 1 to May 30, 1931.

On May 30 the patient complained of general weakness and a slight burning and itching about the face and ears. On June 2 the patient had a generalized erythema and a slight facial œdema. Patient was given 0.3 gram of sodium thiosulphate intravenously. On June 3 and June 4 the patient was given 0.45 gram and 1 gram of sodium thiosulphate, respectively. June 3, a slight exfoliation began and has grown progressively worse. At this time there is slight generalized, well established exfoliation, marked œdema of the ears, eyelids, and neck. June 9, laboratory reports were as follows: Red-blood count, 4,110,000; white-blood count, 8,800; hemoglobin, 75 per cent; polymorphonuclears, 80 per cent; lymphocytes, 19 per cent; transitionals, 1 per cent. Physical examination at this time was essentially negative but for a slight decrease in the exfoliation. Urine has been repeatedly negative. A few days later, date not stated, laboratory examination reported: Red-blood count, 3,970,000; white-blood count, 19,800; polymorphonuclears, 74 per cent; lymphocytes, 25 per cent; transitionals, 1 per cent; hemoglobin, 75 per cent. Urine was negative. Patient has had progressive improvement and recovered from the arsenical poisoning on September 15, 1931.

(42–1931.) A patient who was exposed in Manila, P. I., about February 16, 1931, developed a primary penile lesion, dark-field examination of which was positive for *Treponema pallidum*. On March 14, 1931, the blood Kahn reaction was 3 plus. From Febru-

ary 24 to April 14, 1931, he was given eight intravenous injections of neoarsphenamine of 0.45 grams each, given one week apart. A total of 3.6 grams given over a period of 49 days, or an average of 73 milligrams per day for the course. During this course he received nine intramuscular injections of a bismuth preparation as concurrent treatment. (It is noted in the report that about seven or eight years previously this patient had engaged in spraying plants with an arsenical compound). The neoarsphenamine injections noted above consisted of a 0.9 to 20 aqueous solution slowly injected by the syringe method. The results of three Dickens' tests were reported as follows: March 24, first negative, second 1 plus; April 7, first 3 plus, second 1 plus; April 14, first 4 plus, second 1 plus. The last injection of neoarsphenamine was given on April 14, and the last injection of bismuth on April 17. On April 21 there developed a fine erythemato-papular rash over forearms, arms, and on the trunk. This was markedly erythematous but associated with severe itching. These areas had cleared somewhat in the next two days, but new areas were involved, and in 72 hours the entire body surfaces were involved, with a tender hyperemic condition of the mucous membrane of the mouth. This condition, with new papules appearing and much fine desquamation associated with numerous small pustules as secondary invaders, prevailed for a period of two months. During the latter part of the period the patient ran a septic type temperature with high leukocyte count. The urine was negative for albumin except for a trace on April 27 and May 6, and the urinary output continued in normal limits. Sodium thiosulphate (1 gram) was given intravenously for four doses daily beginning on April 27, 1931. This was discontinued as it appeared to make the symptoms more apparent. From May 12 it was given in 15-grain doses orally twice daily for several days with no apparent benefit. Dickens' tests made on April 29 and May 13 were negative for arsenic. The diet was restricted in proteins and local soothing and antipruritic applications were made. On June 15 the Kahn blood reaction was negative and the skin was clear, with no evident sequelæ on June 17, 1931, 57 days after the onset of symptoms.

(43–1931.) A patient with no history of previous luetic lesions, either primary or secondary, was given a diagnosis of syphilis on March 23, 1931, as the result of 4-plus Kahn blood tests taken on March 2 and March 17, 1931. Patient received his first injection of neoarsphenamine treatment of 0.2 gram on March 24. One week later, March 31, he received his second injection of neoarsphenamine, 0.45-gram dose. The dilution of the second injection was 20 cubic centimeters of distilled water to 0.9 gram of neoarsphenamine and the rate of injection was three to four minutes. No mercury, iodine,

or bismuth was administered. On April 3, 1931, three days after the patient had received his second injection of neoarsphenamine, he developed a slight headache and a feeling of malaise in the morning and a slight sore throat in the evening. Patient stated that he noted a rash over his body and extremities that same evening. Physical examination given the next morning, April 4, 1931, revealed injected conjunctivæ; flushed face; injection of pharyngeal and buccal mucosa; diffuse, coalescent, erythematous, macular rash on body and extremities; temperature 101.6° F.; smear from throat positive for organisms of Vincent's infection. Schultz-Charlton reaction for scarlet fever negative. Sodium thiosulphate, 1 gram, given intravenously at 9.45 a. m. and a second 1-gram intravenous injection of this drug was given at 4.15 p. m. The next day, April 5, a moderate anuria and facial œdema were noted. Patient was given 1-gram sodium thiosulphate intravenously at 11 a. m. April 6, after which the rash began to fade very noticeably. April 10 the rash had completely disappeared. A fine scaling of arms and trunk was noted. April 17 patient felt well and was allowed up. Urine examination for albumin and casts were negative for the first time since onset of reaction. April 21 patient was allowed on active detail and was returned to duty on May 13, 1931, apparently recovered.

(44–1931.) A patient who gave a history of a penile lesion in December, 1930, was given a diagnosis of syphilis based on clinical and serology findings. He was given seven weekly intravenous injections of neoarsphenamine, 0.6 grams each, for a total of 4.2 grams over a period of 42 days, or an average of 100 milligrams per day for the course. The date of the last dose is not given in the report but it is stated that 24 hours after the completion of the seven weekly injections of neoarsphenamine comprising the first course the patient developed an exfoliative type of dermatitis and fever. His urine was negative microscopically and for albumin, and remained so. He was given sodium thiosulphate (1 gram) intravenously April 18, 20, 21, and 22, but because the symptoms did not improve he was transferred to a naval hospital on April 23 for further observation and treatment. At the hospital the patient stated that an eruption appeared on his arms and legs soon after the injection (seventh of a series) of neoarsphenamine, and that he did not report this condition for about a week, going about his work regularly. The rash became worse and he reported to the sick bay where he was put to bed and given the treatment noted above. He further stated that he vomited and became very sick on the day before admission, for the first time since the eruption appeared. Upon admission to the hospital, April 23, his temperature was 100° F., pulse 105, and his body was entirely covered with an acute inflamed dermatitis. The

extremities had a thick crust covering the acutely inflamed skin. The trunk and face were beefy-red in color throughout, with no normal areas of skin. The ankles were not covered by crust and the skin in this region, though acutely inflamed, was the only area of skin through which intravenous injections of sodium thiosulphate could be given during the first few days following his admission. The veins of the arm were impossible to reach because of the thick crust covering the area. He appeared acutely and critically ill on admission; his condition appeared to be mainly due to the fact that he could not eliminate through the skin. His urinary tract, however, was eliminating well on admission and continued so until recovery; there was at no time even a trace of albumin in his urine.

On the second day his temperature rose to 103° F., 101.5° F. on the third day, and 102° F. on the fourth day after admission to the hospital. The temperature on the fifth day was normal, and remained so for two days, following which it rose to 101.5° F. on the seventh day; it was normal again for two days, again rising to 101.5° F. on the tenth day. Temperature was normal on the eleventh day, but one the twelfth it again rose to 101.5° F. The temperature curve up to this time simulated somewhat that of the malarial paroxysm. Following admission to the hospital on the thirteenth day until his discharge, his temperature did not rise above 100° F., and only on three days (fifteenth, seventeenth, and nineteenth days) did it reach this degree. The pulse during the first 15 days averaged 95 and never reached higher than 110. The respiration was normal at all times. During the first week the rash showed no improvement, and the patient's general condition did not seem to be changed to any marked degree. The skin of his body was extremely painful and tender, keeping his in a state of continual shock. He was considered dangerously ill, and was placed on the dangerous list during this time. The only favorable prognostic sign was his urine, which continued to be well excreted and failed to show albumin. There was a definite change for the better in his skin condition 10 days after his admission, when the redness suddenly seemed to fade and the crusts covering the skin of the upper extremities loosened and could be peeled off, showing normal skin underneath. The upper extremity was the first part of the body to clear up completely, followed by the face and trunk, respectively. The lower extremity was much more resistant and was the only part involved during his last month at the hospital; crusts over this area remained tenacious, and it was difficult for the patient to get up and around, thus tending to delay his remaining strength. These parts finally did clear up, however, and on his discharge from the hospital his skin seemed entirely clear and most of his strength

had been regained. His spirit during his entire stay in the hospital was most admirable and was, perhaps, the deciding factor in saving his life. The treatment in this case consisted of giving intravenous injections of sodium thiosulphate, dose 1 gram for the first 10 days, then 1 gram on alternate days for 10 days, then at 5-day intervals in the same dosage until two weeks prior to his discharge. He received daily baths in a solution of sodium bicarbonate, followed by application to the entire body of cottonseed oil. The bowels were kept freely open by saline catharsis at regular intervals and other laxatives as necessary. Fluids were pushed and were handled well by the kidneys. He was discharged to duty on July 20, 1931, after 93 sick days.

(45–1931.) A patient was exposed to infection of syphilis on November 11, 1930, at Philadelphia, Pa. Diagnosis of syphilis was made on December 30, 1930, and was based upon the physical appearance of the initial lesion and a secondary macular rash. The Kahn blood test was negative at this time and there was no record of a dark-field examination. From January 8 to February 19, 1931, the patient received the total amount of 4.7 grams of neoarsphenamine as his first course of treatment. From January 2 to March 13, 1931, he received twenty-one 2-grain intramuscular injections of bismuth as concurrent treatment. Patient began his second course of neoarsphenamine treatment on March 31, 1931, and had received nine weekly injections of 0.45 gram doses each (total 4.05 grams) when he was transferred to the hospital on May 28, 1931, because of a fissure-like sore on the internal surface of the upper lip which appeared slightly indurated and rather raw around the edges. The patient also had a marked dermatitis of the hands which he claimed was due to the use of lye aboard ship. Kahn blood test was positive at this time. Physical examination otherwise negative. Dermatitis condition improved by the application of calamine lotion and vaseline. On June 4, 1931, the second course was reinstituted and the patient received 0.5 gram of neoarsphenamine. One week later, June 11, the patient received another 0.5 gram administration of neoarsphenamine, the dilution of which was 0.1 gram of neoarsphenamine to each 2 cubic centimeters of sterile distilled water with the rate of injection not stated. The total dosage of the last two injections was 1 gram and the total dosage administered for the second course was 5.05 grams. On June 17, six days after the last injection, the patient's diagnosis was changed from syphilis to dermatitis, chemical, because there was an acute exacerbation of dermatitis on his hands. The desquamating dermatitis of both hands improved by the applications of bland ointments. The patient received eighteen 2-grain doses of bismuth between the dates of May 29 and July

31, 1931. On August 5 the patient was returned to duty with the dermatitis entirely healed and further treatment not considered necessary.

(46–1931.) A patient who became infected on September 10, 1931, was given a diagnosis of syphilis on October 1, 1931, because of the finding of *Treponema pallidum* in the serum from the initial lesion appearing on glans penis. Neoarsphenamine treatment was immediately instituted that day, October 1, 1931, and the course of treatment was continued for seven injections, this injection being administered on November 12, 1931. The total dosage administered for this first course was 3.9 grams. The patient was given seven injections, 0.13 gram doses, of bismuth salicylate between September 30 and November 10, 1931, as concurrent treatment (total 0.91 grams). About five days after the patient received his last injection of neoarsphenamine, he reported to the sick bay complaining of itching about axillary region and back of hands. He had a faint rash over his body. Patient was given daily injections of 1 gram of sodium thiosulphate for four days, but symptoms became more severe and edema of hands and face appeared. The patient was transferred to a hospital on November 25, 1931. Upon admission to the hospital the patient had generalized redness and dryness of skin. Later, numerous small ulcerated areas, varying in size from that of a pinhead to a small pea, appeared about the back and arms. These cleared up in about 10 days. Upon admission the patient was administered three 1-gram doses of sodium thiosulphate intravenously the first week. The skin condition became worse. The skin was cleansed with starch water, dried, and cold cream generously applied daily. From December 5, 1931, to January 1, 1932, the patient was given intravenous injections of 0.5 gram doses of sodium thiosulphate. These smaller doses caused considerable improvement. The medical officer reported the following interesting observation: "The urine, which showed a trace of arsenic became negative under sodium thiosulphate. Intradermal injections of sodium thiosulphate caused no local change. The patient continued to improve and was returned to duty on February 26, 1932, apparently well. Kahn blood test was negative at this time."

(47–1931.) A patient was exposed to infection on February 5, 1931, at Chungking, China. Diagnosis was withheld because the patient had a typical ulcerated lesion in coronal sulcus of penis and dark-field examinations were negative for *Treponema pallidum*. Due to the appearance of this lesion, which looked so much like a syphilitic lesion, typical in all respects, the patient was placed under close observation. On February 21 a gland puncture was made and the result was negative. March 1, a rash appeared over the entire body,

and he had mucous patches in his throat. Kahn precipitation test was 4 plus this date. Diagnosis of syphilis confirmed and made this date. First course of neoarsphenamine treatment was instituted on March 5, 1931, and the patient received 0.45 gram. On March 12, the patient received his second injection of 0.45 gram of neoarsphenamine. Total dosage to date was 0.9 gram. Patient was given daily mercurial inunctions for the first two weeks of treatment and two injections of bismosol as concurrent treatment. Shortly after the administration of neoarsphenamine on March 12, 1931, the patient had a slight headache and chill. Three days later, March 15, the patient was admitted to the sick list with complaints of severe chill followed by feverish sensations and a nonsymptomatic rash about the wrists. Physical examination revealed a punctate erythematous rash about the wrists, back, and palms of hands which did not itch or burn. Patient had no coryza or bronchial symptoms, but he did complain of some frontal headache. Temperature, 102° F.; pulse, 86; respiration, 24. Some prominence of border papillæ of tongue. Patient placed in bed and purged with calomel, forced fluids. The next day, March 16, the rash extended to arms and chest, with very little involvement of face but with some slight œdema of the lax tissues below the eyes. Rash on arms and chest tends to be macular, with some tendency to form rings, and continued to be locally symptomless. Laboratory examinations as follows: White-blood count, 6,950; hemoglobin, 80 per cent; polymorphonuclears, 66; lymphocytes, 29; eosinophiles, 2; transitionals, 2. Urine negative for bile and albumin, alkaline in reaction. Temperature, 104.2° F., pulse, 100; respiration, 30. Patient was up and about on March 19, but complained of a burning sensation in both eyes, with the pain tending to increase. Boric-acid packs were applied with good results. March 20, the patient had no complaints. Again on March 21 the patient complained of slight burning sensation in both eyes, which was relieved by acetylsalicylic acid, 5 grains. On March 23, 1931, the patient was returned to duty and was considered fully recovered.

(48–1931.) A case (V. A. P.) in which luetic history was not available was admitted to a naval hospital on June 13, 1931, with a diagnosis of acute rheumatic fever, at which time examination also revealed conjunctivitis, chronic tonsillitis, carious teeth, and pains and aches in both legs. A Kahn blood reaction was 3 plus on June 15, and another test given on July 9 was negative. From July 10 to August 14, 1931, he was given six 0.2 gram weekly intramuscular injections of bismuth and he was given intravenous neoarsphenamine, 0.3 gram, July 14, and 0.6 gram (each) on July 21, 28, August 4, (August 11 skipped because of "sickness") and

August 18, 1931, a total of 2.7 grams. Following the last injection of neoarsphenamine, that given on August 18 (exact elapse of time not stated), the patient developed a mild eczematous condition of the face. This eczematous condition gradually extended and involved the abdomen (mild), flexor surface of both elbows and both groins. He was given 1 gram intravenous injections of sodium thiosulphate on August 31, September 1, and September 2, 1931. The urine tests were all negative albumin and sugar. No Dickens' test was made. The white-blood count made on July 21, was 7,460. Differential: Band forms, 7 per cent; segmented, 64 per cent; eosinophiles, 2 per cent; mononuclears, 5 per cent; lymphocytes, 22 per cent. At the time of the report, which is dated September 5, 1931, the patient was much improved after the discontinuance of neoarsphenamine and intravenous sodium thiosulphate.

(49–1931.) A patient (V. A. P.) who gave a history of syphilis contracted in 1919, was admitted to a naval hospital August 9, 1930, and on August 10, 1930, his blood Kahn reaction was 2 plus. There was a history of the patient receiving treatment for a short period in 1919, and again in May, 1930, when a Wassermann blood test was found to be 4 plus. The amount and type of this previous treatment is not known. From August 26 to October 7, 1930, he received one 0.3 gram intravenous injection and six 0.6 gram intravenous injections of neoarsphenamine, a total of 3.9 grams. On October 28 the Kahn blood test was negative. On December 2, 1930, he was given another 0.3-gram injection of intravenous neoarsphenamine and 0.6-gram doses on December 9, 1930, January 13, and January 20, 1931, a total of 2.1 grams. As concurrent treatment he had received eleven ½-grain intramuscular injections of mercury bichloride (dates not stated). The last dose of neoarsphenamine was given on January 20, 1931, and on that date the patient developed an eczematous condition beginning on the hands and wrists. This condition gradually and slowly extended to the forearms and subsequently there developed a similar condition on the upper anterior surface of the thighs. One gram of sodium thiosulphate was given intravenously on February 7, 8, and 9, together with cathartics. Prompt improvement in the condition was noted. The urine tests on this case were negative throughout. The Dickens' test was positive for the first 3-hour specimen and negative for the second 3-hour specimen on January 23, 1931. No further results of this test were reported.

(50–1931.) A patent who was exposed in Pensacola, Fla., in September, 1931, developed a penile lesion which was positive for *Treponema pallidum* by dark-field examination, and on September 20, 1931, a Kahn blood reaction was 4 plus. He was given six 1 cubic

centimeter weekly intramuscular injections of bismosol from September 17 to October 23, 1931; six 2/5-grain weekly intramuscular injections of succinimide of mercury from September 22 to October 27, 1931; and six 0.6 gram weekly intravenous injections of neoarsphenamine from September 24 to October 29, 1931. The neoarsphenamine was diluted 0.6 gram to 10 cubic centimeters of distilled water and the rate of injection was 4 cubic centimeter of this solution per minute. Five days after the last injection of neoarsphenamine the patient reported to the dispensary complaining of lassitude, nausea, and inability to work. His temperature was 98.3° F., pulse 90, and respiration 22 at this time. The examination was negative except for some slight scaling of general body surface, and Dickens' test was 1 plus on the first specimen of urine voided. He was put to bed, given 1 ounce of magnesium sulphate, soft diet, and 1 gram of sodium thiosulphate intravenously. About three days later, or eight days after the last dose of neoarsphenamine, the patient was considered to have recovered from the reaction, all specimens of urine were negative for albumin, and the Dickens' test was negative for arsenic at that time.

(50A–1931.) A patient was infected in New York, N. Y., on June 20, 1931. A typical lesion appeared on the dorsum of penis. Microscopical examination of the serum from the lesion proved negative for *Treponema pallidum*. A Kahn blood test showed a 2-plus reaction. Patient was then given five intravenous injections of neoarsphenamine from September 14 to October 24, 1931, sizes and dates of doses not stated. Patient was also given 13 cubic centimeters of bismosol, the sizes and dates of administration of which were not stated. Following the fifth injection (October 24) of neoarsphenamine the patient developed exfoliative dermatitis and all treatment was discontinued. Intravenous sodium thiosulphate was given twice weekly, sizes of doses not stated. On November 6, 1931, the patient was transferred to a naval hospital for treatment. Chief complaints on admission to the hospital were slight itching rash on arms and chest, which is increasing progressively and which is of about 2-weeks' duration; patient is hoarse and has some difficulty in talking, pupils react to light and accommodation sluggishly, internal mucous membrane of lips has numerous small ulcers, two small necrotic ulcers on the right tonsil, few cervical lymphnodes, the rash is a diffuse erythematous eruption on the flexor surface of both forearms and elbows, arms, axillæ, side of chest and abdomen with patches of desquamation of whitish, flaky character. On November 5, the temperature was 99.2° F.; pulse, 80; and respiration, 18. The skin was covered with linseed oil. Dobell's solution as a mouth wash and gargle, calomel and saline purgative. November

9, Kahn blood test was negative. November 12, the lesions are clearing up, linseed oil treatment discontinued. Applications of equal parts of ammoniated mercury and zinc oxide ointments started. November 17, the only lesions remaining are two small secondary infected ulcers on flexor surface of both elbows and right axilla. November 18, mouth lesion has disappeared. Treatment continued. Dickens' test showed a very faint trace in the first specimen, each succeeding specimen for the six hours being negative. November 25, all lesions have practically disappeared and the patient is considered about ready for duty. Treatment continued. December 3, all lesions have disappeared. December 4, 1931, the patient was sent to duty well after 28 sick days in the hospital.

(51–1931.) A patient who was exposed in Newport, R. I., on July 15, 1931, was given a diagnosis of syphilis based on a Kahn blood test which was 4 plus on August 12, 1931. He was given 0.3 gram of neoarsphenamine intravenously on August 19, 1931, and 0.6 gram of the same drug intravenously on August 26, 1931. The report states that he received no other concurrent treatment. The last dose of neoarsphenamine consisted of 0.6 gram in 15 cubic centimeters of triple distilled water, and the rate of injection was stated to have been 10 minutes for the 15 cubic centimeters. Two days following this injection the patient complained of severe headache, tightness in the chest, and fever. His temperature was 102° F., and there were generalized papular luetic-like secondaries present on admission. Three days later he developed a generalized blotchy erythema over the trunk and extremities, with none on the hands or feet. These blotchy lesions did not become macular or papular, did not exfoliate, and cleared up after five intravenous administrations of 1 gram of sodium thiosulphate. Five days from onset, or seven days after the last injection of neoarsphenamine, the patient was considered to have recovered from the reaction, though the secondary lueticlike lesions persisted.

(52–1931.) A patient who was exposed in Panama City, Canal Zone, on February 22, 1931, developed penile lesions. On March 16, 1931, he was given a diagnosis of chancroid due to the inability to find *Treponema pallidum* in repeated dark-field examinations. On April 6, 1931, he developed a 4-plus Kahn precipitation reaction of the blood, and the diagnosis was changed to syphilis on the basis of genital sore, general lymphadenopathy and a positive blood reaction. Antileutic treatment was begun on April 14, 1931, when he was given 0.3 gram of neoarsphenamine intravenously, diluted with 6 cubic centimeters of newly distilled water. He felt no effect from this injection. He was given 0.45 gram of the same lot number intravenously on April 21 and April 28, 1931, each diluted with 9 cubic

centimeters of newly distilled water. Following the last injection he felt badly, complaining of vertigo and faintness, and he was distinctly pale. Epinephrin (10 minims) was administered at once, hypodermically, and in 15 minutes he made a good symptomatic rally. On the following morning (April 29) he noticed an erythema over the legs to the thighs and less intensely on the arms. There were no constitutional symptoms or signs. The temperature, pulse, and respiration were normal. The Dickens' test showed 3 plus in the 3-hour specimen. He was put to bed, given catharsis and forced fluids, and an intravenous injection of 0.5 gram of sodium thiosulphate. Following this the Dickens' test was weakly positive. The white-blood count was 6,850. Differential: Polymorphonuclears, 71 per cent; lymphocytes, 21 per cent; mononuclears, 5 per cent; eosinophiles, 1 per cent; transitionals, 2 per cent. On April 30 the erythema was fading. The urine output was 1,025 cubic centimeters in 21 hours, with normal qualitative urinalysis. He was given another intravenous injection of sodium thiosulphate on this date, and the Dickens' test was still faintly positive. In view of this reaction, the patient was transferred to a naval hospital for further antiluetic treatment on April 30, 1931, and at this time there was still a slight reddening of the skin of both legs. He was continued on forced fluids and given daily 1-ounce doses of magnesium sulphate. Recovery from the reaction was complete on May 6, 1931, eight days after the last dose of neoarsphenamine.

(53–1931.) A patient who was exposed at Long Beach, Calif., on or about July 17, 1931, reported to the sick bay on August 10, 1931, with a typical buttonhole lesion at the meatus, dark-field examination of which was positive for *Treponema pallidum*, and on September 14, 1931, a blood Kahn was 4 plus. His antiluetic treatment consisted of intravenous neoarsphenamine, 0.4 gram August 13; 0.5 gram August 19; and 0.6 gram (each) on August 26, September 2, 9, 16, and 30; and 0.1 gram intramuscular bismuth injections on September 7, 14, 21, 28, and October 26, 1931. As noted above, the patient's first injection of neoarsphenamine was given on August 13, 1931, and about three days later he reported to the sick bay complaining of a headache and a rash on his body. Examination revealed an acute coryza with a temperature of 99° F. There was a fine papular rash on the arms and chest which resembled measles. There was no evidence of albumin in the urine nor any evidence of retention of arsenic as shown by the Dickens' test, and the white blood count was normal. The patient was put to bed in the isolation ward, given saline catharsis, and sodium thiosulphate externally and intravenously. His antiluetic treatment was continued and he received neoarsphenamine injections on the dates noted above. On September 23,

he was admitted to the sick list with a diagnosis of urticaria, at which time his chest, arms, and back showed a fine macular, closely set rash. His temperature at this time was normal and the urine was negative. He was given saline catharsis, and sodium thiosulphate externally. The rash cleared up and the patient was sent to duty on September 25, 1931. He received seven injections of neoarsphenamine, the last of which was administered September 30, 1931, and there was no evidence at any time from the Dickens' test of retention of the arsenic.

On October 28, 1931, or 28 days after the last dose of neoarsphenamine, the patient returned to the sick bay complaining of a rash on his body. Examination revealed a fine, closely set papular rash covering the chest, abdomen, and thighs. The skin was dry and the rash was a brownish color. In a few days there was a desquamation of fine scales from the papules. There was no rise in temperature nor any objective symptoms except itching. The urine was negative for albumin and sugar. The white blood count showed a total of 9,000 white cells, and the differential showed 54 polymorphonuclears, 37 lymphocytes, 2 large mononuclears, and 6 eosinophiles. The patient was put to bed in the isolation ward and given saline catharsis, sodium thiosulphate (local applications and intravenous injections), and ⅜-grain ephedrine twice a day for three days. The rash was beginning to disappear and desquamate and the itching was about stopped when on November 2, 1931, he was transferred to a hospital ship for further treatment and disposition. The hospital ship reported the physical examination to be essentially negative except for the skin which showed an exfoliating dermatitis with evidence of scratching. The urine was negative, Kahn blood test was negative, and bromsulphthalein, van den Bergh and icterus index tests all within normal limits. Complete blood counts showed red blood count, 4,650,000; white blood count, 6,800; hemoglobin, 70 per cent; neutrophiles 67, lymphocytes 29, and eosinophiles 4. He was given 1 gram of sodium thiosulphate each day for five days with sterile oil applied to the skin. The skin condition showed no improvement under this treatment and on November 9, 1931, because of clinical appearances, he was placed on scabies treatment after which the skin condition promptly cleared up. The patient was discharged to duty in good condition on November 17, 1931.

(54–1931.) A reaction occurred in a case receiving arsenical treatment for "yaws." The patient, a native of Guam, had no lesions but had repeatedly positive Kahn blood tests. The reaction occurred after the patient had received his seventh 0.6 gram injection of neoarsphenamine of the first course of treatment (total of 4.2 grams). The dilution of this injection was 0.6 gram of neoarsphenamine dis-

solved in 10 cubic centimeters of sterile distilled water and the rate of injection was stated as about two minutes. About 24 hours after the injection the patient complained of fever. Examination revealed a temperature of 99.4° F., with his entire body feeling very hot. A generalized severe exfoliative dermatitis was noted. Liquid petrolatum was applied to the skin and intravenous injections of sodium thiosulphate, 1 gram twice daily was administered. Duration of treatment was not stated. Complete recovery was made in six weeks.

(55–1931.) A reaction occurred in a case receiving arsenical treatment for "yaws." The patient, a native of Guam, had typical lesions and positive Kahn blood tests. The reaction occurred after the patient had received his sixth injection of neoarsphenamine of his first course of treatment (total 2.85 grams). The dilution was 0.6 gram of neoarsphenamine dissolved in 10 cubic centimeters of sterile distilled water and the rate of injection was stated as about two minutes. About 14 days after the injection the patient complained of fever and chilly sensations. Examination revealed a temperature of 99° F., severe exfoliative dermatitis with stiffness, exudation and swelling of the skin, and eyes injected. One gram intravenous injections of sodium thiosulphate were administered twice daily. Duration of treatment was not stated. At time of submission of the report, September 29, 1931, desquamation continued but the patient's temperature was normal and the skin condition was greatly improved.

(56–1931.) A reaction occurred in a case receiving arsenical treatment for "yaws." The patient, a female native of Guam, had typical lesions and positive Kahn blood tests. On September 23, 1931, the patient received her eighth injection of neoarsphenamine of the first course of treatment (total of 4.05 grams). The dilution was 0.6 gram of neoarsphenamine dissolved in 10 cubic centimeters of sterile distilled water and the rate of injection was stated as about two minutes. Thirteen days later, October 6, 1931, the patient complained of fever and chilly sensations. Examination revealed a temperature of 101° F., a swelling of the face and arms and severe exfoliation of the face, neck, and arms. Patient was administered daily intravenous injections of sodium thiosulphate, 1 gram doses. Duration of treatment was not stated. On October 28, 1931, the patient had not recovered but a marked improvement was noted in desquamation.

(57–1931.) A reaction occurred in a case receiving arsenical treatment for "yaws." The patient, a female native of Guam, had typical mother yaws lesions and positive Kahn blood tests. The reaction occurred on October 7, 1931, after the patient had received her fifth injection of neoarsphenamine of the first course of treatment (total

2.25 grams). The dilution was 0.6 gram of neoarsphenamine dissolved in 10 cubic centimeters of sterile distilled water and the rate of injection was stated as about two minutes. Eleven days after the injection, October 18, the patient complained of fever and chilly sensations. Examination revealed a temperature of 101° F., swelling of the face and arms, and severe exfoliation of the face, neck, and arms. One gram intravenous injections of sodium thiosulphate were administered daily. Duration of treatment was not stated. Complete recovery was not made at the time of submission of the report but a marked improvement was noted in desquamation.

Miscellaneous Effects Following the Administration of Arsenical Compounds

Damage to the liver.—There was one instance of a severe reaction of jaundice which was reported during the year 1931 following the use of neoarsphenamine. The case follows:

(58–1931.) A patient who acquired infection in Los Angeles, Calif., through exposure about January 3, 1930, developed an initial lesion on the shaft of penis on February 15, 1930, and diagnosis of syphilis was made by dark-field examination which was positive for *Treponema pallidum.* Between February 15 and March 26, 1930, he received 3 intramuscular injections of mercury succinimide (one-fifth grain each) and 5 intravenous injections of neoarsphenamine (2 of 0.45 gram and 3 of 0.6 gram). Between April 23 and July 30, 1930, he received 10 intravenous injections of neoarsphenamine (1 of 0.45 gram and 9 of 0.6 gram) and 8 injections of mercury succinimide (one-fifth grain each). The report states that on May 15, 1930, he received 0.3 gram of neoarsphenamine, which appears to be in addition to the course just previously mentioned. Between March 11 and July 14, 1931, he received 1 intravenous injection of neoarsphanamine of 0.45 gram and 15 intramuscular injections of bismuth salicylate. On September 2, 1931, he was given an intravenous injection of neoarsphenamine of 0.45 gram and on September 9, 1931, he was transferred to a hospital ship with a notation that "he reacts badly to neoarsphenamine." The blood Kahn reactions as recorded were April 2, 1930, negative; April 13, 1931, 4 plus; July 13, 1931, 4 plus; and September 10, 1931, 4 plus. Upon admission to the hospital ship the patient had no complaint but stated that after the injection of 0.45 gram of neoarsphenamine given on September 2, 1931, he developed a rise in temperature, headache, and general malaise. His physical examination at this time was essentially negative except for a slight tenderness over the mid-epigastrium and some general adenopathy; scleræ and skin were clear; heart not enlarged, sound normal; and blood pressure was 114/70. The urinalysis was entirely

negative and on September 11, 1931, he was given 0.1 gram of bismuth intramuscularly. The patient complained of some gastric distress and loss of appetite, but otherwise appeared to be in good condition. After careful consideration of the case he was given, on September 16, 1931, an intravenous injection of 0.3 gram of neoarsphenamine, administered in 7 cubic centimeters of freshly distilled, sterile water, injected slowly (one and one-half minutes). (Twenty-two other men received injections at the same time, using the same kind of materials and in the same manner, without any untoward effects whatever.)

The injection given this patient was followed in about 45 minutes by an intravenous injection of 1 gram of sodium thiosulphate, and no immediate reaction was noted. Because of gastric symptoms and persisting 4 plus Kahn reaction, a spinal examination was done on September 17, 1931, the results of which were cell count 2, globulin negative, Kahn negative, and colloidal gold no change. On September 18, 1931, he was given 0.1 gram of intramuscular bismuth. On September 21 he continued to complain of indefinite gastric distress, his tongue was coated, and the scleræ showed a slight icterus. On September 23 a definite jaundice of scleræ and skin developed; van den Bergh: Qualitative, direct immediate reaction; quantitative, 3.6 milligrams per 100 cubic centimeters; icterus index, 60. There were clay colored stools and the urine was positive for bile. The patient was placed on a fat-free, high carbohydrate diet, forced water and sodium phosphate each morning. Sodium thiosulphate was given in 1-gram doses intravenously each day for five days. On September 28 the van den Bergh was 5 milligrams per 100 cubic centimeters and the icterus index was 50. On October 5 the icterus was subsiding—van den Bergh, 3.3, and the icterus index, 21.5. On October 6 the urine was negative for bile. On October 12 the patient was feeling well, the jaundice had cleared; van den Bergh, 1.5 milligrams, and the icterus index, 6. October 26, van den Bergh, 1.5 milligrams per 100 cubic centimeters, icterus index, 8.8. November 3, bromsulphthalein test showed light color after 5 minutes and negative after 30 minutes. By November 4 the patient had apparently recovered from his hepatic attack and was in a very good physical condition, and on November 6, 1931, he was discharged to duty, 51 days after the last injection of neoarsphenamine, with a recommendation that arsenical preparations be considered as contraindicated in the future treatment of this case.

Aplastic anemia.—During the year 1931 there was one nonfatal case reported from this cause following the use of neoarsphenamine. This reaction was classed as severe. Case history reported is as follows:

(59–1931.) A patient was exposed to infection about April 29, 1931, at Honolulu, Hawaii. Diagnosis was made by clinical symptoms of headache, mild fever, general malaise, generalized typical macular syphilide, generalized adenopathy, and an indurated painless nonulcerative lesion on mucous membrane of prepuce near coronal sulcus which apparently was the initial lesion. Diagnosis was confirmed on June 30, 1931, by a 4 plus Kahn blood test. The first course of treatment was started on June 30 by an administration of 0.3 gram of neoarsphenamine and the course was continued for seven injections with the seventh injection, 0.6 gram, being administered on July 21, 1931 (total 3.75 grams). Dilution and the rate of injection of the last dose was not stated. As concurrent treatment the patient was administered seven 1-cubic-centimeter intramuscular injections of bismuth from July 1 to July 23, 1931. After the administration of the first injection on June 30 the patient had a slight reaction with complaints of headache and a temperature of 102° F. Patient was given an intravenous injection of 1 gram of sodium thiosulphate. The course of treatment was continued and the patient had no other complaints during the course. On July 23, 1931, two days after the last or seventh injection, the patient complained of headache, sore throat, and pain in the joints. Temperature 101.2° F., pulse 92. Laboratory examination showed: White blood count, 5,150; neutrophiles, 27; lymphocytes, 64; eosinophiles, 7; and mast cell, 2. July 25, 1931, the laboratory examination was as follows: Red blood count, 2,680,000; white blood count, 5,500; hemoglobin, 70 per cent; and a differential count of neutrophiles 19 (premyelocytes, 4; myelocyte, 10; juvenile, 5), lymphocytes, 75; eosinophiles, 4; mast cells, 1. On July 27 the patient presented a local picture of extensive Vincent's angina involvement about the gums, which were ulcerated in areas, and involvement of the tonsils. In view of the clinical picture of low grade septic fever, disproportionately poor pulse, profound prostration and weakness, associated with anginal ulceration of oral cavity and tonsils and on consideration of laboratory picture of practically total absence of granular cells from the blood and a rapidly appearing secondary anemia of moderate degree, there can be no doubt that a marked bone marrow depression has taken place. The cause of the above condition is most probably due to the recent extensive treatment with arsenicals, and the condition can be described as an agranulocytic angina. It appears that at present the bone depression has not gone far enough to produce a very severe secondary anemia, although the symptoms are very alarming. The reaction in this case was a cumulative one, probably due to either an idiosyncrasy or to a too-vigorous treatment with arsenicals. The first symptoms appeared after three weeks of

intensive treatment and the symptoms subsided rapidly after stopping arsenicals. Sodium thiosulphate was not used after the first dose. The patient improved under corrective diet and cod-liver oil and was returned to duty on September 18, 1931, apparently well. The blood picture at time of return to duty was as follows: Red-blood count, 4,520,000; white-blood count, 4,850; hemoglobin, 90 per cent; neuthrophiles, 57; lymphocytes, 34; transitional, 1; eosinophiles, 8; nuclear index, 6.1.[9]

Herxheimer reactions.—It is difficult to determine a line of demarcation between a certain type of vasomotor-phenomena reaction and a Herxheimer reaction. However, one reaction occurred in 1931 that appears to be of the Jarisch-Herxheimer type and is included here under the heading of "Herxheimer reactions." This reaction has been considered to be of a mild type. Case history is as follows:

(60–1931.) A patient whose time and place of exposure was not stated, reported to the sick bay on April 6, 1931, with a small ulcer on coronary sulcus of penis and had a general adenopathy. Dark-field examination was positive for *Treponema pallidum* and on the following day (April 7), after the usual examination and preparation, the patient received 0.3 gram of neoarsphenamine intravenously at about 11 a. m. There had been no previous treatment. No complaints were registered immediately after the treatment but about 5 p. m. the same day the patient reported to the sick bay complaining of headache and chill. At 6 p. m. he was seen by a medical officer, at which time his temperature was 105.6° F. and his pulse was 120. He was rational and his body was covered with a generalized maculo-papular eruption which had not been evident prior to the injection of neoarsphenamine. The patient was immediately transferred to a naval hospital, where, according to a later report, elimination and ice cap were prescribed. The next morning the temperature, pulse, and respiration were normal and the rash had disappeared. Routine Dickens' test showed no elimination of arsenic, the urine otherwise was negative. The Kahn blood test at this time was returned 4 plus. One week later the patient returned to duty aboard his ship and antiluetic treatment was continued with potassium bismuth tartrate intramuscularly.[10]

[9] The medical officer reported "it does not appear that the particular preparation of neoarsphenamine was responsible for the reaction."

[10] The reporting medical officer describes the technique used as follows: "Enough neoarsphenamine is made up for six injections (0.6 gram being our maximum dose). Four ampules (0.9 each) of crystalline neoarsphenamine are dissolved in 60 cubic centimeters of distilled water, making a mixture of 0.6 gram to 10 cubic centimeters of solution. This being this patient's initial injection, he received only 0.3 gram, or 5 cubic centimeters, of solution. It is interesting to note that at least five others received an injection of the same mixture and the same lot number without untoward effects.

Reactions of minor importance.—The following reaction, classed as a mild type, has been included under this heading because of the lack of information. The reaction was considered to be due to neoarsphenamine treatment.

(61–1931.) A reaction occurred which probably should be included under the heading of exfoliative dermatitis but the report did not contain sufficient data for classification. The patient was admitted to the sick list, as shown by an N. M. S. Form F card, with a diagnosis of "Dermatitis exfoliativa (probably due to neoarsphenamine treatment)" on July 11, 1931, and was returned to duty August 5, 1931, after 25 sick days. The report shows that the condition cleared up upon the administration of sodium thiosulphate on July 11, 13, 16, 18, 19, and 20, and local applications of boric acid ointment. After being discharged from the sick list he was transferred with his records to another activity and the reply to a questionnaire in this case was somewhat indefinite.

REPORT ON TRAFFIC INJURIES, DESTROYERS, BATTLE FORCE, FOR FISCAL YEAR 1932 [1]

By W. L. MANN, Captain, Medical Corps, United States Navy

The following are some of the interesting features that were elicited from an analytical study of the injuries and disabilities due to traffic accidents caused by motor vehicles operated or used by the personnel of destroyer squadrons, battle force, during the fiscal year ending June 30, 1932:

(*a*) *Rank and rating.*—The following table shows the ratio of admissions to the sick list in relation to the rank or ratings of owners of motor vehicles. If the injuries due to accidents, classified as unavoidable, were eliminated the difference in the ratio for the ratings would show even a greater dispersion. For instance, the elimination of the injury to a chief warrant officer, caused by the unavoidable stalling of an automobile on a mountainous road, would advance the ratio of the officer group to 1–231, or almost an irreducible minimum for these hazardous environs.

Rank or rating	Complement of destroyers, battle force	Total injured (admissions)	Number owning motor vehicles [a]	Ratio of injuries to owners of motor vehicles [a]
Commissioned, chief warrant and warrant officers	487	3	462	1 to 154.
Chief petty officers	559	3	429	1 to 143.
Petty officers, first class	1,083	13	610	1 to 47.
Petty officers, second class	973	15	292	1 to 19.
Petty officers, third class	522	14	104	1 to 7.
Nonrated men	2,406	40	160	1 to 4.

[a] Ownership estimated, based on census of U. S. S. Melville.

[1] Because of lack of space, this report has been abbreviated.

(*b*) *Age incidence.*—The average age was approximately 26½ years. Arranged in age groups, they are as follows:

Age group	Injuries	Per cent
23 years or less	37	42.05
24 to 28 years	24	27.27
29 to 32 years	13	14.77
Over 32 years	12	13.64
Age not recorded	2	2.27

(*c*) *Final disposition.*—Those admitted to the sick list were disposed of (either by ship or by hospital to which patients were transferred) as indicated in the succeeding table:

Disposition	Number	Sick days on board	Sick days in hospital	Total sick days	Average number of sick days per case
Died:					
With sick days	[1] 1	76	77	153	.....
Do	1	2	0	2	.....
Without sick days	11	0	0	0	.....
Total	13	78	77	155	11.9
Returned to duty	65	146	2,361	2,507	38.6
Invalided from the service	5	15	875	890	178.0
Remaining on the sick list	3	0	[2] 392	392	130.7
Discharged at expiration of enlistment	1	0	37	37	37.0
Dishonorable discharge	1	0	110	110	110.0
Total	75	161	3,775	3,936	52.5
Grand total	88	239	3,852	4,091	47.6

[1] Fracture fourth, fifth, sixth, and seventh dorsal vertebræ with resultant paralysis.
[2] Estimated according to reported prognosis.

(*d*) *Character of injuries.*—The location and type of disabilities are tabulated as follows:

Type of injury	Location of injury								Total
	Upper limbs	Lower limbs	Spine and back	Chest	Skull	Head, face, and neck	Multiple	Miscellaneous	
Fractures	5	13	1	1	6	.....	1	.....	27
Lacerations	.....	5	.....	.....	.....	4	8	.....	17
Contusions	1	4	2	.....	.....	1	9	.....	17
Sprains	1	5	2	.....	.....	.....	.....	.....	8
Multiple injuries, extreme	.....	.....	.....	.....	.....	.....	4	.....	4
Intracranial injuries	.....	.....	.....	.....	.....	4	.....	.....	4
Abrasions	1	1	.....	.....	.....	.....	.....	.....	2
Crush	.....	1	.....	1	.....	.....	.....	.....	2
Burns	1	.....	.....	.....	.....	.....	1	.....	2
Strains	1	.....	.....	.....	.....	.....	.....	.....	1
Punctured wounds	.....	1	.....	.....	.....	.....	.....	.....	1
Incised wounds	.....	.....	.....	.....	.....	.....	1	.....	1
Hemorrhage into eyeball	.....	.....	.....	.....	.....	.....	.....	1	1
Injury, extent not recorded	.....	.....	1	.....	.....	.....	.....	.....	1
Total	10	30	6	2	6	9	24	1	88

(*e*) *Causes of accidents.*—The immediate causes of the accidents have been classified under the captions of the following table. Note the relatively large number, 16 instances, due to overturning, which fact indicates that the vehicle was traveling at a high rate of speed at the time of the accident.

Cause of accident	Automobile	Motorcycle	Motor vehicle (type not stated)	Total
Collision:				
With an automobile	24	15	1	40
With an immovable object	3	3	0	6
Overturning:				
Skidding	3	4	0	7
Skidding and collision	1	0	0	1
Running off road	2	0	0	2
Breaking of parts	2	0	0	2
Cause not stated	8	0	0	8
Miscellaneous:				
Fall from automobile	3	0	0	3
Stepping from automobile	1	0	0	1
Cranking, backfired	1	0	0	1
Scalded by hot water from radiator	1	0	0	1
Cause of accident not stated	8	1	0	9
Struck in eye by pebble	0	1	0	1
Struck by automobile while a pedestrian	6	0	0	6

(*f*) *Contributory factors.*—Of 88 persons admitted to the sick list, 6 showed evidences of intoxication and 78 gave no evidence of alcohol, while in 4 cases there was no record. Those injured while driving, riding as passengers, etc., are listed below:

	Automobile	Motor cycle	Motor vehicle (type not stated)	Total
Driver	16	18	0	34
Passenger	29	4	0	33
Pedestrian	6	0	0	6
Other (cranking)	1	0	0	1
Not recorded	12	1	1	14

(*g*) *Comparative figures for this force.*—The following table gives the fiscal year 1932 traffic-injury figures for this force, showing the number of admissions, sick days, deaths, and those invalided from the service, with the rate per 100,000 for each group. Figures for the Navy as a whole for 1929 and 1930 are also shown for the purpose of comparison.

An extenuating explanation for this unfavorable comparison consists of the fact that San Diego, Calif., the home port of this force, has a higher traffic death rate than any of the other 86 large cities of this country.

	Admissions	Rate per 100,000	Sick days	Rate per 100,000	Deaths	Rate per 100,000	Invalided from the service	Rate per 100,000
Destroyers, battle force, fiscal year 1932	88	1,494	4,091	69,516	13	221	5	85
Entire Navy (calendar year):								
1929 [1]	1,011	861	32,585	27,758	48	41	33	28
1930 [1]	1,181	1,006	42,512	36,195	58	49	54	46

[1] Annual Reports of the Surgeon General, United States Navy, 1930 and 1931.

SUMMARY

The foregoing discussion of traffic injuries in the personnel of destroyers, battle force, is hereby summarized:

(1) Traffic injuries resulted in:

(*a*) Admissions to the sick list---------- 88
(*b*) Men killed---------- 13
(*c*) Men invalided from the service---------- 5
(*d*) Cases of hospitalization---------- 65
(*e*) Sick days---------- 4,091

(2) Such accidents represent over $200,000 loss to the Government.

(3) The higher ranks and ratings are strikingly less predisposed to traffic accidents.

(4) Chief petty officers proved over 35 times safer drivers than nonrated men.

(5) The majority of such accidents are preventable, as indicated by the negligible number of injuries to commissioned officers.

(6) Youth was a contributory factor.

(7) The traffic death rate of this force is about four times greater than that for the Navy as a whole.

HEALTH OF THE NAVY

The general admission rate, based on returns for diseases, injuries, and poisonings occurring in July, August, and September, 1932, was 604 per 1,000 per annum. The corresponding rate for the first quarter of the year was 465, and for the second quarter, 521. The median rate for the third quarter, as indicated by the records of the preceding five years, is 540. The comparatively high admission rate for July, August, and September was due to the unusual prevalence of common infections of the respiratory type and the appearance of catarrhal fever and cases diagnosed as influenza in epidemic form

in the United States Fleet. The admission rate for disease was 549 per 1,000 per annum and the 5-year median for the corresponding three months was 470. The admission rate for accidental injuries was 54, compared with 65, the median or expected rate for the preceding five years.

Health conditions ashore in the United States were satisfactory for the quarter. Diseases of the respiratory type were less prevalent than has been the experience of the past five years. Only 452 cases of acute catarrhal fever were reported by all shore stations in the United States, 149 of which were notified in July, 182 in August, and 121 in September. Sixty per cent of the total cases occurred among the naval activities in the vicinity of San Diego, Calif. The United States naval training station, San Diego, Calif., reported 175 cases of acute catarrhal fever. The other three naval training stations were remarkably free from this disease.

The outbreak of German measles, which occurred at the United States naval training station, Norfolk, Va., during the second quarter, terminated in July.

One fatal case of typhoid fever was reported by the United States naval hospital, Washington, D. C. The patient was received from the marine barracks on the 16th of July and died from an intestinal hemorrhage 12 days later. His health record indicates that he had received three injections of typhoid prophylaxis 3 years and 10 months prior to infection.

The admission rate, all causes, for forces afloat was 590 per 1,000 per annum. The median rate for the third quarter of the preceding five years is 454. The increase above the expected rate was due to the epidemics of acute catarrhal fever and influenza which appeared on board several ships of the Navy during the quarter. A total of 2,896 cases of acute catarrhal fever and 685 cases of influenza were notified by forces afloat. Ships reporting 25 or more cases are listed in the following table:

	Catarrhal fever				Influenza				Grand total
	July	August	September	Total	July	August	September	Total	
Battle force:									
U. S. S. Colorado	0	249	2	251	0	0	0	0	251
U. S. S. West Virginia	1	9	9	19	219	0	0	219	238
U. S. S. Maryland	171	14	7	192	0	0	0	0	192
U. S. S. New York	159	6	11	176	0	0	0	0	176
U. S. S. Tennessee	125	12	14	151	0	0	0	0	151
U. S. S. Saratoga	17	24	96	137	0	0	0	0	137
U. S. S. Texas	105	11	13	129	1	3	0	4	133
U. S. S. California	93	16	0	109	0	0	0	0	109
U. S. S. Oklahoma	54	48	5	107	0	0	0	0	107
U. S. S. Concord	0	0	0	0	49	30	0	79	79
U. S. S. Nevada	1	48	17	66	0	0	0	0	66
U. S. S. Omaha	4	1	0	5	0	37	0	37	42
U. S. S. Pennsylvania	33	3	1	37	0	0	0	0	37
U. S. S. Melville	1	16	1	18	5	0	11	16	34
U. S. S. Talbot	0	0	0	0	0	10	16	26	26
U. S. S. Cincinnati	3	0	1	4	12	9	0	21	25
Scouting force:									
U. S. S. Arkansas	52	31	14	97	43	24	1	68	165
U. S. S. Lexington	78	71	7	156	0	0	0	0	156
U. S. S. Chicago	18	64	5	87	0	0	0	0	87
U. S. S. Vestal	7	5	0	12	48	0	0	48	60
U. S. S. Pensacola	0	32	27	59	0	0	0	0	59
U. S. S. Whitney	11	22	13	46	1	0	0	1	47
U. S. S. Augusta	14	11	3	28	11	4	0	15	43
U. S. S. Northampton	22	3	7	32	0	1	1	2	34
U. S. S. Manley	0	31	1	32	0	0	0	0	32
U. S. S. Richmond	20	11	0	31	0	0	1	1	32
U. S. S. Bernadou	0	14	14	28	0	0	0	0	28
U. S. S. Yarnall	0	17	8	25	0	0	0	0	25
Asiatic Fleet:									
U. S. S. Rochester	0	21	22	43	0	0	0	0	43
U. S. S. Black Hawk	9	24	2	35	0	0	0	0	35
U. S. S. Asheville	0	25	1	26	0	0	0	0	26
All other ships:									
U. S. S. Medusa	57	1	5	63	0	0	0	0	63
U. S. S. Chaumont	45	8	2	55	0	0	0	0	55
U. S. S. Nitro	0	1	1	2	0	24	10	34	36
U. S. S. Henderson	2	23	9	34	0	0	0	0	34
U. S. S. Holland	1	23	12	26	0	0	0	0	26

One case of scarlet fever was notified by the U. S. S. *Oklahoma* in July and one case by the U. S. S. *New York* in September. The U. S. S. *Monocacy*, at Hankow, China, reported 1 case of typhoid fever, 1 case of paratyphoid fever, and 1 case of diphtheria in September. One fatal case of Asiatic cholera was notified by the U. S. S. *Smith Thompson*, at Chefoo, China. The patient, 21 years of age, was admitted to the sick list seriously ill. Death occurred in the afternoon of the ninth day.

Most of the shore stations outside of the continental limits of the United States reported small outbreaks of acute catarrhal fever during the quarter. The expeditionary forces, China, notified 122 cases; the naval station, Guam, 55 cases; and the expeditionary forces, Nicaragua, 41 cases. The marine detachment, American Legation, Peiping, China, reported 1 case of typhoid fever in July and 1 in August. Both patients had received two courses of typhoid vaccine within two years.

An epidemic of measles among the native population of the island of Guam was reported during the quarter.

TABLE No. 1.—*Summary of morbidity in the United States Navy and Marine Corps for the quarter ended September 30, 1932*

	Forces afloat	Forces ashore	Marine Corps	Entire Navy
Average strength	70,796	38,903	16,668	109,699
All causes:				
Number of admissions	10,438	6,122	3,299	16,560
Annual rate per 1,000	589.75	629.46	791.70	603.83
Disease only:				
Number of admissions	9,776	5,287	2,713	15,063
Annual rate per 1,000	552.35	543.61	651.07	549.25
Communicable diseases, exclusive of venereal disease:				
Number of admissions	4,544	1,485	1,056	6,029
Annual rate per 1,000	256.74	152.69	253.42	219.84
Venereal diseases:				
Number of admissions	2,816	1,044	888	3,860
Annual rate per 1,000	159.11	107.34	213.10	140.75
Injuries:				
Number of admissions	654	826	584	1,480
Annual rate per 1,000	36.95	84.93	140.15	53.97
Poisoning:				
Number of admissions	8	9	2	17
Annual rate per 1,000	0.45	0.93	0.48	0.62

TABLE No. 2.—*Deaths reported, entire Navy, during the quarter ended September 30, 1932*

		Navy			Marine Corps		Nurse Corps	Total
		Officers	Midshipmen	Men	Officers	Men		
Average strength		9,386	1,729	81,394	1,190	15,501	499	109,699
CAUSE—DISEASE								
Primary	Secondary or contributory							
Abscess, brain	None			1				1
Abscess, liver	Ulcer, intestines			1				1
Abscess, multiple	Septicemia	1						1
Abscess, peritonsillar	Pneumonia, broncho			1				1
Do	Septicemia	1						1
Adhesions, intraabdominal	Dilatation, cardiac, acute			1				1
Angina, pectoris	Myocarditis, chronic	1						1
Appendicitis, acute	Peritonitis, general, acute			1				1
Carcinoma, prostate	Metastases, liver and lung	1						1
Carcinoma, cervix uteri	None						1	1
Cholecystitis, acute	do					1		1
Cholera, Asiatic	do			1				1
Cirrhosis, liver	do			1				1
Encephalitis, lethargic	do			1				1
Embolism, cerebral	Arteriosclerosis, general					1		1
Gastritis, acute	Dilatation, stomach, acute			1				1
Gastroenteritis, acute	Dilatation, cardiac, acute					1		1
Hemorrhage, cerebral	None				1			1
Influenza	Encephalitis, acute			1				1
Do	Meningitis, cerebral			1				1
Intestinal obstruction from external causes.	None				1			1
Malaria	do				1			1
Meningitis, cerebrospinal, acute.	Septicemia			1				1
Myocarditis, chronic	Dilatation, cardiac, acute			1				1
Nephritis, chronic	Uremia	1						1
Obstruction, intestinal, from internal causes.	Peritonitis, general, acute					1		1
Osteomyelitis, right scapula	Septicemia			1				1
Pneumonia, broncho	None				1			1
Sarcoma, fibula	do					1		1
Sinusitis, maxillary	Dilatation, cardiac, acute			1				1

TABLE No. 2.—*Deaths reported, entire Navy, during the quarter ended September 30, 1932*—Continued.

		Navy			Marine Corps		Nurse Corps	Total
		Officers	Midshipmen	Men	Officers	Men		
Average strength		9,386	1,729	81,394	1,190	15,501	499	109,699
CAUSE—DISEASE—Continued								
Primary	Secondary or contributory							
Thrombosis, coronary artery.	Myocarditis, chronic			1				1
Tonsillitis, acute	Septicemia					1		1
Tuberculosis, chronic, pulmonary.	None					1		1
Do	Pneumopyothorax					1		1
Tuberculosis, acute, general miliary.	None					1		1
Tuberculosis, intestines	do			1				1
Tumor, malignant, mixed, carcinoma, brain.	do	1						1
Typhoid fever	Hemorrhage, intestinal					1		1
Valvular heart disease, aortic insufficiency.	Myocarditis, chronic					1		1
Total for diseases		6		17	4	11	1	39
CAUSE—INJURIES AND POISONING								
Asphyxiation, illuminating gas.	None			1		1		2
Burn, face, chest, and arm	do			1				1
Burn, multiple	do			1				1
Crush, chest	do			1				1
Crush, head	do			1				1
Drowning	do	1		3	1	1		6
Fracture, compound, skull	do			5		1		6
Do	Pneumonia, broncho			1				1
Do	Hemorrhage, intracranial			2				2
Do	Hemorrhage, femoral artery.			1				1
Do	Meningitis, cerebral			1				1
Do	Intraspinal injury			1				1
Injuries, multiple, extreme	None	1	1	10		1		13
Do	Septicemia					1		1
Do	Hemorrhage, traumatic, lung.			1				1
Intracranial injury	None			1				1
Rupture, traumatic, lung	do			1				1
Rupture, traumatic, small intestine.	Hemorrhage, intraabdominal.			1				1
Wound, gunshot, brain	None			2				2
Wound, lacerated, aorta	Hemorrhage, traumatic, lung, aorta.					1		1
Wound, punctured, lung	None			1				1
Poisoning, acute, amyl alcohol.	do			1				1
Poisoning, acute, potassium cyanide.	do	1						1
Total for injuries and poisoning.		3	1	37	1	6		48
Grand total		9	1	54	5	17	1	87
Annual death rate per 1,000:								
All causes		3.84	2.31	2.65	16.81	4.39	8.02	3.17
Disease only		2.56		.84	13.45	2.84	8.02	1.42
Drowning		.43		.15	3.36	.26		.22
Injuries		.43	2.31	1.62		1.29		1.46
Poisoning		.43		.05				.07

ADMISSIONS FOR INJURIES AND POISONING, THIRD QUARTER, 1932

The following table, indicating the frequency of occurrence of accidental injuries and poisonings in the Navy during the third quarter, 1932, is based upon all Form F cards covering admissions in those months which have reached the bureau:

	Admissions, July, August, and September, 1932	Admission rate per 100,000, per annum	Admission rate per 100,000, year, 1931
INJURIES			
Connected with work or drill	348	1,269	2,433
Occurring within command but not associated with work	531	1,936	1,649
Incurred on leave or liberty or while absent without leave	601	2,191	1,603
All injuries	1,480	5,396	5,685
POISONING			
Industrial poisoning	2	7	17
Occurring within command but not connected with work	7	26	35
Associated with leave, liberty, or absence without leave	8	29	22
Poisoning, all forms	17	62	74
Total injuries and poisoning	1,497	5,458	5,759

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	
	July, August, and September, 1932	Year, 1931	July, August, and September, 1932	Year, 1931	July, August, and September, 1932	Year, 1931
Per cent of all injuries	23.5	42.8	35.9	29.0	40.6	28.2
Per cent of poisonings	11.8	23.4	41.2	46.8	47.0	29.8
Per cent of total admissions, injury and poisoning titles	23.4	42.5	35.9	29.3	40.7	28.2

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 1932 worthy of notice from the standpoint of accident prevention.

STATISTICS RELATIVE TO MENTAL AND PHYSICAL QUALIFICATIONS OF RECRUITS

The following tables were constructed with figures taken from monthly reports submitted by naval training stations:

Cumulative data

	Number	Per cent of recruits received	Per cent of recruits reviewed
JAN. 1 TO DEC. 31, 1931			
All naval training stations:			
Recruits received during the period	7,071		
Recruits appearing before board of medical survey	227	3.21	
Recruits recommended for discharge from the service	122	1.73	53.74
JULY, AUGUST, AND SEPTEMBER, 1932			
United States naval training station, Hampton Roads, Va.:			
Recruits received during the period	305		
Recruits appearing before board of medical survey	6	1.97	
Recruits recommended for discharge from the service	6	1.97	100.00
United States naval training station, Great Lakes, Ill.:			
Recruits received during the period	453		
Recruits appearing before board of medical survey	7	1.55	
Recruits recommended for discharge from the service	7	1.55	100.00
United States naval training station, San Diego, Calif.:			
Recruits received during the period	535		
Recruits appearing before board of medical survey	2	.37	
Recruits recommended for discharge from the service	2	.37	100.00
United States naval training station, Newport, R. I.:			
Recruits received during the period	308		
Recruits appearing before board of medical survey	13	4.22	
Recruits recommended for discharge from the service	1	.32	7.69
Recruits held over pending further observation	12	3.90	92.31

The following table was prepared from reports of medical surveys in which disabilities or diseases causing the surveys were noted as existing prior to enlistment. The time which elapsed from date of enlistment to date of medical survey is noted in each case. With certain diseases survey followed enlistment so rapidly that it would seem that many might have been eliminated in the recruiting office. The difficulty in establishing a diagnosis in nervous and mental cases is demonstrated by the time interval in the table. An exception in this group is epilepsy which may or may not diagnose itself promptly. Certain groups, of course, present difficulties in diagnosis at the time of enlistment due to lack of equipment.

Cause of survey	Number of surveys	Number of days between enlistment and survey
Ankylosis, elbow	1	10
Astigmatism	1	7
Caries, teeth	1	5
Color blindness	1	11
Constitutional psychopathic inferiority without psychosis	1	103
Do	1	138
Constitutional psychopathic state, inadequate personality	1	197
Do	1	60
Effort syndrome	1	133
Goiter	1	6
Intracranial injury	1	216
Malocclusion teeth	1	8
Otitis, media, chronic	1	3
Do	1	3
Do	1	58
Psychoneurosis, hysteria	1	45
Psychosis, manic depressive	1	46

Cause of survey	Number of surveys	Number of days between enlistment and survey
Syphilis	1	16
Do	1	16
Do	1	26
Do	1	65
Ulcer, duodenum	1	171
Urethritis, chronic, nonvenereal	1	20
Valvular heart disease, mitral stenosis	1	9

VOL. XXXI JULY 1933 No. 3

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
LIEUTENANT COMMANDER JOHN HARPER
MEDICAL CORPS, U.S. NAVY, IN CHARGE

Compiled and published under the authority of Naval Appropriation Act for 1933, Approved June 30, 1933

UNITED STATES
GOVERNMENT PRINTING OFFICE
WASHINGTON : 1933

For sale by the Superintendent of Documents, Washington, D.C. - - - - - - See page II for price

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.

SUBSCRIPTION PRICE OF THE BULLETIN

Subscription should be sent to Superintendent of Documents, Government Printing Office, Washington, D.C.

Yearly subscription, beginning July 1, $1; for foreign subscriptions add 35 cents for postage.

Single numbers, domestic, 25 cents; foreign, 35 cents, which includes foreign postage.

Exchange of publications will be extended to medical and scientific organizations, societies, laboratories, and journals. Communications on this subject should be addressed to the Surgeon General, United States Navy, Washington, D.C.

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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 indorse 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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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 2 months prior to the date of the issue for which they are intended.

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U.S. NAVAL MEDICAL BULLETIN

VOL. XXXI JULY 1933 No. 3

SPECIAL ARTICLES

SEVERE HEAD INJURIES

By LUCIUS W. JOHNSON, captain, Medical Corps, United States Navy, and THOMAS G. HAYS, lieutenant (junior grade), Medical Corps, United States Navy

Treatment of head injuries has advanced greatly during the last few years but has, so far, failed to reach a satisfactory standard, as evidenced by the different methods employed by our leading authorities.

There are those who believe that conservative treatment with intravenous administration of hypertonic fluids and routine spinal punctures gives the best results. On the other hand, there are those who condemn the use of these methods, labeling them not only as useless but as actually harmful and dangerous. They recommend watchful inaction with operative treatment if there are unfavorable developments.

With these different opinions in mind, we have studied a series of about 130 cases of head injuries that we have treated in the San Diego Naval Hospital within a limited time, 50 of which were serious enough to justify minute analysis. We have given careful attention to the claims of each school of thought and have tried to draw conclusions as to the efficacy of the different methods employed. We regard the difference of opinion as beneficial because it stimulates close observation and thoughtful study of the problems which surround head injuries.

Vehicular hazards have replaced drowning as the principal cause of death in the Navy. In 1931 there were, among Navy personnel, 1,216 admissions with 44,297 sick days, 81 deaths and 69 invalided from the service because of injuries by motor vehicles. From the table of injuries for that year one can select at least 164 admissions and 42 deaths from severe craniocerebral injuries. At this hospital, nearly all the deaths caused by vehicular accidents were due to the craniocerebral injuries and most of the severe head injuries were due to vehicular hazards. As long as a second-hand car can be bought for as low as $10 down, this close association of motor vehicle accidents and head injuries will continue to be a matter of grave concern to the Navy.

In analyzing these cases we have tabulated the cause of the injury, type of head injury, degree and duration of shock, complicating injuries, discharge of spinal fluid from ears or nose, degree and duration of unconsciousness, spinal fluid pressure and presence of blood, blood pressure, temperature, pulse rate, the treatment given, intravenous medication and its effect, lumbar punctures and their effect, eye ground changes, localizing signs, operations performed, X-ray findings, outcome and disposition of the case, length of time in hospital, cause of death, and autopsy findings in fatal cases.

Study of the cause of injury showed motor vehicles predominating. Twenty-three were due to automobiles and six to motorcycles. Falls were responsible for 11, assaults for 4, fighting for 2, and boxing for 1. Basket ball caused 1 and an explosion 1. One was struck by a mooring line.

Shock was a prominent symptom in one third of the cases. The usual evidences of shock are subnormal temperature, diastolic pressure of 60 or below, rapid pulse above 120, rapid and shallow respiration. The patient may or may not be unconscious. The duration of shock varies greatly. It may last for only a few moments or it may continue and increase in gravity until death occurs. Certain things should be done during shock and there are other things that must not be done until this stage is past.

Things that should be done to combat shock are to cover the patient with warm, dry clothing; provide external heat, especially to the extremities; give hot drinks if the patient is able to take them; lower the head; give proctoclysis or retention enema of fluid at a temperature of 110° F.; perhaps intravenous administration of 50 percent glucose. External heat and rest are essential. The amount of fluid given during shock should be recorded and given consideration when restriction of fluid intake becomes necessary in a later stage.

Various writers recommend the use of atropine, pituitrin, strychnine, and other drugs to produce peripheral vasoconstriction but warn against the use of cardiac stimulants such as caffeine or adrenalin. Cutting (12) advises the use of morphine as "perhaps the most important of all—the hypodermic administration of morphine in relatively large doses, usually a half grain at a time." Sharpe also speaks highly of the value of morphine during shock, but this is condemned by others, notably Dandy, who contends that morphine should not be given for the same reason that one should not give it to relieve the pain in a case of suspected appendicitis; that it may mask the development of important signs and symptoms. It is interesting to note that those who oppose lumbar puncture and the use of intravenous hypertonic solutions are also against the use

of morphine. Several writers who enthusiastically endorse lumbar puncture and hypertonic glucose recommend the use of morphine in the stage of shock and state that the objections to its use disappeared with the advent of present methods of treatment. Transfusion is recommended in cases with considerable loss of blood, and Fay states that intravenous 50 percent glucose is an important means of combating shock and preventing the development of cerebral edema.

We have repeatedly observed remarkable improvement in shocked patients after intravenous administration of 50 percent glucose solution. In several cases with diastolic pressure practically at zero it has increased rapidly and risen to normal after this treatment. The benefits from its use are more quickly and definitely shown at this stage than at any other time. The only patient who failed to show any response to this treatment died without recovering consciousness. Autopsy revealed extensive extradural, subdural, and cortical hemorrhages.

Things not to be done during shock are equally important to remember. The patient should not be transported while in this condition if it can be avoided, but it is often necessary to remove him to a place where better facilities for treatment can be secured. No elaborate examinations, which may fatigue or expose the patient, should be made. No surgery should be done, except to control hemorrhage from accessible vessels. Dehydration by oral or rectal administration of concentrated solution of magnesium sulphate is absolutely contraindicated. Lumbar puncture and X-ray examination should be deferred until the patient recovers from shock.

Discharge of cerebrospinal fluid or blood from the ears or nose was noted in 9 of our cases and 5 of them died. Those who survived ran a mild course, and we are convinced, from observation of these and other cases, that those who decompress themselves in this way are fortunate in that they escape the evil effects of increased intracranial pressure if their injuries are not fatal. Nothing should be done to stop or interfere with the discharge of this fluid.

Unconsciousness was present in 42 cases and 8 died without regaining consciousness. The duration of unconsciousness in those who lived varied from a few minutes to 6 days. Of those who remained unconscious for 2 days or more, 2 had a convalescent period shorter than the average and in 3 it was slightly longer than the average. Their combined average stay was 1 day longer than the general average for all cases. So it does not appear that prolonged unconsciousness gives a bad prognosis.

Lumbar puncture as a diagnostic and therapeutic measure is surrounded by a cloud of controversy. Its proponents recommend it as

a routine measure, while those opposed to it consider it as absolutely contraindicated and liable to do more harm than good.

Fay (1) states that "lumbar puncture with careful manometric pressure readings must be done and irrespective of the claims made by those opposed to lumbar puncture, an intelligent management of the case is impossible without knowledge of the pressure mechanism and the presence of bloody spinal fluid." Ochsner (2) maintains that "of greatest importance, from a diagnostic and prognostic point of view, is the determination of the cerebrospinal fluid pressure, as well as the character of the fluid." Sharpe (13) does a diagnostic lumbar puncture routinely and states that repeated lumbar puncture and spinal drainage have been of the greatest value in his series of more than 1,500 adults with acute brain injury. Cutting (12) believes that "the therapeutic removal of cerebrospinal fluid is a procedure of the utmost value in the more severe types of increased tension which progress unfavorably in spite of attempts at dehydration." He quotes the teaching of Cushing, that the presence of blood or a xanthoproteic reaction in the cerebrospinal fluid is an especial indication for the performance of repeated spinal punctures.

Thus it appears that many writers, including those who report the largest number of patients treated, do lumbar puncture in all cases of craniocerebral injury. Some do it only in the severe cases or when dehydration fails, and a few are definitely opposed to it. Dandy is again the leader of the opposition. He contends (6) that "in the presence of intracranial pressure from whatever source, lumbar punctures are always dangerous. * * * The reason for this danger is that the brain is traumatized as its mass is suddenly shifted in the readjustment necessary to equalize the changed intracranial pressure. * * * One of the principal claims made in the use of repeated lumbar puncture is that blood is removed from the subarachnoid space. The amount, however, is but a tiny fraction of the great hemorrhage which may exist and which, having clotted, cannot be removed by puncture. * * * Many enthusiasts advise doing a lumbar puncture not as a treatment but to ascertain by manometric reading the exact pressure of the cerebrospinal fluid. That information can be far better and at the same time more safely obtained by carefully studying the patient's signs and symptoms. * * * Spinal punctures * * * are contraindicated in efforts to reduce the intracranial pressure. Nor do spinal punctures yield any information of value in diagnosis."

To follow this teaching literally would deprive us of the use of lumbar puncture in the treatment of cerebrospinal fever as well as in brain injuries. In each condition, we have seen severe headache relieved almost immediately by lumbar puncture and extremely rest-

less patients resting quietly in comfort following the treatment. If lumbar puncture did no more than allay the symptoms, we should consider its use justified.

Lumbar puncture has been done in about 25 percent of the cases here reported and in all of our later cases, both as a diagnostic and a therapeutic measure. We have observed no untoward effects from its use and, in several cases, have observed patients who were extremely restless and showing signs of increased intracranial pressure respond nicely to its use and drop off into a quiet sleep. Its effect is, of course, temporary, but in many cases a single spinal drainage is sufficient to carry the patient past the period of danger. Others require repeated punctures, every 24 hours or oftener, to control the symptoms.

The increased intracranial pressure interferes with the normal absorption of the cerebrospinal fluid, impedes the circulation and reduces the amount of oxygen available. It will eventually produce death unless relieved by natural or artificial means. Assuming always that there is no evidence of severe intracranial hemorrhage, this pressure can be safely relieved for a varying length of time by spinal drainage. As a general rule, one removes enough fluid to reduce the excess of pressure above the normal (10 millimeters of mercury or 135 of water) by one half. This method has been used in thousands of cases with no demonstrable damage and much apparent benefit.

Bloody spinal fluid has a special significance since it indicates hemorrhage into the subarachnoid space from laceration of the brain or rupture of a vessel. Most authorities recommend that, when the spinal fluid is found to be bloody, all the fluid obtainable in the recumbent position be drained with the hope of removing as much of the blood as possible. The debris resulting from disintegration of the blood is believed to interfere with reabsorption of the fluid. Dandy believes drainage in such cases to be useless and dangerous, while Cushing recommends repeated spinal punctures when the spinal fluid is found to be bloody.

Nine of our patients in whom spinal drainage was done had bloody spinal fluid. Five of these died and the autopsies showed subarachnoid hemorrhage in four. The fifth died from meningitis. Four patients recovered, but one remained blind in one eye and deaf in one ear, probably due to basal hemorrhage.

When we pause to consider the physiology of the cerebrospinal fluid, we see that it is formed in the lateral ventricles as a secretion through the cuboidal cells of the choroid plexus. It then passes through the foramen of Monro into the third ventricle and from the third ventricle into the fourth ventricle through the acqueduct

of Sylvius. From the fourth ventricle it passes through the foramina of Luschka and Magendie into the subarachnoid space and the cisternae whence it is absorbed into the blood stream. The method of reabsorption is not satisfactorily settled, but there is probably a constant slow current of the fluid from the place of secretion toward the place of absorption. Increased amount of the fluid may arise either from increased secretion or interference with its absorption.

We know that the hypertonic solution given intravenously withdraws fluid from the body tissues and builds up the blood volume. The opponents of the intravenous administration of hypertonic solution method state that there is a secondary rise of spinal fluid pressure above the original pressure about 2 or 3 hours following the injection. This is still debatable but, assuming that it is true, we can explain it on the basis of increased volume of blood flow through the choroid plexus causing an increased volume of cerebrospinal fluid to be formed. If this is true, a lumbar puncture with removal of the excess cerebrospinal fluid is an important adjunct to the administration of hypertonic solutions and is probably most beneficial about 3 or 4 hours after the intravenous treatment.

One must realize that in cases having a blockage some place between the lateral ventricles and the subarachnoid space, a lumbar puncture is useless and subtemporal decompression is indicated. But in the vast majority of cases, lumbar puncture is sufficient and we advocate its use in conjunction with intravenous administration of 50 percent glucose.

Drainage of the cisterna magna has been recommended for cases in which there is increasing intracranial pressure with no increase in the spinal fluid pressure. We used this in one case when the increased pressure developed several days after injury. Sixteen cubic centimeters of clear fluid were removed and this arrested the progress of the symptoms for about 8 hours. Bilateral subtemporal decompression was done 18 hours later and the patient recovered. One of us (L. W. J.) had done cisterna puncture in 2 earlier cases of craniocerebral injury with excellent results in 1 case and no apparent effect in the other. It would appear that the usefulness of this procedure is limited to the very small group of cases in which there is a block below the level of the cisterna magna. But it is quite possible that further experience and study may develop a more important role for this safe and simple procedure.

Blood pressure observations are most valuable during the stage of shock. They indicate the degree of shock, the urgency of shock treatment and the success or failure of the shock treatment. If the blood pressure is taken every 15 or 30 minutes it makes an excellent barometer and a guide to treatment. Fay teaches that if the pulse

pressure tends to rise or approach the pulse rate, it is an indication for further relief of intracranial pressure by lumbar puncture or an enema of magnesium sulphate solution.

The pulse rate gives us much valuable information. During the period of shock it is likely to be very rapid and feeble. Some writers consider that the period of shock is over when the pulse rate falls to 120 or less. Progressive slowing of the rate to 60 or less with a full, bounding pulse suggests the development of cerebral edema. In the late stage of edema, when the regulating function of the centers in the medulla becomes impaired, the pulse usually becomes very rapid, thready, and irregular. The prognosis is then very poor. Several of our patients had a pulse rate as low as 52 and one as low as 40, but recovered. Dandy teaches that rapid changes in the rate and force of the pulse are a most valuable warning that a break of compensation is impending.

The temperature is useful as an index of the condition of the patient during the stage of shock and again later, when the advent of increasing intracranial pressure is awaited. Sharpe believes that the temperature is no indication of the degree of intracranial pressure, while Dandy considers it frequently the most sensitive indicator of increasing intracranial pressure. It appears to be a general opinion that below 102° F. there is relative safety and above that, great danger. The only one of our patients who had a temperature above 102° F. died. It is not rare to see, in the terminal stage of cerebral edema, temperatures of 105° F. or even much higher.

Intravenous injection of hypertonic solutions for control of intracranial pressure is the most important advance made in this field during recent years and it is generally regarded as the foundation of modern therapy of intracranial injuries. In 1919 Weed and McKibben (11) published reports of their experimental work showing that the volume of the intracranial contents could be decreased by intravenous injection of hypertonic salt solution and increased by using hypotonic solutions. They also showed that these changes in volume could be accurately measured by the pressure of the cerebrospinal fluid as shown by lumbar puncture and the use of a manometer.

These articles aroused great interest and stimulated others to study these phenomena. The findings of Weed and McKibben were verified by other workers and the effects of many chemicals were tried. It was later shown that the use of hypertonic salt solution was followed by a secondary wave of edema, probably due to storage of sodium chloride in the brain tissues. This secondary edema was found not to occur after the use of glucose solution and the effects of glucose were proven to be much more prolonged than those produced by salt. For these reasons the use of hypertonic salt solution

for chemical decompression has been supplanted by the use of hypertonic glucose, usually in 50 percent solution. Excellent discussions of this subject can be found in the articles by Swift and Flothow (8), by Dandy, Fay, and Cutting.

Most writers, and especially those who report the largest groups of cases treated, are agreed on the great value of hypertonic solutions and on 50 percent glucose as the ideal dehydrating agent. But, once more, there are dissenters. Dandy (6) insists that "intravenous injections of hypertonic glucose or sodium chloride are contraindicated in efforts to reduce the intracranial pressure. They may cause serious harm." He states that subtemporal decompression is the treatment of choice when intracranial pressure demands relief.

Miller and Hurwitz (7) injected a man with 200 cubic centimeters of 50 percent dextrose and observed a drop of 3 millimeters of mercury in the spinal fluid pressure which lasted 1 hour. It was followed by a gradual rise of pressure which, 1½ hours later, was 6 millimeters above the initial level of 8 millimeters. The results of this single test on a man were similar to those in several tests on cats.

These widely divergent opinions and observations on an important and much-used therapeutic measure are most striking. We have given careful consideration to them in treating our cases and have reached the conclusion that intravenous 50 percent glucose is a valuable agent for controlling intracranial pressure. We have used it in most of our cases and have seen no ill effects from it. We attach great significance to the fact that those who see the greatest number of head injuries and publish studies based on many hundreds of cases are the most enthusiastic in its praise. On the other hand, we have been several times disappointed in its action when the effects have been very slight and of short duration. There can be no doubt that it is valuable in the treatment of shock and when so used it appears to lessen or prevent the cerebral edema of the later stages. It is also most useful in treating the headaches and other symptoms during the late convalescent period.

Magnesium sulphate solution, given by mouth or by enema as a means of dehydration, receives less attention than it formerly did. Its purpose is to reduce the total of body fluids thus reducing indirectly the bulk of the cerebral contents and the intracranial pressure. To give it by mouth may be impossible because of unconsciousness or vomiting and its rectal use is frequently quite troublesome. The resulting fluid bowel discharges, frequently involuntary, are annoying and may require considerable handling of the patient at a time when complete rest and relaxation are most important. This treatment should never be used during shock. Compared with the effect

of hypertonic glucose solution on intracranial pressure, we have found the action of magnesium sulphate to be slow, unreliable, and of short duration.

Restriction of fluid intake is an important point that has only recently received the attention that it deserves. Both the intake and output of fluid should be carefully measured and charted from the beginning. The fluid balance should be computed for each 24 hours and carefully considered in prescribing the diet. Fay (1) recommends that the fluids be restricted to 20 ounces per day except when there is the demonstrated presence of blood in the spinal fluid, in which cases he increases the allowed fluids by the amount withdrawn during the daily spinal fluid drainage or to as much as 30 ounces. He usually employs a solid, dry diet after the first 24 hours and finds that it is well tolerated, even when liquid and soft foods are vomited. Thirst is controlled by giving the fluids in small quantities at regular intervals.

Eyeground examinations were made in about one-third of our cases and we found them of little value except as confirmatory evidence. Papilledema develops rather slowly and is seldom useful during the first 48 hours after injury. In the convalescent period, when occasional headaches and drowsiness occur, we have found that watching the changes in the eyegrounds helped us to determine whether or not active intervention was necessary. The disappearance of papilledema after decompression operations is often very rapid and a normal appearance may return within 24 hours.

Operation for relief of intracranial pressure is not often advisable. No operation should be done during the stage of shock except to stop hemorrhage from accessible vessels. It is seldom necessary to operate during the first 48 hours unless there are definite signs of extradural hemorrhage. In the past 10 years conservatism has grown to be the rule, as a result of better understanding of the indications for operation and what can be accomplished by it.

Wortis and Kennedy (3) recommend right subtemporal decompression for comatose patients with marked papilledema who do not respond to other decompression methods within three hours. Rodman (4) states that "I must confess to being somewhat in doubt as to the wisdom of ever doing a subtemporal decompression. As a general rule it will do no good in the cases which fail to respond to the nonoperative plan * * *, and yet there is the occasional case, progressing slowly toward the terminal stage of compression in spite of the most active nonoperative plan, that will, I believe, benefit by decompression." Sharpe and Sharpe (5) state that the patient with brain injuries should not be allowed to reach the dangerous stage of medullary compression due to high intracranial pressure.

It should be anticipated by the accurate diagnostic methods now known and if a marked increase of the intracranial pressure is present and the expectant palliative measures fail then an early operative relief of it should be advised.

Fay (9) states that he has entirely abandoned decompression and operates only when all other means have failed or definite focal signs indicate a subdural or epidural clot. He mentions two indications for operation, (*a*) compound or comminuted fractures with extension into cerebral substance and (*b*) depressed fractures sufficiently large to cause pressure and encroachment on important cerebral areas. Seven patients died out of eight on whom he operated early. Delay of operation beyond the tenth day was not associated with increased mortality. "When all other measures toward control of cerebral edema and pressure have failed, operation has also failed to preserve these cases." Dandy recommends surgical decompression if there are evidences of increasing intracranial pressure after the first 5 or 6 hours following the injury.

We have followed a conservative course and have done decompression with ventricular drainage in only three of this group of cases. The first, complicated by abscess of the frontal lobe, was temporarily benefited but the eventual death was not averted. The other two were unquestionably saved by operation and are now completely recovered. Another case, previously reported by one of us (10), a young girl, may be added to this group of patients who were undoubtedly saved by operation. Therefore we are firmly of the opinion that while conservatism is the best general policy, there is an occasional case in which operation will avert an otherwise certain death. It is important to be always on the watch for such cases.

Most authorities urge that operation for depressed fractures be done soon after recovery from shock. We have seen no ill effects from a wait of several days and believe that the patients stand operation much better if it is delayed.

The differences of opinion, whether or not to operate and when to operate are by no means of recent origin. Hippocrates became involved in a controversy on the same subject and issued the dictum that vomiting, aural hemorrhage, unconsciousness and convulsions following head injuries were indications for operation. In the early part of the eighteenth century there was a bitter dispute, in which the best-known men of the time took opposing sides, on whether or not to trephine as a routine measure in head injuries.

A brief résumé of our operative cases follows:

(1) A man of 30, while fighting a forest fire, was struck by a falling boulder and knocked several hundred feet down the mountain side, sustaining lacerations

and several fractures about the fronto-parietal region. He recovered consciousness about 48 hours later and, after a few days, was apparently well. On the twenty-second day he noticed double vision. On the thirty-fifth day he complained of headache and was conscious of strange odors. He vomited and slept all day. His pulse was about 60. He then passed through several cycles consisting of 2 or 3 days of drowsiness with headache, followed by a few days of complete relief. These symptoms were at first relieved by hypertonic fluids, catharsis and restriction of fluids but these measures later failed to help. Three times we prepared to operate, suspecting a frontal-lobe abscess, and three times postponed operation because all symptoms disappeared. On the ninety-fifth day we did a right subtemporal decompression. Intracranial pressure was found to be increased and 15 cubic centimeters of fluid were removed from the ventricle. He was greatly improved for 8 days, then gradually became comatose. The ventricle was again drained by a needle passed through the scalp and the trephine opening. This gave relief for about 12 hours but then he grew worse and died 47 days after the operation. Autopsy revealed an abscess occupying most of the left frontal lobe.

(2) A man, aged 21, injured in an automobile accident. He had only a brief period of unconsciousness. Eighteen hours later he began to complain of headache and soon became drowsy. Examination showed evidence of a fracture of the floor of the the anterior fossa. He responded well to treatment by hypertonic glucose. About the twenty-sixth day he showed signs of increased intracranial pressure which could not be controlled by the usual means. He gradually developed marked papilledema, profound coma, slow pulse, and stertorous breathing. A transverse incision was made across the vertex from ear to ear; the anterior flap was peeled forward and a trephine opening made over each frontal lobe. The dura was incised and the ventricle drained on each side. Consciousness returned almost immediately and by the following morning all symptoms were relieved. He recovered completely and returned to duty. Strangely enough, 3 days later he was knocked down, his head struck the concrete deck and he was returned to the hospital with a new fracture in the temporo-parietal region. Recovery was again complete.

(3) A young man fell 22 feet into a drydock. He was admitted unconscious and did not recover consciousness for 48 hours. Symptoms of increased intracranial pressure were controlled by nonoperative means. He was not fully oriented until several days later. On the tenth day he became increasingly irritable, irrational, restless, and then comatose. There was a high degree of papilledema and the pulse gradually fell to 45. Intravenous hypertonic glucose produced no improvement. Spinal puncture showed no increased pressure so a cisternal puncture was done. Sixteen cubic centimeters of fluid under slightly increased pressure were removed and this produced some improvement for a few hours. The symptoms then progressed in spite of all treatment, so on the fifteenth day a bilateral subtemporal decompression was done. Both sides showed fluid under pressure beneath the dura. Both ventricles were drained but the amount of ventricular fluid and the pressure were not apparently increased. After the operation there was improvement in his condition and he eventually returned to duty.

We have been constantly on the watch for evidences of extradural hemorrhage but only one patient showed signs that might have justified operation for control of a middle meningeal hemorrhage. He had such severe complicating injuries that death seemed inevitable, no matter what was done.

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Fracture of the skull is a relatively unimportant factor and, unless it is compound or depressed, makes little difference in the treatment or prognosis. In patients who survive, we are usually dependent on the X-ray to demonstrate the presence and location of the fracture and this is by no means infallible, especially in basal fractures. We have had several cases with discharge of cerebrospinal fluid or late ecchymoses which indicated a fracture but the X-ray did not show it. Twenty-three of our fifty cases had fractures which showed in the X-ray films or were found at autopsy. Two of the eight fatal cases had extensive laceration of the brain with hemorrhage but no fracture of the skull.

The average stay in hospital of our cases that survived was 65 days. In comparing this with the average stay in a civilian hospital it must be remembered that the civilian can leave the hospital after the acute stage is past and can complete his convalescence at home. When the Navy man is returned to duty from the hospital he must be fully recovered and fit to perform all of his duties at sea. So a longer stay is to be expected for patients in a Navy hospital.

Eight of our patients died and six of these had localizing signs. Three who had localizing signs lived and two of these were invalided from the service, one because of blindness in one eye and deafness in one ear, one because of dizzy spells with blurred vision. Seven patients showed pupillary changes, two had positive Babinski and Oppenheim reactions. Those with dilated and fixed pupils uniformly died.

Of the 8 patients who died, death in 2 cases was due to intracranial injury with pulmonary lacerations and pulmonary hemorrhage; in 3 cases to intracranial injury with cerebral laceration; in 1 case to intracranial injury with massive collapse of a lung; in 1 case to intracranial injury with compound comminuted fracture of tibia and fibula; in 1 case to intracranial injury with meningitis and pericardial hemorrhage.

Summary

A group of about 130 cases of acute intracranial injury was observed and 50 cases were carefully analyzed.

Fifty percent glucose solution, given intravenously was found valuable in combating shock and treating intracranial pressure.

Lumbar puncture was found useful both in treatment and diagnosis. No ill effects from it were observed.

Operation (subtemporal decompression) was done in 3 cases and 2 of the patients recovered. It is believed that both would have died without operation.

Eight patients died: three were invalided from the service.

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CARBOXIDE GAS: A NEW INSECTICIDAL FUMIGANT FOR BEDBUGS AND COCKROACHES

By E. W. Brown, captain, Medical Corps, United States Navy

INTRODUCTION

The usual methods of extermination of insect pests on naval vessels or at shore stations fall under three groups, i.e. (1) the use of contact insecticides such as mineral oil emulsions or solutions of pyrethrin in a volatile solvent applied as sprays, (2) the employing of stomach insecticides such as insect powders containing arsenicals or fluorine compounds, and (3) flaming, usually limited to bunk frames and bunk springs. Occasionally a vessel may be assigned to a fumigation station of the Public Health Service for fumigation with hydrocyanic-acid gas, but ordinarily this involves too serious an interference with the operating schedule to be feasible.

The procedures under (1) and (2) are unsatisfactory in that the insecticide does not obtain sufficient access to the places affording harborage to the insects. They are, therefore, of temporary value only, as the insects hidden away are not reached. On the other hand, a gas tends to be the ideal insecticide in that it expands to occupy all available space and penetrates into every crack and

crevice where the insects may be concealed. For the destruction of bedbugs, cockroaches, weevils in wheat, beetles in flour, etc., a fumigant is the proper insecticide in naval practice provided it fulfills the following requirements: lethal effectiveness for the insects; freedom from fire hazard; low toxicity for man; no damaging effect on fabrics, furniture, food products, or metals; and economical cost.

Hydrocyanic-acid gas is a highly toxic fumigant, the lethal dose for insects being also fatal to man. Extreme precautions are imperative in its fumigant use and subsequent removal from the spaces of the ship. It is, therefore, unsafe for use except in expert hands and not adapted for routine application by naval personnel aboard naval vessels. Carbon bisulphide forms an inflammable and explosive mixture with air in the concentration necessary for insect destruction. Furthermore, it is a liquid at ordinary temperature and a complication is introduced in securing sufficiently rapid vaporization of the compound. Sulphur dioxide is also excluded on account of its destructive action on linen or cotton goods, furnishings, paint, and metals.

An insecticidal fumigant which does not possess the undesirable characteristics of the compounds just discussed and is, therefore, promising for naval use, has been recently developed by the Carbide & Carbon Chemicals Corporation, New York, N.Y., and is designated as ethylene oxide. Ethylene oxide is a colorless mobile fluid which boils at 50.9°F. and is, therefore, a gas at ordinary temperatures. It has a specific gravity of 0.887 at 7°/4°C., is soluble in water and organic solvents in all proportions, and has a faint but distinctive etherlike odor. The vapor is approximately 1.7 times as heavy as air and exhibits remarkable penetration into dense materials such as wheat flour. It is marketed in steel containers under pressure, resembling the ordinary oxygen cylinder.

Cotton and Roark (1) were the first to study the insecticidal value of ethylene oxide. They reported that 1 pound per 1,000 cubic feet in a fumigation vault in 20 hours completely killed the clothes moth, carpet beetle, rice weevil, Indian meal moth, saw-toothed grain beetle, red-legged ham beetle, and the flour beetle. The insects were contained in cotton stoppered glass vials buried in overstuffed furniture, sealed in cartons of cereal and buried in jars of rice. Excellent power of penetration was demonstrated, the temperature ranging from 60° to 75° F.

Hoyt (2) found shortly afterward that adults and larvae of the confused flour beetle, larvae of the Indian meal moth and larvae of the clothes moth were entirely destroyed by a concentration of 2 pounds per 1,000 cubic feet with 24 hours' exposure in a fumigation vault at a temperature of 75° to 80° F., the insects being so placed as to demand relatively deep penetration.

Cotton and Roark (3) in additional tests reported that the larvae of the clothes moth, black carpet beetle and of the furniture beetle, all species highly resistant to fumigants, were completely exterminated in a fumigation vault at a concentration of 1 pound of ethylene oxide per 1,000 cubic feet for 24 hours at 75° F., the insects being contained in cotton-stoppered vials and buried in overstuffed furniture. They recommended for commercial work that 2 pounds of ethylene oxide be the standard lethal dosage per 1,000 cubic feet of space for a contact period of 20 hours.

Ethylene oxide has one drawback in that it is a combustible and explosive gas when mixed with air. The lower limit of inflammability is 3.67 pounds per 1,000 cubic feet which exceeds the insecticidal concentration of the gas. The possibility, however, of fire hazard resulting from an accidental overdose or the concentration in one spot of an excess amount of the fumigant must be considered.

Jones and Kennedy (4) determined that mixtures in air of ethylene oxide and carbon dioxide in the ratio of 1 part of the former to 7.5 parts or more of the latter by weight are noninflammable. The presence of carbon dioxide removes the fire and explosion hazard attending the use of ethylene oxide and, therefore, satisfies the requirements of the Underwriter's Laboratories in this respect. It is significant in this connection that ethylene oxide and carbon dioxide have practically the same vapor density and so do not tend to separate or stratify.

Cotton and Young (5) demonstrated that the presence of carbon dioxide practically doubled the insecticidal action of ethylene oxide as a result apparently of acceleration of the respiration of the insects. Specimens of the rice weevil and confused flour beetle buried in the middle of tightly sealed cartons in a vacuum tank required a dosage of 6 pounds of ethylene oxide alone per 1,000 cubic feet for a complete kill in 45 minutes. Only 3 pounds per 1,000 cubic feet were necessary for a similar result when combined with 14 pounds of carbon dioxide.

Back, Cotton, and Ellington (6) determined the comparative dosages of ethylene oxide alone and combined with 14 pounds of carbon dioxide per 1,000 cubic feet in a vacuum tank, required to kill all insects infesting various types of food products. In the presence of carbon dioxide the dosages never exceeded one half of that for ethylene oxide alone for the same contact period.

The Carbide & Carbon Chemicals Corporation now supplies a mixture of ethylene oxide and carbon dioxide designated by the trade name of carboxide, compressed in cylinders in liquid form in the ratio of 1 part of ethylene oxide to 9 parts of carbon dioxide by weight. The cylinders are equipped with eductor tubes for the rapid withdrawal of the mixture atomized to a fine mist and immediately

assuming the gaseous form. Ten pounds of carboxide contain 1 pound of ethylene oxide by weight. From the insecticidal viewpoint, however, 10 pounds of the mixture corresponds roughly to 2 pounds of ethylene oxide.

There is no reason to believe that ethylene oxide alone, or combined with carbon dioxide as carboxide, will not be as effective against such pests as bedbugs, cockroaches, fleas, and lice, as against the types of insects which have already been considered. There are no published data in this connection. A personal communication, however, has been received by the writer from Dr. E. A. Back, principal entomologist, Bureau of Entomology, United States Department of Agriculture, presenting the following results of tests of the insecticidal effect of ethylene oxide on bedbugs:

In a 6-liter flask all stages of the bedbug, including the eggs, were killed in a 3-hour exposure by a concentration of 8.8 ounces per 1,000 cubic feet; adults in 24 hours by 1½ ounces; in 1 hour by 1½ pounds; and in 30 minutes by 2½ pounds per 1,000 cubic feet. The insects were exposed directly, the factor of deep penetration not being involved. The effect of carboxide was not studied.

The object of the present paper was to determine the minimum lethal dosage of carboxide gas for bedbugs and cockroaches under conditions requiring deep penetration for such periods of exposure as would be practicable on board ship without material interference with normal routine activities.

This research was conducted at the naval supply depot, Brooklyn, N.Y., as a combined project of the Bureau of Medicine and Surgery and the Bureau of Supplies and Accounts.

THE TOXICITY OF ETHYLENE OXIDE

Waite, Patty, and Yant (7) of the Bureau of Mines Experiment Station, Pittsburgh, Pa., determined the toxicity of ethylene oxide in air to guinea pigs with the following results:

	Ethylene oxide		Carboxide
	Percent	Pounds per 1,000 cubic feet	Pounds per 1,000 cubic feet
Kills in short time	5–10	5–10	50–100
Dangerous in 30 to 60 minutes	0. 3– 0. 6	0. 3– 0. 6	3–6
Maximum for 60 minutes without serious effects	. 3	. 3	3
Slight symptoms after several hours or maximum amount without serious harm	. 025	. 025	. 25

Carboxide was not included in the study but the third column has been calculated and added by the writer for comparison. These data for carboxide, in the case of insects, should be reduced by approximately one half. The toxicity of the mixture for guinea pigs has not been ascertained but it may be assumed that the toxicity of ethylene oxide in the presence of high carbon dioxide and for the same exposure time would be materially increased incident to the stimulating effect upon respiration. The toxicity of ethylene oxide is of the same general order as ammonia. The above authors point out that although ethylene oxide does not possess a distinctive enough odor to give warning of its presence in relatively low concentrations, it is fortunately an irritant and in that manner gives warning.

Comparison with the toxicity of hydrocyanic acid gas: Kobert (8) reported that 0.13 to 0.16 ounce of hydrocyanic acid gas per 1,000 cubic feet was dangerous in 30 to 60 minutes exposure. Waite, Patty, and Yant (7) found that 4.8 to 9.6 ounces was the corresponding figure for ethylene oxide for the same period of time. Hydrocyanic acid gas is, therefore, approximately 37 to 60 times more toxic than ethylene oxide. Ethylene oxide, therefore, in contrast to hydrocyanic acid gas is not highly toxic. It is now being widely used by fruit and nut shippers, in grain elevators, warehouses, and by commercial fumigators. It is generally recognized that with ordinary care in fumigation a gas mask is not required for ethylene oxide and expert personnel is unnecessary.

TECHNIQUE

(A) The Fumigating Chamber

The tests were conducted in a practically airtight chamber 18 feet long, 10 feet wide, and 11 feet in height; the cubical capacity being 1,980 cubic feet. One end of the chamber is illustrated in figure 1. The chamber is constructed for fumigation operations, the walls being of terra-cotta tiles, cement plastered inside. The floor and ceiling correspond to that of the space in the building from which the chamber has been sectioned off. The walls and ceiling are sealed by two coats of shellac. There is one window in the outside wall provided with reinforced glass. The airtight door is equipped with a special type of rubber gasket.

A ventilating system is provided for recirculating the air of the chamber in order to afford rapid admixing of the fumigant. This consists of a motor blower assembly located outside of the room, of a capacity of approximately 167 cubic feet per minute, with piping of an insidde diameter of 5 inches. This effects 2½ theoretical changes of the air of the chamber in 30 minutes. Figure 2 shows

the motor blower assembly A, the exhaust line from the chamber B and the supply line C. The exhaust line B by proper shifting of valves can be quickly bypassed to line D, which connects with the outside air. The chamber was rapidly cleared of the fumigant in this manner at the end of an experiment.

In figure 1 is shown the end of the exhaust line B projecting through the wall of the chamber. The supply line C enters close to the ceiling and extends to the opposite wall of the chamber.

(B) Introduction of the Carboxide

A cylinder of carboxide as shown in E in figure 3, was placed upon the platform of a set of scales, the discharge tube of the cylinder being connected by flexible tubing through the valves of the manifold as shown, to the inlet pipe F which is of about ¾-inch internal diameter. The entrance of F into the chamber is shown in figure 1. This pipe continues downward finally discharging its contents into the chamber at a short distance above the floor. The discharge end of F is contained within a galvanized iron casing, open above and below but enclosed on four sides, as shown in figure 1.

The procedure for the setting up of a definite concentration of carboxide in the fumigating chamber was as follows: the door was first securely sealed. The cylinder of carboxide was then balanced on the scales shown in figure 3 and the latter then adjusted to the number of pounds of carboxide which it was desired to release. The valves of the manifold being opened to pipe F, the valve of the cylinder was quickly opened wide and immediately closed on the latter again reaching equilibrium. The blower was immediately placed in operation and the air of the chamber recirculated for the first 30 minutes of the experiment.

(C) Position of the Insects

The insects were contained in large-sized round pasteboard pill boxes in the great majority of the tests, the cover being punctured with numerous pin holes to afford sufficient air for breathing. In a few instances a sliding form of box of medium size of the type employed for powders was used. The boxes were placed in various positions affording varying degrees of harborage for the insects, which are described and indicated by numerals as follows:

Key no.

1. In pill box with perforated cover in open.
2. Between pages of magazine on deck.
3. Between mattress and springs of bunk.
4. Upper top compartment of chiffonier, middle of clothing, doors open.
5. Upper top compartment of chiffonier, bottom under clothing, doors open.
6. Lower top compartment, middle of clothing, doors open.
7. Drawer 1, middle of clothing, open.

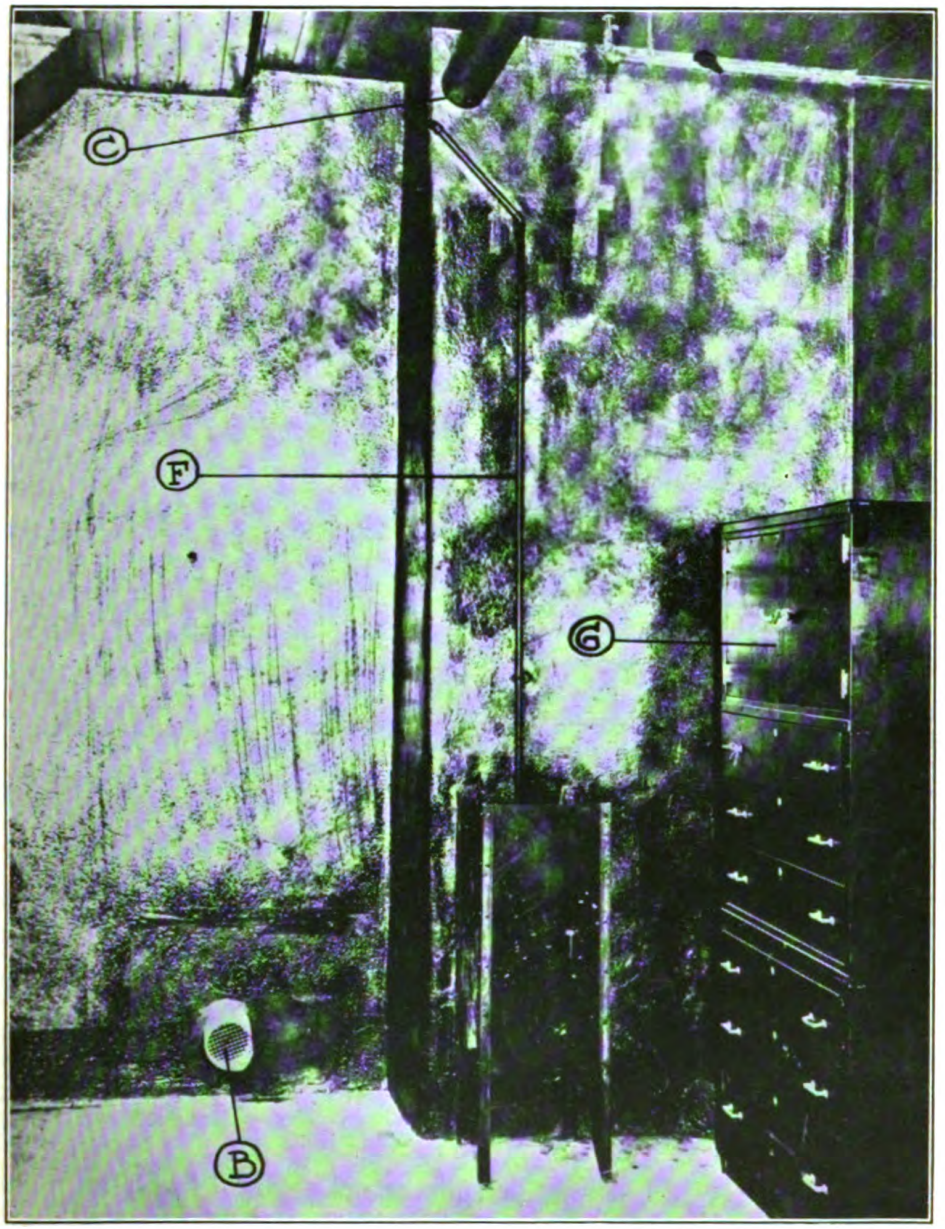

FIGURE 1.—END VIEW OF INTERIOR OF FUMIGATION CHAMBER

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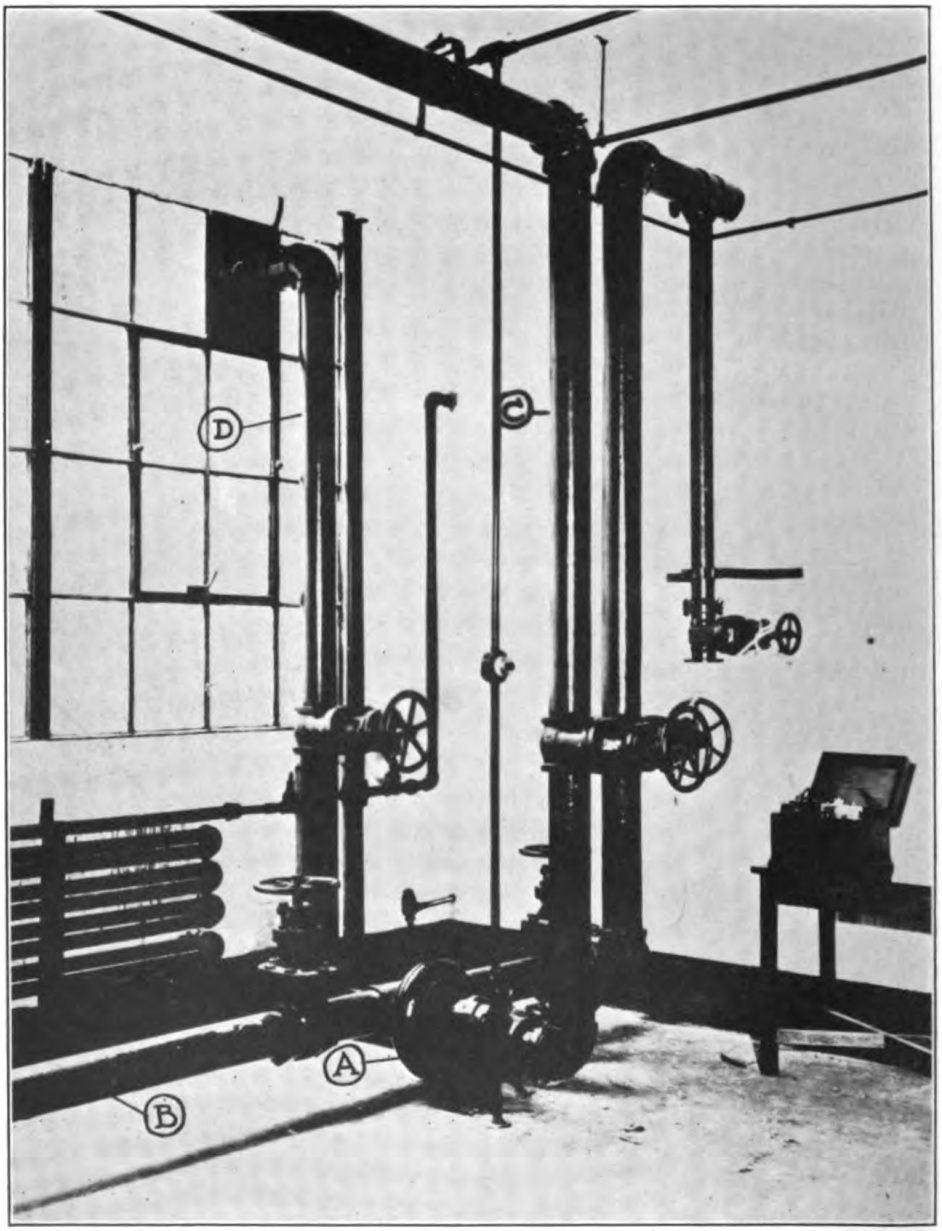

FIGURE 2.—VENTILATION SYSTEM FOR FUMIGATION CHAMBER.

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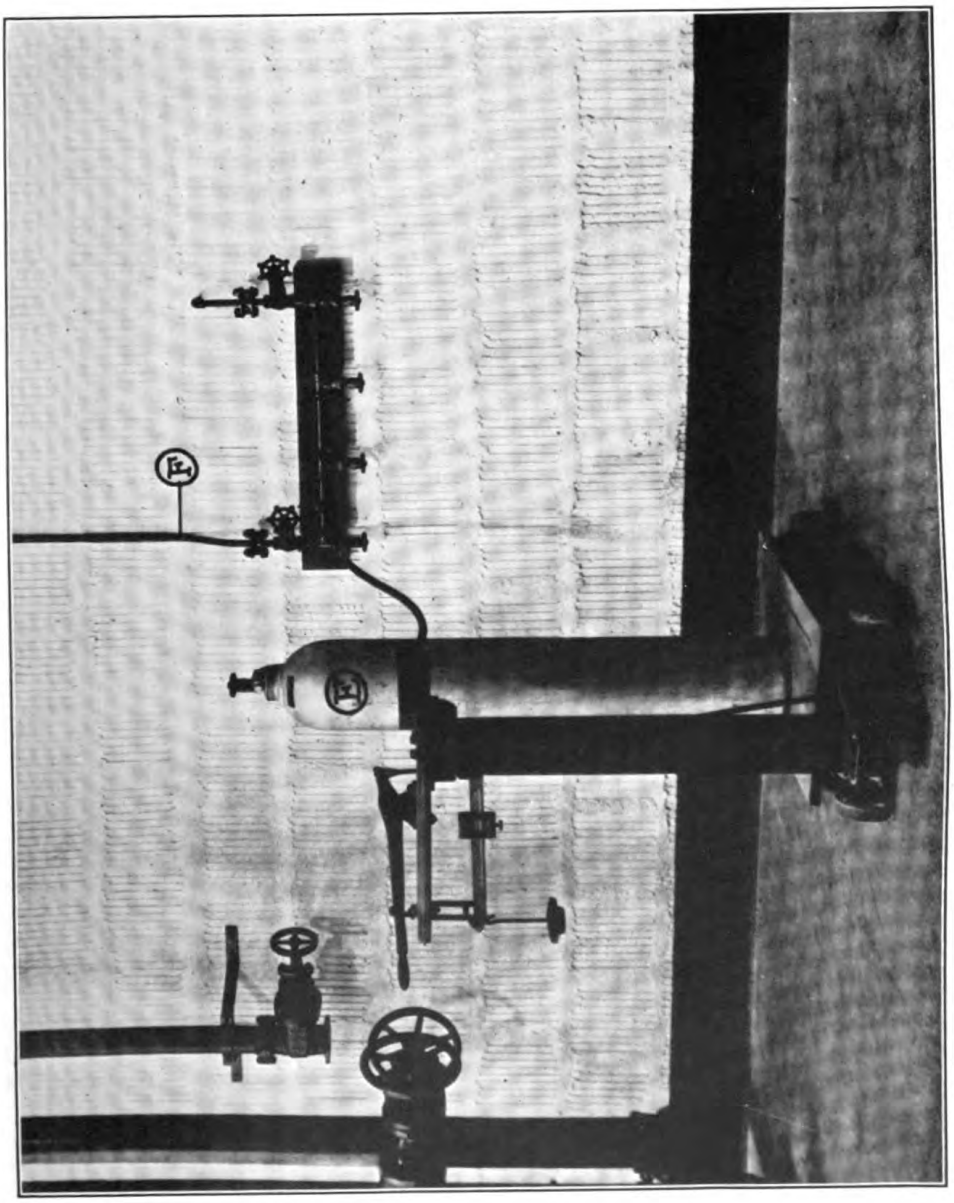

FIGURE 3.—CYLINDER OF CARBOXIDE GAS CONNECTED TO FUMIGATION CHAMBER.

Key no.

8. Drawer 1, bottom under clothing, open.
9. Drawer 5, bottom under clothing, open.
10. Drawer 5, middle of clothing, open.
11. Drawer 6, middle of clothing, open.
12. Drawer 6, bottom under clothing, open.
13. Furniture cover, 15 by 9 feet, turned in from sides and rolled tight, with final eight thicknesses; in middle of.
14. Furniture cover, 15 by 9 feet, turned in from sides and rolled tight, with final 16 thicknesses; in middle of.
15. Tarpaulin, 28 by 9 feet, turned in at sides and rolled up; in middle of.
16. Bale of rags, 24 inches long, 20 inches wide, 20 inches high; in middle of.
17. Closed bag of kidney beans; buried 6 inches below surface.
18. Four to seven mattresses stacked on top of each other; between center of bottom mattress and deck.
19. Filing cabinet; in closed drawer between papers in file holder.
20. Metal locker; in closed section of, 5 feet above deck.
21. Between two blankets at center of bunk made up as usual.
22. In inside pocket of coat suspended with other clothing in close contact on both sides.
23. Office desk, closed drawer; inside of, on top of papers.
24. Office desk, middle drawer closed; in middle of thick layer of papers.
25. Office desk, on bottom of closed drawer under mass of papers filling the drawer.
26. Office desk; wrapped tightly in four layers of cotton cloth in closed drawer.
27. Office desk; inside of small pasteboard carton in closed drawer.
28. Center of hair of standard Navy mattress rolled up and secured in that position.
29. Center of hair of mattress on top of desk enclosed in mattress cover, and made up with 2 sheets and 2 woolen blankets to simulate a bed.
30. As under (21) except that specimen placed between mattress cover and sheets.
31. Upper top compartment of chiffonier, middle of clothing, doors closed.
32. Upper top compartment of chiffonier, bottom under clothing, doors closed.
33. Lower top compartment of chiffonier, middle of clothing, doors closed.
34. Lower top compartment of chiffonier, bottom under clothing, doors closed.
35. Drawer 1 of chiffonier, middle of clothing, closed.
36. Drawer 1 of chiffonier, bottom under clothing, closed.
37. Drawer 5 of chiffonier, middle of clothing, closed.
38. Drawer 6 of chiffonier, middle of clothing, closed.
39. Drawer 6 of chiffonier, bottom under clothing, closed.

Nos. 2 to 12 require a moderate degree of penetration for the fumigant to reach the insects; nos. 13 to 39, a relatively high degree.

The chiffonier G above referred to is shown in figure 1. The actual position, however, in the tests, instead of being as here shown, was on the opposite side of the entrance to the chamber but otherwise in the same relative location. The inside measurements of each of the two top compartments of the chiffonier are 8½ inches high, 32 inches long, and 20 inches wide; of the drawers, 6½ inches high, 32 inches long, and 20 inches wide. The top compartments and

drawers were completely filled with clothing as tightly packed as possible, consisting of blue and white jumpers and trousers such as are issued as part of the clothing allowance for enlisted personnel.

The desk is of the office type, the top measuring 30 inches above the floor; the inside measurements of each drawer being 23 inches long, 10 inches wide, and 4½ inches high. This is the desk referred to above in connection with the make-up of the bed under (29). The mattresses used were 74 inches long, 28 inches wide, and 3 inches thick.

(D) The General Procedure of the Tests

The test boxes of insects for each experiment varied in number from 4 to 10. The number of insects per box was ordinarily uniform for an individual experiment but varied from 4 to 10 in different tests, averaging 6 and 7. At the close of the experiment the door of the chamber was opened a short distance and the blower immediately started, the air of the chamber then exhausting through line D of the ventilation system to the exterior of the building. At the end of approximately 5 minutes the test boxes were removed to the outside of the room, the number of dead insects recorded and the boxes then placed in an incubator at a temperature of 74° F. overnight. The following morning at 9 a.m. the insects were again inspected to determine the final result.

The range of temperature of the fumigation chamber during each test was recorded in degrees Fahrenheit by means of a maximum and minimum type of thermometer. The chamber was heated by means of an ordinary steam radiator.

(E) Controls

Control tests were conducted in the great majority of the experiments. The insects were distributed in approximately the same number of containers and same number per container and kept outside of but in the vicinity of the fumigation chamber during the experiment. In certain tests more controls than test insects were used. At the conclusion, they were placed in the incubator along with the test specimens, a record of the number of fatalities, if any, being made the following morning.

DISCUSSION OF EXPERIMENTAL DATA

(A) Tests with Bedbugs

The detailed data of the tests with bedbugs are presented in table 1.

The headings of the tables are in general self explanatory. The number and location of specimen boxes are indicated under Position of Insects. By Immediate Lethal Effect is indicated the lethal effect on the insects as shown at the end of the exposure period. The end

results are classified with respect to complete, partial, and no immediate lethal effect. Under Ultimate Kill is recorded the result the following morning after remaining in an incubator overnight. The data for temperature for tests 2a, 4, and 30, were taken at the end of the tests. In the remainder the minimum and maximum temperatures of the test are indicated. For the sake of brevity, throughout the remainder of this paper, the concentration of carboxide will be designated only as pounds, the volume of 1,000 cubic feet of air space being understood.

TABLE No. 1.—*Carboxide tests with bedbugs*

Test no.	Time exposed, hours	Concentration, pounds per 1,000 cubic feet	Number of insects exposed	Position of insects	Temperature, Fahrenheit	Ultimate kill	Number of controls	Number of controls dead	Immediate lethal effect
30	3	15	24	29, 34, 36, 39.	70	Complete	24	None	Partial: 34, 36, 39. None: 29.
2a	3	12.7	48	25, 18, 3, 19, 2 "20, 21, 22.	68	do	None		Partial: 3, 19, 2, 21, 22. Complete: 20. None: 25, 18.
4	3	9	5	13	74	do	5	None	Partial: 13.
6	3	9	20	14, 16, 23, 28.	68–70	do	20	4	None: 14, 16, 23, 28.
5	3	6	15	1, 13, 16, 28.	68–70	do	20	None	Partial: 28. None: 1, 13, 16.
33	3	6	24	29, 34, 36, 39.	73–74	do	24	None	Partial: 29. None: 34, 36, 39.
39	3	5	24	29, 34, 36, 39.	72–74	do	24	None	None: 29, 34, 36, 39.
40	3	5	24	29, 34, 36, 39.	73–76	do	24	2	Do.
13	3	4	53	1, 29, 30, 31, 36, 38.	85–88	Part: 31. Others complete.	None		None: 1, 30, 31, 36, 38. Partial: 29.
14	3	4	70	10, 16, 29, 30, 31, 35, 39.	75–77	Complete: 35, 10, 39. Part: 16, 24, 30, 31.	60	None	
16	3	4	46	10, 29, 30, 31, 38.	70–72	Complete	None		None: 10, 31, 38. Partial: 29, 30.
23	3	4	42	16, 29, 30, 33, 36.	69–72	do	24	None	Partial: 16, 29, 30, 33, 36.
15	3	2	60	29, 30, 31, 36, 10, 38.	74–76	None	None		None.
29	6	12	30	29, 30, 33, 36, 38.	65–69	Immediate	None		Complete.
37	6	12	24	29, 34, 36, 39.	72–76	do	24	None	Do.
7	6	9	20	14, 16, 26, 28.	69–71	Complete	10	2	None: 16, 26. Complete: 14, 28.
8	6	6	30	14, 16, 24, 26, 27, 28.	72–73	do	35	None	None: 14, 16, 26, 27. Partial: 24, 28.
9	6	6	80	1, 4, 6, 7, 9, 11, 12, 25.	71–73	do	60	14	None killed.
10	6	6	51	4, 6, 7, 9, 12, 25.	70–84	do	50	1	Do.
11	6	4	62	1, 10, 29, 30, 31, 35, 36, 38.	76–78	do	None		Partial: 1, 10, 30, 36. None: 31, 35, 38. Complete: 29.
12	6	4	80	1, 29, 30, 31, 35, 36, 37, 39.	75–78	do	60	7	Complete.
17	6	4	34	29, 30, 32, 38.	72–74	do	25	None	None killed.
18	6	4	30	10, 29, 30, 32, 36, 38.	74–78	do	None		Do.
41	6	3	24	29, 34, 36, 39.	75–76	Immediate	24	None	Complete.
42	6	3	24	29, 34, 36, 39.	72–74	do	24	2	Do.
19	12	4	60	10, 29, 30, 32, 36, 38.	74–78	do	40	None	Do.
20	12	4	42	10, 16, 29, 30, 33, 36, 38.	70–72	do	60	2	Do.
31	12	3	24	29, 34, 36, 39.	72–75	do	24	2	Do.
38	12	3	24	29, 34, 36, 39.	70–72	do	24	2	Do.
26	18	10	60	10, 29, 30, 33, 36, 38.	63–68	do	None		Do.

TABLE No. 1.—*Carboxide tests with bedbugs*—Continued

Test no.	Time exposed, hours	Concentration, pounds per 1,000 cubic feet	Number of insects exposed	Position of insects	Temperature, Fahrenheit	Ultimate kill	Number of controls	Number of controls dead	Immediate lethal effect
25	18	7.5	36	10, 12, 29, 30, 33, 36.	68–70	Immediate	None		Complete.
27	18	5	36	10, 29, 30, 33, 35, 38.	67–70	do	36	3	Do.
24	18	2.5	36	10, 29, 30, 33, 36, 38.	72–75	do	None		Do.
28	18	2.5	36	29, 30, 33, 35, 37, 38.	65–70	do	36	1	Do.
34	18	2	24	29, 34, 36, 39.	72–74	do	24	2	Do.
36	18	2	24	29, 34, 36, 39.	69–72	do	36	2	Do.
32	24	2.5	24	29, 34, 36, 39.	72–76	do	24	3	Do.
21	24	2	42	10, 16, 29, 30, 33, 36, 38.	70–73	Complete	36	None	Partial: 29, 30, 33, 36. Complete: 10, 16, 38.
22	24	2	42	10, 16, 29, 30, 33, 36, 38.	67–72	do	24	None	Partial: 10, 29, 30, 33, 36, 38. Complete: 16.
35	24	2	24	29, 34, 36, 39.	72–73	do	24	None	Partial: 29, 36. Complete: 34, 39.

Exposure of 3 hours.—The concentration of carboxide varied from a maximum of 15 to a minimum of 2 pounds. There was a complete ultimate kill down to and including a dosage of 5 pounds. With 4 tests at 4 pounds each, 2 experiments resulted in complete and 2 in partial extermination. With 2 pounds all insects escaped destruction.

It will be noted in these, and practically all of the subsequent tests of the table, that bedbugs tended to exhibit a peculiar delayed effect in response to carboxide, specimens showing only partial or even no lethal effect at the end of the test but on the following morning a complete kill. Thus in experiment 5 we find 1 lot showing a partial immediate kill, the remaining 4 no immediate fatalities; in experiment 6 no immediate lethal effect whatever. This delayed action was characteristic throughout. It was frequently observed, however, that the insects even though surviving the period of exposure were greatly reduced in activity.

Test 2a is of especial interest in that it was conducted abroad a tug, the U.S.S. *Sagamore*, at the New York Navy Yard, in place of the fumigation room. The crew's berthing space, 3,175 cubic feet capacity, was selected for the test. Connecting with and open to this compartment from below was a magazine space of 756 cubic feet capacity, the total air space fumigated being approximately 3,931 cubic feet. The compartment was readily rendered airtight by closing all air ports, the access hatch above and one deck ventilator. Air movement was maintained in the compartment during the test by means of a standard portable motor blower ventilating set provided with a long canvas duct on the discharge side. By means

of this arrangement air was drawn from one corner of the space at the deck level and discharged at a point just under the access hatch above. The test boxes of insects were located in such positions as the following: In closed drawer of filing case between papers; inside pocket of a coat suspended on a hanger with adjacent clothing in close contact on both sides; under mass of papers in closed desk drawer; underneath seven mattresses stacked up one above the other on deck; between blankets on mattress. Sufficient carboxide was admitted to set up a concentration of 12.7 pounds.

At the completion of the exposure time the hatch was opened and the canvas discharge duct of blower so directed as to discharge toward the outside air. The compartment was entered at the end of 13 minutes and specimens of insects examined, no evidence of ethylene oxide being present beyond the faintest perceptible odor. The temperature on opening compartment was 68° F. All insects of this test ultimately succumbed to the fumigant.

This berthing compartment on the U.S.S. *Sagamore* had been heavily infested with bedbugs for many weeks up to the time of the present experiment. Careful and detailed inspections for bedbugs 10, 21, and 30 days subsequent thereto resulted negatively, thus affording presumptive evidence of destruction of the eggs.

It will be noted from table 1 that control specimens in numbers from 5 to 60 were included in 9 of the 13 tests with a 3-hour exposure period. Only 4 insects in 1 and 2 in another test failed to survive.

Exposure of 6 hours.—The ultimate kill was complete for all concentrations employed, i.e., 12, 9, 6, 4, and 3 pounds. The immediate lethal effect was complete at 12 and 3 pounds. If complete at 12 and 3 pounds the question naturally arises why was the immediate effect incomplete at intermediate concentrations? Differences of temperature of the chamber in different tests were not considerable and, therefore, cannot be regarded as a causative factor. Variation in the strain of the insects with increased susceptibility to the fumigant may be a factor. The delayed lethal response to carboxide already alluded to was frequently observed in this group.

Control specimens were carried in 9 of the 12 tests, the number varying from 10 to 60. All controls survived in 4 tests; in the remaining 5 experiments the maximum number dying was 14 out of 60 insects; the minimum, 1 out of 50.

Exposure of 12 hours.—At concentrations of 4 to 3 pounds, respectively, an immediate complete kill resulted, the controls showing a negligible number of fatalities.

Exposure of 18 hours.—At concentrations of 10, 7½, 5, 2½, and 2 pounds, respectively, an immediate complete kill resulted in each instance, the control examinations yielding an occasional fatality.

Exposure of 24 hours.—A complete ultimate kill resulted with concentrations of 2½ and 2 pounds.

Additional control tests.—Two tests were conducted in which the test boxes of insects were actually placed for a period of 12 hours in the positions ordinarily occupied by the experimental group, but not exposed to carboxide. In 1 test a total of 60 bedbugs was used, distributed equally in the following positions: 9, 11, 16, 29, 31, 33; in the second test a total of 30 in positions 29, 30, 33, 35, 37, 28. The number of insects succumbing was negligible; 5 in the first and 4 in the second test.

The factor of temperature.—An increase of temperature up to a certain point has a stimulating effect upon the respiratory processes and, therefore, in itself increases the susceptibility of an insect to a toxic gas. Such ranges of temperature were recorded in the 3-hour tests as 68–70, 72–74, and 73–76; in the 6-hour tests as 71–73, 72–74, and 76–78; in the 12-hour tests as 70–72 and 74–78. It is concluded that the fumigation of bedbugs with carboxide proceeds satisfactorily at temperatures of 68 and above.

(B) Tests with Cockroaches

The number of experiments with cockroaches was considerably less than with bedbugs on account of the difficulty of securing insect material at the time. Different species of the insect were included but no attempt at identification was made. The detailed data are presented in table 2.

Three-hour tests with concentrations of 9, 6, and 4 pounds all resulted in a complete ultimate kill. Six-hour tests with dosages of 9, 6, and 4 pounds resulted similarly. A single test for 12 hours with a dosage of 4 pounds showed an immediate lethal effect. The tendency to a delayed lethal action of carboxide already emphasized in the tests with bedbugs was also prominent in these experiments with cockroaches. The temperatures of the fumigation chamber were of the same general order as in the experiments with bedbugs.

Seven of the ten experiments were supplemented by control tests. The mortality of the insects was negligible in comparison with the experimental specimens.

The conclusion is drawn from this rather limited set of tests that the adult cockroach approximates the bedbug in susceptibility to the toxic effect of carboxide. The writer has been advised as a result of consultation with representatives of the Bureau of Entomology, United States Department of Agriculture, that the cockroach tends to be no more resistant to the lethal effect of fumigants in general than the bedbug.

TABLE No. 2.—*Carboxide tests with cockroaches*

Test no.	Time exposed, hours	Concentration, pounds per 1,000 cubic feet	Number of insects exposed	Position of insects	Temperature, Fahrenheit	Ultimate kill	Number of controls	Number of controls dead	Immediate lethal effect
4	3	9	15	1, 13, 16, 28.	74	Complete	None	-------	Partial: 1, 13, 16, 28.
6	3	9	10	14, 16	68–70	do	10	3	None: 14, 16.
3	3	6	56	1, 13, 15, 16, 17, 28.	74–75	do	24	4	None: 13, 28. Partial: 1, 15, 16, 17.
5	3	6	20	1, 13, 16, 28.	68–70	do	20	None	None: 1. Partial: 13. Complete: 16, 28.
14	3	4	30	35	75–77	do	None	-------	None: 35.
7	6	9	20	14, 16, 26, 28.	69–71	do	20	5	None: 26. Partial: 16. Complete: 14, 28.
8	6	6	14	14, 16, 23, 26, 27, 28.	72–73	do	19	3	None: 14, 16, 23, 26, 27. Complete: 28.
9	6	6	16	1, 5, 7, 9, 11.	71–73	do	None	-------	None killed.
17	6	4	32	8, 16, 37	72–74	Immediate	5	None	Complete: 8, 16, 37.
19	12	4	10	10, 36	74–78	do	10	None	Complete: 10, 36.

THE QUESTION OF DESTRUCTION OF THE EGGS

It was not practicable to include in the present work an adequate study of the minimum time-concentration dosages of carboxide necessary to destroy the eggs of the bedbug and cockroach. The unpublished data of Back already referred to on page 256, disclosed that a dosage of 8.8 ounces of ethylene oxide per 1,000 cubic feet in a 3-hour exposure was lethal to both bedbug eggs and the various stages of the adult. Assuming that the toxicity of ethylene oxide in the form of carboxide is doubled, this dosage would be equivalent to 2.7 pounds of carboxide per 1,000 cubic feet. While the above concentration of 8.8 ounces was not a minimum lethal dose, still it adduces presumptive evidence that the egg approximates the adult in susceptibility. It is presumed that the bedbug egg, being laid singly and not encapsulated, would not offer marked resistance to penetration by ethylene oxide. It is probable that a single fumigation with the dosage of the minimum lethal order for the adult will be adequate for the eggs. This, however, has not been experimentally established.

The eggs of the cockroach instead of being laid separately are brought together within the abdomen of the mother insect into a hard horny pod or capsule. The capsule will naturally offer increased resistance to penetration in contrast to that of the bedbug egg. It is probable, therefore, that a minimal lethal dosage for the adult stages of the cockroach will not kill the eggs, a second fumigation being required at the end of 10 days to destroy the newly hatched insects.

APPLICATION TO FUMIGATION OF NAVAL VESSELS

The question arises as to the efficacy of the minimum lethal dosages of carboxide determined in these tests as applied to the spaces of naval vessels. If an entire watertight compartment is under consideration, these concentrations should be adequate, as complete airtightness is assured. On the other hand, such spaces as the following will require special preparation for sealing: galleys, pantries, officers' staterooms, chief petty officers' berthing spaces, and sick bays. The effectiveness of these dosages for the exposure periods specified will depend upon the relative degree of airtightness secured. It may be necessary to increase the dosage of carboxide under these conditions but in all probability not greater than a doubling of the concentration as a maximum. Thorough measures should be taken to reduce the harborage of insects to a minimum in any space to be fumigated.

An adhesive tape available in various widths, known commercially as masking tape, is now available for the convenient sealing of such locations as the seams of doors and transoms, and the cracks at entrances of piping through bulkheads. It has the great advantage of leaving no residue adhering to wood or metal when removed. Ethylene oxide will penetrate ordinary paper used for sealing but this is prevented by smearing the surface with vaseline or engine grease.

Practical tests should be conducted aboard ship with the object of developing a simple standardized procedure for sealing all such spaces and determining the minimal lethal dosage of carboxide adequate under all conditions.

SUMMARY

Tests were conducted in a relatively airtight chamber of approximately 2,000 cubic feet capacity, with the object of determining the minimal lethal dosage of carboxide gas for bedbugs and cockroaches, under conditions imposing relatively deep penetration.

The insects were contained chiefly in pill boxes, the latter being buried in such locations as the following: middle of a bale of rags; in the middle of a furniture cover turned in from the sides and rolled tightly in 16 thicknesses; at the bottom of a closed chiffonier drawer completely filled with clothing; center of hair of a mattress with mattress cover and made up with sheets and two heavy blankets to simulate a bed.

The following tests were conducted with bedbugs with the dosages in pounds per 1,000 cubic feet and results as indicated:

Twenty-four-hour exposure: 2½ and 2 pounds; both completely lethal.

Eighteen-hour exposure: 10, 7½, 5, 2½ and 2 pounds; all completely lethal.

Twelve-hour exposure: 4 and 3 pounds; both completely lethal.

Six-hour exposure: 12, 9, 6, 4, and 3 pounds; all completely lethal.

Three-hour exposure: 18, 12, 9, 7, 6, 5, 4, and 2 pounds; complete lethal down to and including 5 pounds, incomplete or negative at 4 and 2 pounds.

The following tests were conducted with cockroaches with results as indicated:

Twelve-hour exposure: 4 pounds; lethal.

Six-hour exposure: 9, 6, and 4 pounds; all completely lethal.

Three-hour exposure: 9, 6, and 4 pounds; all completely lethal.

The great majority of the tests with both bedbugs and cockroaches were controlled with a corresponding number of insects subjected to practically parallel conditions except exposure to carboxide. The mortality of the control specimens was negligible.

The following minimum lethal concentrations per 1,000 cubic feet, in a relatively airtight space, are recommended for both bedbugs and cockroaches: 5 pounds for 3 hours, 3 pounds for 6 hours, 3 pounds for 12 hours, 2 pounds for 18 hours, and 2 pounds for 24 hours.

Carboxide gas is noninflammable and nonexplosive; noninjurious to fabrics, furniture, or food products; of about one thirty-seventh to one sixtieth of the toxicity of hydrocyanic acid gas for man; and is not prohibitive from the standpoint of cost.

ACKNOWLEDGMENTS

The cordial interest and generous cooperation of Capt. J. F. Hatch, Supply Corps, United States Navy, Lt. F. Schwab, Supply Corps, United States Navy, and Mr. Frederick Krassner, chief chemist of the naval supply depot, Brooklyn, N.Y., is gratefully acknowledged. The writer is particularly indebted to Lieutenant Schwab for his unremitted energy and highly efficient assistance in the conduct of all of the experiments.

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ACUTE PANCREATIC NECROSIS OCCURRING DURING GENERAL ANESTHESIA

With Report of Three Cases [1]

By D. J. Cracovaner, lieutenant (junior grade), Medical Corps, United States Navy

In the April 1932 number of the United States Naval Medical Bulletin, there appeared a preliminary notice of three cases dying under a general anesthetic in which the tissues from autopsy showed an acute hemorrhagic necrosis of the pancreas of extreme degree as the cause of death.

In this article the writer reports these cases in more detail and discusses the possible modes of causation of these unusual catastrophies.

A nonexhaustive search of the literature has failed to yield a single case of acute pancreatic necrosis occurring during the administration of a general anesthetic. Gwathmey in his text on anesthesia does not mention this condition as a possible complication of general anesthesia.

Case Reports

Case I.—D. B. B., age 30 years. Admitted to the hospital January 29, 1930, with a diagnosis of gastritis, chronic. Patient complained that he had had a "sensitive" stomach since 1927. During those 3 years his appetite had been indifferent and he suffered varying degrees of distress in the epigastrium after meals. For a period of 2 weeks prior to admission he had constant pain in the epigastrium, more pronounced after ingestion of food, the pain radiating to back and under the lower ribs. His appetite was very poor. The roentgenologist reported "typical deformity of duodenal ulcer." The patient was placed on a Sippy diet and improved symptomatically. On March 14, 1930, X-ray examination showed the duodenal cap deformed; the 24-hour plate revealed the second portion of the duodenum to be dilated; the 48-hour film showed marked stasis of the appendix. The impression was that the upper intestinal tract findings might be due to reflex from a chronic appendicitis. The patient was prepared for operation.

[1] The pathological studies were made by Lt. Comdr. O. Wildman, Medical Corps, United States Navy, who published a preliminary notice of these cases in the United States Naval Medical Bulletin, vol. XXX, no. 2, April 1932.

Operation.—The patient was placed on the table in good condition. The choice of anesthetic was a combination of ethylene, oxygen, and a small amount of ether. A laparotomy was performed revealing abdominal adhesions, a band of which compressed the duodenum. The patient had been under the anesthetic for 15 minutes when he suddenly ceased breathing, became pulseless, the heart stopping shortly afterwards. All attempts at resuscitation were fruitless.

At autopsy the pancreas was found to be soft, mushy and diffusely hemorrhagic. Anatomical diagnosis was acute hemorrhagic pancreatitis; abdominal adhesions; atheromata of the aorta; scar formation in the liver.

Sections of the pancreas in ten percent formalin were submitted to the United States naval medical school for pathological examination. The pathological report follows:

Gross.—Specimen consists of five pieces of tissue from pancreas. The capsule is whitish in color. The lobulations are grayish-white indistinct areas surrounded by dark brown zones resembling degenerated blood.

Microscopical.—Necrosis is extreme and wide-spread, the parenchymatous tissue in some sections being completely necrosed, in others showing small portions of acini in which the nuclear structure remains. The interlobular tissue contains large masses of degenerated blood in which only the shadows of red cells can be made out. Some of the necrotic lobules also contain masses of degenerated blood. Areas of fat necrosis are also seen. There is no inflammatory infiltration. The hemorrhage indicates that this is not a post-mortem change.

Pathological diagnosis.—Acute hemorrhagic necrosis, pancreas.

Case II.—O.A.H., age 38 years. While on duty in Haiti (November 1928–April 1931) patient had suffered from recurrent right submaxillary abscesses with formation of salivary calculi. In 1929 a radical operation for the removal of the calculus-forming right submaxillary gland had been performed. On September 17, 1931, patient experienced pain and swelling in the right inframandibular region, which became progressively worse, swallowing proving painful and somewhat difficult. The patient was admitted to the hospital September 24, 1931. Temperature was 100.2° F.; pulse, 84; respiration, 20. Physical examination revealed the floor of the mouth swollen, pushing the tongue upward and backward; the right side of the neck, in the region of the angle of the jaw, was swollen and tender; pressure in this area caused a little pus to exude in the floor of the mouth at two places, at the opening of Wharton's duct and on the floor of the mouth opposite the right first molar. The scar of the previous operation was noted. Physical examination was otherwise negative.

The patient was put to bed, treated palliatively with large, hot, moist boric acid compresses continually to face and neck, fluids forced, purge, etc. He spent a restless night. The next morning, September 25, he complained of increased pain upon swallowing and examination showed the swelling in the neck and floor of the mouth to have increased. It was decided to operate.

Operation.—The anesthetic was begun at 2:15 p.m. Ether by drop method was chosen as the safest and best type of anesthesia in this case, and was administered by a trained anesthetist. The operation was incision and drainage of the abscess in the floor of the mouth. About 75 cubic centimeters of pus were evacuated, care being taken that the patient did not aspirate any. A calculus was felt in the abscess cavity and it was apparently encysted. It was decided to remove the calculus at a later date. The anesthesia was stopped at 2:50 p.m., the patient having been under its influence 35 minutes. At no time during the operation was the patient beyond a mild surgical

anesthesia. At 3 p.m. the patient suddenly went into shock and ceased breathing. All efforts at resuscitation, continued for an hour, proved futile and the patient was pronounced dead at 4 p.m.

An autopsy was performed at the United States naval medical school. Pathological diagnosis was as follows: Acute hemorrhagic parenchymatous necrosis, pancreas; acute parenchymatous degeneration, brain, heart, liver, kidneys, duodenum; pulmonary edema and congestion; acute passive congestion, brain, liver, spleen, adrenals, kidneys; abscess, right submaxillary, incised. The tracheo-bronchial tree was free of any pus, blood, or other foreign substance.

The findings in the pancreas were as follows:

Gross.—Normal in size, rather soft, dark red in color. On section the cut surface is diffusely and markedly hemorrhagic. Lobulations are indistinct. Weight, 165 grams.

Microscopical.—There is almost complete necrosis of parenchymatous tissue, some portions having undergone complete disintegration. Connective tissue and blood vessels show effects of digestive action and considerable hemorrhage has occured. Yellowish pigmentation in the fat is noted, due to fat necrosis.

Case III.—R.C., age 37 years. Patient was admitted to the hospital November 10, 1931, with the diagnosis of osteochondritis, left sterno-clavicular articulation, traumatic. The patient had suffered a stab wound over the sternum in April 1930 and been operated upon the same night. The wound failed to heal and drained continuously. The condition had become progressively worse up to the time of admission. Deep breathing caused sharp pain in the region of the manubrium. He complained of considerable morning cough, productive of frothy material which frequently had been blood streaked. Physical examination revealed a shallow depressed ulcer about 1 centimeter in diameter at the junction of the third rib and the sternum. A sinus extended from the ulcer upward and backward to the left clavicle. From the sinus exuded a suppurative discharge. The physical examination was otherwise negative. A month of conservative treatment yielded unsatisfactory results and operative interference was deemed advisable.

Operation.—Choice of anesthetic was nitrous oxide and ether which was begun at 11:20 a.m. The operation was to be incision and curettement of the sinus tract and packing with iodoform gauze. On the table the patient did not stand the anesthesia well. The operation was not completed and the anesthetic was stopped at 12:10 p.m. The patient reacted very poorly from the anesthesia and a special watch was assigned to him. At 1 p.m. the patient gave a sudden gasp and ceased breathing. Efforts at stimulation were of no avail and patient was pronounced dead at 1:10 p.m.

At autopsy the pancreas was found to be soft and through the capsule numerous hemorrhagic areas could be seen. On cut section there were multiple hemorrhagic areas throughout the pancreas. Sections of the organs in 10 percent formalin were submitted to the United States naval medical school for pathological examination. The following pathological diagnosis was made: Acute hemorrhagic necrosis, pancreas; acute parenchymatous degeneration heart, liver, kidneys; pulmonary edema; acute passive congestion, lungs, spleen, liver, kidneys.

Microscopical examination of the pancreas showed practically all the parenchymatous tissue to be obliterated due to acute necrosis and digestion. Interstitial tissue was also necrotic. There was marked hemorrhage in which the red blood cells appeared largely as shadows. Peripheral fatty tissue showed yellowish areas of fat necrosis.

Discussion

Acute pancreatic disease was first classified by Fitz (1) in 1889 into three types—hemorrhagic, gangrenous, and suppurative pancreatitis. Opie and Meakins (2) have shown that the essential pathological lesion is not inflammatory but a necrosis of the pancreatic cells or masses of cells. This is primary. They suggested what had been called acute hemorrhagic pancreatitis ought to be called acute hemorrhagic necrosis of the pancreas. Later, Guleke (3) suggested using the simpler term acute pancreatic necrosis since hemorrhage, gangrene, and suppuration are merely complications.

Most authorities believe the immediate cause of the gland necrosis is the activation of the pancreatic juice by some agent or other within the substance of the gland. In normal conditions the trypsinogen of this secretion is converted into trypsin by an enterokinase in the duodenum. A large number of experiments has shown that a variety of irritating substances—practically all of which have been injected into the pancreatic duct and the parenchyma, by means of syringe and needle—are capable of causing, in animals, the same lesions that are observed in human cases that come to autopsy. A partial list of such substances include a mixture of bile and sweet oil, gastric juice, duodenal contents, weak solutions of a number of acids such as hydrochloric, nitric, and chromic acids, also alkalies, formalin, and fatty acids. In 1901 Opie (4) demonstrated that injection of bile into the pancreatic duct of an animal would cause necrosis of the organ. In 1906 Flexner (5) showed experimentally that the essential constituent of the bile responsible for the necrosing effect in the pancreas lay in the bile salts, of which the taurocholate was much more active than the glycocholate. Nordman (6) has demonstrated that the injection of infected bile produced lesions far more consistently than did sterile bile. Archibald (7) believes the effect of bacteria in the bile is to precipitate its mucin thereby increasing the concentration of the bile salts.

No attempt will be made to review all the experimentation designed to show the *modus operandi* of acute pancreatic necrosis, but merely to consider those results which seem applicable to the common circumstance of the three cases reported above.

The question may well be asked, Is it possible, with the retching and vomiting that not infrequently accompanies the administration of a general anesthetic, that duodenal contents might have been forced back through the papilla into the duct of Wirsung? Such a theory is maintained by Williams and Bush (8). However, Archibald (7) has demonstrated by experiment that it is impossible to force back duodenal contents through the ampulla of Vater. Using

an iron solution, introduced into a small loop of duodenum closed by ligatures from just above to just below the ampulla, under a pressure of 1,000 millimeters of water maintained for an hour, he demonstrated that none of the solution would enter the common bile duct or the duct of Wirsung. However, in this connection, we should recall the duct of Santorini. Opie (9) studied the pancreatic ducts of 100 specimens and classified their varieties and their relative frequency as follows:

I. Ducts in anastomosis:	
(1) Duct of Wirsung larger:	
(*a*) Duct of Santorini patent	63
(*b*) Duct of Santorini not patent	21
(2) Duct of Santorini larger or equal:	
(*a*) Duct of Wirsung patent	6
(*b*) Duct of Wirsung not patent	0
II. Ducts not in anastomosis:	
(1) Duct of Wirsung larger	5
(2) Duct of Santorini larger	5

Thus, in 11 percent of cases the duct of Santorini was found to be as large as or larger than the duct of Wirsung (fig. 1). The duct of Santorini opens directly into the duodenum without any protective mechanism at its termination, such as the ampulla of Vater has in the spincter of Oddi. Thus, the entrance of duodenal contents into the duct of Santorini may take place under conditions which would be powerless to effect an entrance into the duct of Wirsung.

Is there a possibility in the above cases of a conversion of the common bile duct and the duct of Wirsung into a single continuous channel? The association between cholelithiasis and acute pancreatic necrosis has been known for many years, especially since attention was directed to this subject in 1901 by Opie, in his Diseases of the Pancreas. In our cases pathological examination failed to reveal evidence of a cholecystitis or the presence of gall stones. In considering this question, however, the work of Archibald (7) is again of interest. His attention was attracted to the fact that in about 50 percent of cases of acute pancreatic necrosis operation failed to demonstrate the presence of either cholecystitis or gall stones. He has suggested that in such cases the mechanism by which the 2 ducts are converted into 1 may be the sphincter of Oddi. The following brief account of Oddi's work is given by Archibald:

> Oddi studied this muscle both physiologically and anatomically. In brief, he found that the sphincter in dogs was able to resist a pressure of 50 millimeters of mercury, which equals about 675 millimeters of water. He demonstrated in microscopical sections, that the sphincter was composed of a special bundle of circular fibers. He found that the common duct, outside its course through the bowel wall, possesses no muscular fibers. From the physiological

side, he discovered that this sphincter could be put into spasm by mechanical irritation of the duodenal mucosa, or by the application of dilute hydrochloric acid in either the duodenum or the stomach; and that even mere cutting of the bowel to expose the papilla would cause spasms lasting from 20 to 30 seconds. Stimulation of the vagus apparently provoked a very prompt and intense contraction of the sphincter. A like result was obtained by stimulating the central end of the cut sciatic, while stimulation of the splanchnic has no effect. He also observed dilatation of all the extra-hepatic ducts in dogs

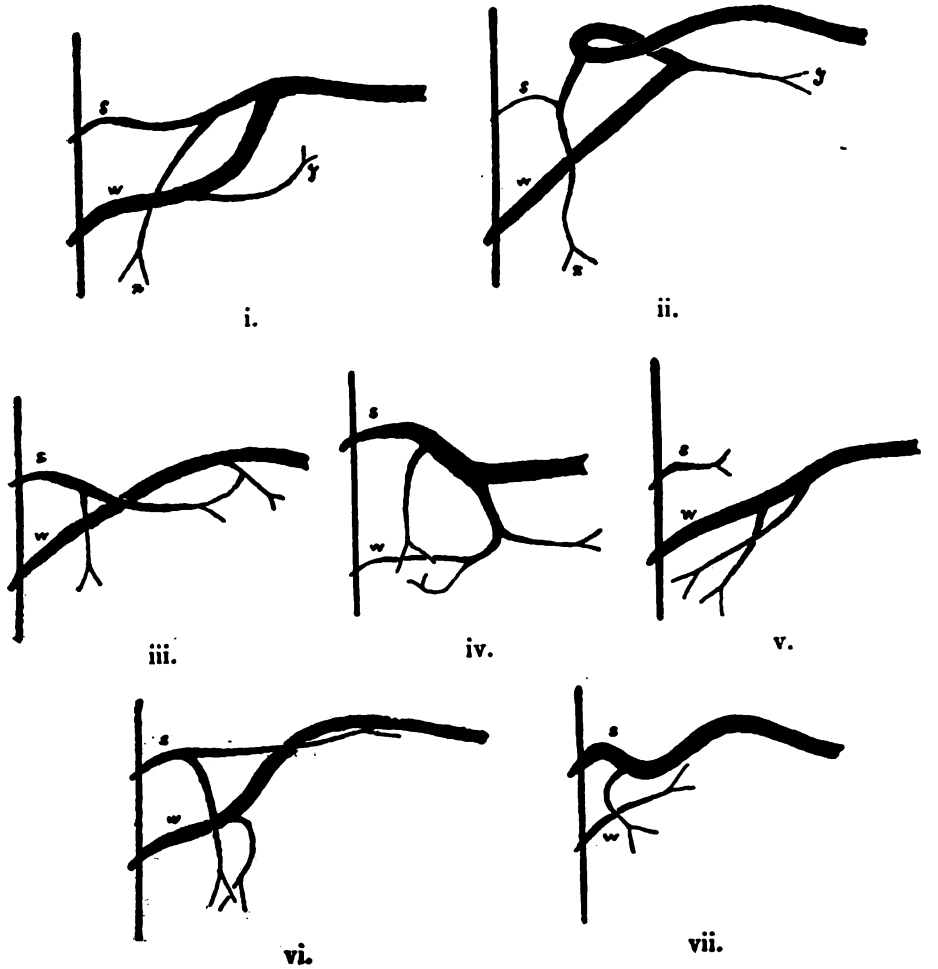

FIGURE 1.—VARYING RELATIONS OF THE DUCT OF SANTORINI (S) TO THE DUCT OF WIRSUNG (W).

(From Diseases of the Pancreas by E. L. Opie, Second edition, J. B. Lippincott Co., 1910.) Attention is particularly directed to specimens IV and VII in which the duct of Santorini is much larger than the duct of Wirsung.

deprived of their gall bladder. He thought a catarrhal condition in the duodenum was a stimulus to the sphincter, and that this might explain some cases of icterus where other causes could not be found.

Before becoming acquainted with the original articles of Oddi, Archibald (10) demonstrated that the hydrostatic pressure in the common duct, that is, the pressure which the sphincter at the ampulla of Vater will oppose to a column of water, was surprisingly

high. The sphincter was rarely overcome by any pressure less than 600 millimeters. After a further series of experiments, Archibald concluded that acute pancreatic necrosis could be brought about entirely through the action of the sphincter of Oddi combined usually with some increase in pressure in the biliary system behind the sphincter.

On the basis of Archibald's work, Mann (11) undertook a series of investigations upon the antomy of the sphincter of Oddi in man. It is evident that if the sphincter is to be able to convert the 2 ducts

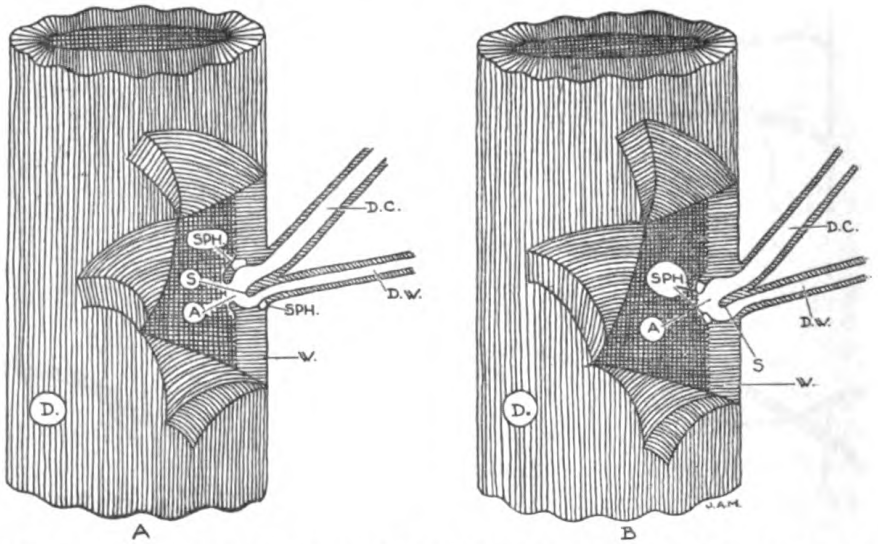

FIGURE 2.—D., DUODENUM; W., DUODENAL WALL; D.C., COMMON BILE DUCT; D.W., DUCT OF WIRSUNG; A., AMPULLA OF VATER; S., SEPTUM; SPH., SPHINCTER OF ODDI.

A. Diagram shows sphincter of Oddi (Sph) situated proximal to the termination of the two ducts. Spasm of the sphincter merely results in compression and narrowing of their lumina.
B. Showing the sphincter of Oddi (Sph) situated distal to the termination of the two ducts, at the outlet of the ampulla of Vater. Contraction of the sphincter will convert the two ducts into one channel. This disposition of the fibres of the sphincter of Oddi is present in a small percentage of humans.

into 1 channel it must lie distal to the entrance of both the ducts into the ampulla; otherwise it would merely compress the lower ends of the 2 ducts. In most instances Mann found that the position of the muscular fibers was proximal to the termination of the common bile duct and that some fibers also passed around the lowest part of the duct of Wirsung. By their contraction, the two ducts would be narrowed. Mann does cite, however, two cases in which that disposition of the sphincter was found which would close only the outlet of the ampulla and so lead to a free communication between the ducts.

Thus, it is seen that three factors are required: (1) The particular disposition of the fibers of the sphincter of Oddi about the outlet of the ampulla of Vater, (2) spasm of the sphincter, (3) increased biliary pressure. Might we not have had these anatomical and physiological conditions favorable for the development of acute pan-

creatic necrosis in this manner present in the above cases? Unfortunately, we do not know the anatomical arrangements of the parts at the termination of the two ducts in these cases. We have not been able to find anything in the literature concerning the effect of anesthetic gases, injected in the duodenum, upon the sphincter of Oddi. However, may it not be reasonably suggested that these patients swallowed enough of the anesthetic gas to irritate the duodenal mucosa sufficiently to cause spasm of the sphincter. Certainly, in case I we do not require such an assumption since there is pathological evidence of duodenitis. Mann (11) has shown that the mechanical effect of the abdominal muscles and of the diaphragm produce marked changes upon the pressure of bile within the common duct. Deep respiratory movements, struggling, retching and especially vomiting, caused the pressure to reach as much as 1,000 millimeters of bile. Such activities are only too common during the administration of a general anesthetic and it may be reasonably inferred that in these cases there was sufficient bile pressure in the common duct to force bile into the duct of Wirsung.

Usually, the immediate cause of death in acute pancreatic necrosis is the absorption of split products from the necrosed tissue of the organ. But death occurred so soon after the beginning of the anesthetic in these cases that hardly enough time elapsed to attain sufficient amount of absorption. The nervous system had already been injured by a varying degree of surgical shock and it seems probable that the added gross insult of such abdominal catastrophe was too much for it to bear.

It is impossible to judge the adequacy of anesthetics and shock as causes of death because their effects cannot be measured in the dead body. The cases in which death is attributed to anesthetics or shock should, therefore, receive a most thorough and detailed pathological examination. The reported cases indicate that the pancreas can be the *raison d'être* in such situations, and should receive the utmost consideration.

Conclusions

1. There is herewith presented three cases of acute pancreatic necrosis occurring during the administration of a general anesthetic.
2. Possible modes of causation are discussed.
3. Only by a careful study of specimens is it possible to determine the anatomical peculiarities which have a physiological significance and constitute an important factor in the production of this condition. In future cases it is recommended that particular attention be paid to the comparative size of the pancreatic ducts, Wirsung and Santorini, and to the relation of the septum separating the com-

mon bile duct and the duct of Wirsung in the ampulla of Vater to the sphincter of Oddi. Also that a culture for organisms be made of the bile in the gall bladder.

4. The following alternative is suggested in those cases in which facilities are not available for a complete anatomical study of these structures, both gross and microscopical: (*a*) Remove gall bladder, common and cystic bile ducts, duodenum, and pancreas intact; (*b*) cross-section pancreas midway between its middle and its tail end. Inject the common pancreatic duct with methylene blue by means of a needle and syringe. First, inject with duct of Wirsung occluded, then inject with duct of Santorini occluded. Inject the common bile duct near its origin. (*c*) Place organs intact in 10 per-cent formalin solution and submit to the Naval Medical School.

5. It is suggestive that the most dangerous stage of anaesthesia for the production of acute pancreatic necrosis is during the second stage of induction and when the patient is coming out. It is then when reverse peristalsis, retching, vomiting, struggling, etc. are most prevalent.

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THE SELECTION OF A RELIABLE, SAFE MYDRIATIC FOR FUNDUS EXAMINATION [1]

By W. D. Horner, lieutenant commander, Medical Corps, United States Naval Reserve

We are in daily need of a suitable mydriatic for examination of the media and fundus of the eye. The term, suitable mydriatic, presupposes certain things. It must be safe, convenient for use, efficient, and of minimum discomfort to the patient.

Safety in a mydriatic is only a relative term. No mydriatic is safe in a person predisposed to glaucomatous attacks. The safest drug is that which dilates the pupil quickest and for the shortest possible time. It should yield to the contrary action of a miotic

[1] From the department of ophthalmology, University of California Medical School.

when desired and the latter should be used routinely in patients over 40 following the completion of the examination.

Convenience includes a reasonable efficiency in that the time of both surgeon and patient should be conserved. It should also interfere as little as possible with the patient's accommodative power.

Before discussing mydriatic drugs, we may, with advantage, briefly review the anatomy and physiology concerned.

The iris is provided with circular and radial musculature through whose alternate contraction the pupil is narrowed or widened. The circular muscle or sphincter of the iris is supplied by parasympathetic postganglionic fibers from the oculomotor nerve, which terminate among the cells of the ciliary ganglion, from whence they pass on as the short ciliary nerves to the sphincter. The nerve fibers to the antagonistic radial muscle or dilator of the iris, arise in the midbrain, pass down the spinal cord, terminating in the lower cervical region. From this point, the path is continued by spinal neurones which leave the cord in the eighth cervical and first and second thoracic spinal nerves and pass by way of corresponding rami-communicantes into the sympathetic chain at the level of the first thoracic ganglion. The fibers then pass upward in the cervical sympathetic to the superior cervical ganglion from which the post-ganglionic sympathetic fibers run through the carotid plexus past the ciliary ganglion as the long ciliary nerves to the muscle.

Stimulation of the sympathetic system results in dilatation of the pupil, possible disturbance of accommodation, slight exophthalmos, widening of the palpebral fissure and in constriction of ocular vessels. Stimulation of the oculomotor mechanism, however, results in contraction of the pupil, spasm of accommodation, and often a decrease in intraocular tension. It is clear that a dilatation of the pupil may be caused either by a contraction of the dilator muscle or inhibition of the sphincter, while in constriction, the effect may be due to a contraction of the sphincter, or an inhibition of the dilator. Under normal conditions, the sphincter and the dilator muscles are kept more or less in tonic activity by impulses received through their respective motor fibers, thus figuratively balancing each other. The sympathetic plays mainly the part of a reciprocal antagonist according to the law that the contraction of one muscle is always accompanied by an inhibition of its antagonist.

Psychic or sensory impulses such as excitement give the balance of power to the dilator fibers. On the other hand, sleep narcosis or central depression cause the pupils to become small through the unopposed oculomotor tone.

Mydriatic drugs used in ophthalmic work are divided into two main classes, namely:

1. Those causing mydriasis, plus cycloplegia, as atropine, hyoscyamine, scopolamine, homatropine, or cocaine.

2. Those causing mydriasis without cycloplegia, such as epinephrine, ephedrine, and euphthalmine.

Of the first group, only homatropine and cocaine will be considered since the others are far too potent to be used in simple ophthalmoscopy.

Homatropine is the second weakest cycloplegic of group 1 and is probably the most widely used mydriatic for fundus work among general medical men. In weak solution, one half to 1 percent, gives a rapid dilatation, which does not last long and which is further shortened by the subsequent use of a miotic. The cycloplegic effect is a disadvantage but is transient. It is the strongest mydriatic which is ever justified for fundus work and, strickly speaking, should be replaced by one of the weaker drugs. In no case should homatropine be used without first determining the intraocular tension at least manually. Should the anterior chamber appear shallow or the cornea small, its use is contraindicated. An acute attack of glaucoma is not infrequently precipitated by the injudicious use of mydriatics. Such a case recently came to our attention.

A woman, about 50 years of age, developed a monocular glaucoma following the use of homatropine for a fundus examination. The physician responsible erred in two ways—firstly, by failing to observe the shallowness of the anterior chamber and secondly, by not instilling eserine or pilocarpine immediately following the examination. While a miotic was used the following day, an unchecked dilatation was allowed to remain overnight.

Homatropine in 2 percent solution gives complete mydriasis in 35 minutes, which disappears completely in 1 to 1½ days. Cycloplegia is maximum in 1 hour and recovery is complete in 1 day. Action of the drug is due to a paralysis of the third nerve endings in the iris and ciliary body.

Cocaine is dispensed as the neutral salts of the alkaloid, the best known of these being the hydrochloride. A 1 percent solution is ordinarily employed for mydriasis, stronger solutions giving an unnecessary anesthetic effect. Due to its dessicating or loosening effect on the epithelium, it enhances the action of other alkaloids with which it may be combined by increasing penetration into the substantia propria. The homatropine cocaine discs used in refraction are an example. The eye should be kept closed during cocaine instillation in order to prevent exfoliation of the corneal epithelium. Mydriasis is not so great as with homatropine, but is complete in 30 minutes and is fully gone within 24 hours. Cycloplegia is weak, manifests itself within one half hour, and is gone in about 2 hours.

Light reaction is retained and the pupil can be contracted promptly by eserine. The action differs from homatropine, in that dilation is due to a stimulation of the sympathetic system.

The second group of drugs is mydriatic in action but does not have any appreciable effect upon the ciliary muscle. These are epinephrine, ephedrine, and euphthalmine.

Epinephrine in stock solution (1:1000) has very little mydriatic effect when dropped into the eye. By prolonged application in which a wick of cotton is saturated and placed beneath the upper lid, fair dilatation and a reduction of intraocular pressure occur.

In concentrated solution, suprarenin, derived from glands or synthesized (if laevorotatory) is a vigorous mydriatic. Two percent solutions of laevosuprarenin synthetic alkaloid, also known as laevoglaukosan or laevosuprarenin synthetic bitartrate (1), used to some extent in the medical treatment of chronic simple and secondary glaucoma cause a marked drop in intraocular tension in addition to a dilatation greater than that produced by atrophine. Their use, therefore, provides a safe dilatation of the pupil in cases of glaucoma where other mydriatics are absolutely contraindicated. Miotics should, however, be used for several hours following the examination.

Ephedrine is an alkaloid, derived from the Chinese plant, ma huang. It is quite soluble in water and is usually dispensed as the hydrochloride. The first pure isolation was by Nagai in 1887.

Ephedrine is a relatively harmless, convenient and efficient mydriatic for Caucasians. It has little influence in Chinese or Negroes (2) (9). Its action is of short duration, 6 to 9 hours, and accommodation is scarcely influenced. The pupillary light reflex is not abolished unless homatropine be added. In nonglaucomatous eyes, the intraocular pressure is not materially increased. Pilocarpine easily overcomes its action. It is useless in refraction and in iritis, but is valuable in ophthalmoscopy. A combination of ephedrine and homatropine was put out by a well-known drug company under the proprietary name of mydrin about 1894. This consisted of ephedrine 100 parts and homatropine 1 part and was used in a 10 percent solution.

In 1895, Groenouw in Germany (3), reported 100 cases which he had studied after such an ephedrine-homatropine solution. He obtained a mydriasis beginning in 8½ minutes, a maximum in 34 minutes, and a duration of 4 to 6 hours. The pupil at maximum dilatation was 5 to 6 millimeters wide. Accommodation was not interfered with.

This work was reviewed by Suker (4) in 1895 and Stephenson (5) in 1898. All of these men enthusiastically advocated the use of the

mixture for fundus work, as it was rapid, did not affect accommodation, was relatively short in duration and apparently harmless. Its cost at that time was too high for routine work, however. A substitute for mydrin was offered by Groenouw (3) consisting of ephedrine 5 percent solution containing five hundredths of 1 percent homatropine. This is essentially half strength mydrin. Groenouw claimed its action was similar though slower. The onset was the same (8½ minutes), full mydrasis occurring within 40 minutes, which maximum continued for 20 minutes, and was completely gone in 3½ hours. This solution does not deteriorate rapidly, remaining active for more than 3 months.

We have used the stronger solution, consisting of homatropine 0.01 gram, ephedrine 1 gram, and distilled water 10 grams, as a mydriatic for fundus work in both office and clinic practice for the past year with eminent satisfaction. We find it superior to euphthalmine formerly employed, in that it accomplishes its purpose more rapidly and disappears more quickly. The weaker (Groenouw) dilution also performs well.

Euphthalmine, generally prescribed as the hydrochloride, is a colorless crystalline powder derived from beta eucaine, which is a proprietary remedy chemically allied to cocaine.

Euphthalmine has long been a favorite among ophthalmologists. Two drops of a 5 percent solution produce complete dilatation of the pupil with very little effect on the ciliary muscle in 60 minutes. This lasts about 2 hours with a return to the normal size in 24 hours. Jackson (6) advocated its use combined in equal parts (2 percent) with cocaine. This combination produces, according to Wood (7) a most satisfactory mydriasis, in from 15 to 30 minutes. Its use is free from pain, vascular injection, appreciable rise of intraocular tension and corneal irritaton. The usual precautions for cocaine are required. Chen and Poth (8) (9) found that euphthalmine showed even less tendency to produce rise of intraocular tension than ephedrine, although ephedrine had less effect on accommodation. They also experimented with a mixture of euphthalmine and ephedrine. This gave a greater pupillary dilatation by one half to 1 millimeter than singly but the mydriasis acted longer than ephedrine alone.

Summary

Summarizing the virtues of the foregoing drugs for fundus work, and with due regard for the qualities of safety, reliability, convenience, speed of effect and recovery, I would rate the mydriatics discussed as follows:

1. An ephedrine-homatropine mixture, such as homatropine hydrochloride 0.01 gram, ephedrine hydrochloride 1 gram, and distilled water 10 grams or the 50 percent weaker combination of Groenouw.

2. A cocaine-euphthalmine combination in equal parts as advocated by Jackson and Wood, using 2 percent solution.
3. Eupthalmine alone in 3 to 5 percent solution.
4. Ephedrine alone in from 3 to 5 percent solution.
5. Homatropine alone in 1 percent solution.
6. Cocaine solution in 1 to 2 percent, and lastly
7. Concentrated epinephrine solutions (in exceptional cases).

In highly pigmented people, I would particularly advocate the use of homatropine in combination or alone.

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JOINT TRAINING OF ARMY AND NAVY MEDICAL RESERVE

By JOHN R. HALL, major, Medical Corps, United States Army, and REUBEN H. HUNT, lieutenant commander, Medical Corps, United States Navy

Like many other worthwhile things, military training of Medical Department reserve officers on an inactive duty status, in combination with postgraduate clinical instruction at established medical centers, was born of necessity. Of the factors which called for this new method of training two stand out as most important: One, that the funds appropriated for active duty training were never sufficient to permit ordering to active duty all those who wanted it; two, there were many who were deeply interested in the reserve movement and desired such training as they would get on an active duty status, yet for good and sufficient reasons were not able to accept active duty in the summer camps even when it was available. Out of these needs grew the method of training which has now become known as the "Skinner plan" due to the interest shown and personal attention given to it by Col. George A. Skinner, Medical Corps, surgeon, Seventh Corps Area.

The necessity of postgraduate study for those in the medical profession is well understood both by the doctor and the public in general. This applies, under modern conditions, to the dentist and veterinarian as well as to the internist and surgeon. So true is this that it appears trite to state it here.

As equally true, though not so well appreciated by the rank and file, is the importance of some military knowledge for the performance of one's duty in our scheme of national defense. The Medical Department reserve officer should have a comprehensive yet balanced picture of the fundamentals of the military service so that he may be able to see the problems which will confront him on reporting for active duty in time of an emergency and study them with some degree of discernment and understanding of the relationship of one problem to another. Thus he will have insight and resourcefulness in seeing the things, new to him, that need to be done.

This method of training fulfills these two requirements in a most acceptable manner in that the student officer gets two weeks of excellent postgraduate work, without fee and without any greater loss of time from his practice than would pertain for a similar period of postgraduate work anywhere. There is an additional advantage in that the clinical material is arranged and presented so that he may get a maximum amount of instruction during this two weeks.

The cost to him of such clinical work is only that incurred for the necessary travel and incidentals while at the place where the course is being given. At the same time he gets instruction along military lines with special reference to the part played by the Medical Department; this gives him a background for the study of his problems in their entirety, enables him to state them with clarity, and to solve them satisfactorily.

This method of training was first put into effect at the Mayo Clinic in 1929 and was so successful that it has been extended. Reports of such courses in various parts of the country are now appearing in the literature on the reserve corps.

The purpose of this paper is to set forth an account of the successful course that was held during February at the Washington University in St. Louis. This was the second course to be conducted there under the direct supervision of Colonel Skinner, assisted this year by Lt. Comdr. James A. Fields, Medical Corps, United States Navy, Maj. James E. Phillips, Medical Corps, United States Army, Maj. Clement J. Gaynor, Dental Corps, United States Army, and the writers.

The program, which was prepared by Col. W. Lee Hart, Medical Corps, United States Army, medical inspector, Seventh Corps Area, is of especial interest in view of the two fundamental innovations he embodied.

One was the inclusion of the Navy and the consideration of the Medical Department activities of the two services, both separately and in joint operations. This was a definite advance in the preparation of the medical personnel of the country for its responsibilities in national defense. It is obvious to all thinking people that any future emergency in which our country may be involved will result necessarily in joint operations, and that it is of paramount importance that there should be the greatest possible understanding not only between the two services but especially between the two medical departments. Not only should the reserve officer of the Army have some picture of the Navy medical service but likewise the reserve officer of the Navy should have a similar picture of the Army medical service.

Enthusiastic cooperation was shown by the line of the Navy in this course, as was demonstrated by the personal attention and time that Commander Blankenship and other naval officers gave to it.

The second important innovation was the handling of topics on key subjects by specially selected men. Heretofore it has been the policy to furnish selected reserve officers, generally those in the junior grades, with source material from which they gave the lecture on the topic assigned. Here we selected preeminent authorities who lectured from the wealth of their own experience and research.

The following titles selected from among the many interesting subjects presented during the military part of the day indicate the scope of the military instruction:

Military History, with special reference to the development of our military policy.

The Organization of the Government of the United States with particular reference to the State, War, and Navy Departments.

The work of the Assistant Secretary of War in Procurement Planning.

The Organization of the Army of the United States.

Joint Medical Service Incident to an Overseas Expedition.

Military Preventive Medicine.

The lectures in the lyceum part of the course were of absorbing interest and in each case found the audience reluctant to depart. Illustrative of the subjects considered, the following are mentioned:

Serum Therapy and Its Accidents.

Medical Aspects of Submarine Work.

Biology of the Human Person.

Navigation.

South Sea Islands.

Intelligence Tests.

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Originally the course had but two parts, the clinical and military, using the morning and afternoon hours and leaving the evening hours free for the men. Many of them were at a loss as to how to spend their evenings, and impromptu lectures and discussions at night were a natural development to fill this time. These have been perfected in the present lyceum section of the day, which as each course brings improvements becomes more and more interesting and inspiring.

So here each day of the course was divided into three parts. The first part, the morning hours, was given over to clinics. This series of clinics was arranged by the faculty of the Washington University; they were wide in their scope and thorough in their presentation, making an excellent postgraduate course for the Medical Department officer. Special notation should be made of the clinics demonstrating the latest work in dentistry which were put on by the dental faculty. Such a course amply recompensed the reserve officers for their time and for the cost of attending the course. Also it fulfilled the requirement that where the reserve officer accepts training on an inactive duty status he must be given definitely some new ideas which he can carry back to his practice and which will enable him to function better in his profession.

The afternoon hours, the second part of the day, were given to the consideration of military subjects, an effort being made to sketch a background upon which the various reserve officers could predicate their actions were they called into active service in time of an emergency. The lectures were held along broad lines and each subject was handled by one thoroughly competent to speak authoritatively on it.

The evening sessions, the third part of the day, were given over to a lyceum course in which an effort was made to present lectures of high cultural value and interest. Also during the evening hours interesting map problems were worked out, lantern slides being used to show the tactical movements and disposition. This feature served well to make plain the principles involved. Perhaps the most interesting and instructive map problem was that of the medical service incident to joint operations in a forced landing where the ambulance ship was used as an off-shore evacuation hospital covering the landing operations.

The student officers were the guests of the city of St. Louis in a tour of inspection of its medical social service institutions, water works, and sewage disposal plants. Under the guidance of the city commissioner of health, First Lt. Curtis H. Lohr, Medical Reserve, United States Army, who was the active host of the day, these various public health facilities were visited and the working of each was demonstrated. Too much credit cannot be given to those

who arranged and participated in this interesting demonstration. The entire trip was so well arranged and correlated that the group went from one interesting feature to another without loss of time and without diminution of enthusiastic interest.

A few of the highlights of the tour may be mentioned. The student officers were guests of the city at a most enjoyable lunch given at the St. Louis Training School. At the water purification plant at Howard Bend on the Missouri River the engineer in charge described how by the most modern methods the water from the Missouri River is converted into a safe water supply for the city. At the Isolation Hospital the method of segregating contagious disease patients was demonstrated. Here an intensely interesting symposium on typhoid fever was given by the staff.

The student officers were also guests of the St. Louis Medical Society at its monthly meeting held in the society's commodious building. The medical officers of the service participated in the addresses, Colonel Skinner being the guest speaker.

The record of the course would not be complete without expressions of appreciation to Chancellor George Reeves Throop, Ph.D., Washington University, Prof. Roland G. Usher, Prof. C. E. Cullen, Dean Alphonse M. Schwitalla, S.J., Ph.D., Prof. Edward S. West, and Prof. John P. Nafe, for their wonderfully interesting and instructive lectures.

The Medical Corps Reserve owes to Dean W. McKim Marriott and the faculty of the medical school of Washington University its keen gratitude for making this course available.

The enthusiastic expressions of the value of the course by those who attended indicate the recognition of the merits of this tried and successful method of training.

THE TREATMENT OF SUPRACONDYLAR FRACTURES OF THE HUMERUS[1]

By DENIS S. O'CONNOR, M.D., F.A.C.S., lieutenant, Medical Corps, United States Naval Reserve

A fracture is a traumatic lesion of a bone due to a stress greater than a particular part of a bone is designed to bear. Fractures occur in certain bones, or parts of certain bones, with a frequency proportional to a frequency of certain violent postural strains imposed upon them. For example, fracture of the lower end of the radius is very common because, in falling, the palm of the hand is used to cushion the fall and a hyperextension of the hand brings a greater strain on the lower end of the radius than it can stand. The damage resulting varies from a faintly visible transverse fracture line, through a point within the distal inch of the bone, to a

[1] From Yale University, school of medicine.

complete separation of the fragments and a backward displacement and rotation of the distal fragment.

The gradual dissemination of the knowledge of the mechanism in the production of a fracture of the distal end of the radius has resulted in a better understanding of the logical mechanism for replacement of the fragments and consequently better functional end results.

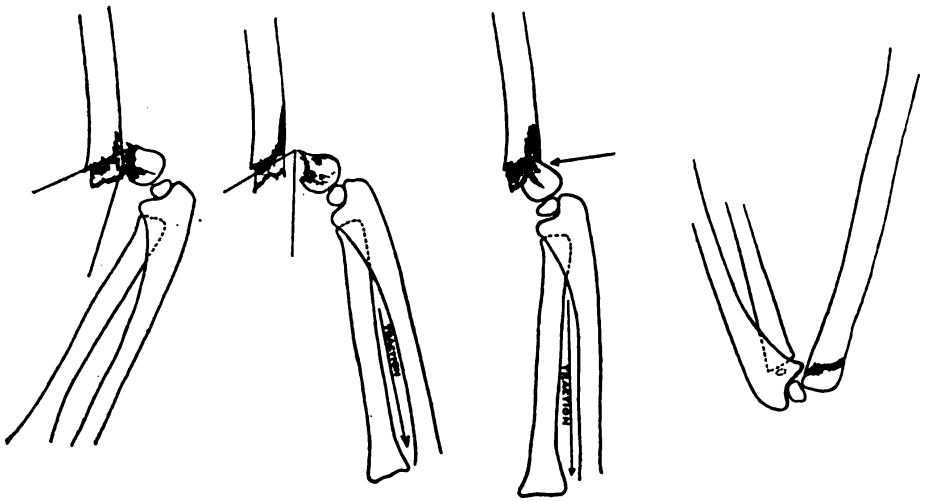
Fracture of the distal end of the humerus is analogous to fracture of the lower end of the radius in the respects that the fracture is commonly due to a violent strain on the distal end of the bone secondary to hyperextension of the contiguous joint, the elbow, and that the displacement of the distal fragment is backward, as is also the rotation. If the analogy were carried to the treatment of supracondylar fractures of the humerus, the results, now frequently marked by flexion deformity at the elbow, limited range of motion, and Volkman's contracture of the hand, would be correspondingly good.

Fractures of the lower end of the humerus are very common in children, uncommon in adults. Why this is so is clear, when it is realized that in childhood the range of motion in the elbow includes a definite degree of hyperextension which is only uncommonly present in adults. Hyperextensibility of the elbow is apparently a prerequisite to the production of the type of fracture under consideration.

The fracture is usually transverse in type but may be converted into a T-shaped fracture by a fracture line running down between the condyles into the elbow joint. This complication does not void the essential principles underlying the understanding of fracture of the lower end of the humerus. While most supracondylar fractures of the humerus are transverse, oblique fractures are not uncommon. If the fracture line is oblique, the principles of reduction are the same as if the fracture were transverse, but the difficulty of maintaining the position is greater under these conditions. Once reduction of the fracture is obtained, the position should not be disturbed for at least a week, at the end of which time sufficient consolidation of the tissues will have taken place so that the danger of loss of position will be minimized.

Supracondylar fractures of the humerus are accompanied or complicated by rather marked subcutaneous hemorrhage which spreads through the soft tissues about the elbow joint until the skin of the lower part of the arm and the upper part of the forearm is under considerable tension. In this condition, if the elbow is flexed even slightly, the constricting effect of the skin about the elbow joint is increased and, if not watched, may impair the main arterial blood supply to the forearm and hand with resulting ischemic paralysis or Volkman's contracture of the hand.

The treatment of supracondylar fractures of the humerus cannot be begun too early. The first step in treatment is to decrease the swelling about the elbow by careful and patient effleurage massage of the extremity, beginning proximal to the most proximal evidence of swelling and gradually working distal to the obvious swelling. The direction of stroking in the massage is always in the direction of the return circulation. Sometimes, this massage must be supplemented by elevation of the extremity and by the judicious application of moist heat. Until one has developed proficiency in the treatment of this type of fracture, it would be well to use all three methods of reducing the swelling.



OUTLINE DRAWING OF BONES ABOUT ELBOW FROM LATERAL VIEW OF ROENTGENOGRAM.

The first drawing represents the fracture before any attempt has been made to reduce it. It will be noted that the lower fragment is displaced backward and upward and that the articular surface of the distal fragment is rotated backwards.
The second drawing represents the fracture after traction on the forearm and hyperextension of the elbow has been done. This represents the first step in the reduction of this type of fracture. Note the gaping between the proximal fragment and the anterior margin of the distal fracture surface.
The third drawing represents the fracture after the distal fragment has been pushed forward into the plane of the proximal fragment.
The fourth drawing represents the fracture completely reduced after the forearm has been acutely flexed allowing the fracture surfaces to engage with each other. (O'Connor).

The damage to the soft tissues which occurs at the time of the original injury and the importance of the soft tissue damage in the result makes it advisable to reduce the fracture with a minimum of further trauma to the soft tissues.

Under general anaesthesia, preferable in children because of the fear of pain and the necessity of obtaining complete relaxation, extension is made on the forearm by traction at the wrist, the direction of the traction being gradually carried into an abnormal hyperextension of the elbow to permit the anterior margin of the distal fracture surface to gape and to engage the fracture surface of the proximal fragment (see accompanying figure). Engagement of the fracture surfaces on each other is absolutely necessary to the reduc-

tion. Once reduction of the fracture surfaces is accomplished, acute flexion of the elbow is possible without the use of force, and the maneuver of flexion firmly fixes the fragments together so that further displacement is unlikely.

After reduction of the fracture and the fixation of the elbow in acute flexion by means of an adhesive plaster dressing, the radial pulse must be watched for at least 12 hours and, if the arterial circulation shows signs of embarrassment, so much of the flexion in the elbow must be temporarily sacrificed as will permit free arterial circulation. No trouble from the circulation will be encountered, however, if the massage is properly done before the reduction of the fracture and if unreasonable force is not used in the reduction.

Giant bleb formation on the skin, due to blocking of the lymphatic circulation by the skin tension, and a common complication in fractures about the elbow joint, will seldom occur if supracondylar fractures of the humerus are treated early in the method described above.

A supracondylar fracture of the humerus seldom gives anything but a perfect functional result if accurately reduced and the circulation maintained. The length of treatment and disability may be materially shortened by skillful aftertreatment.

Within 24 hours after reduction, the adhesive supports are released, except in the oblique fractures, without disturbance of the position of the elbow. The forearm is then permitted to go into that degree of extension to which it will go, solely by the force of gravity acting on the forearm. Careful effleurage massage to the arm and forearm, with the patient in a recumbent position, using the same technique as that used to reduce the subcutaneous hemorrhage before the reduction of the fracture, is given. During the massage, the elbow will go into some degree of extension but should go back into acute flexion without the use of force and unaccompanied by pain. After this treatment, the forearm is fixed in acute flexion again by an adhesive plaster dressing.

After 14 to 20 days, the forearm may be supported in a sling in moderate flexion, in place of the acute flexion maintained by adhesive plaster. The inevitable active motion of the elbow, while supported by the sling, and the active assisted motion of the elbow within the limits of pain at daily treatments invariably give rapidly increasing range of motion to the ultimate complete range of motion within 6 weeks.

The absence of pain in the treatment is indicative of the normal reposition of the fractured parts, freedom from trauma, and the normal reparation of the tissues.

ROENTGENOGRAPHY OF THE NASAL ACCESSORY SINUSES

A Simple Device Producing Accurate Exposures With Standard Equipment

By W. A. Fort, lieutenant commander, Medical Corps, United States Navy

The radiographic examination of the nasal accessory sinuses requires extremely accurate exposures. Interpretation of faulty negatives should never be attempted. The chief difficulty of sinus technique is the directing of the central beam in the exact sagittal plane of the skull. Simultaneously the central beam must be directed in a plane forming precisely the desired angle with the plane of the base. To require any less of these exposures is to sacrifice accurate shadow values and invite errors of interpretation. Unfortunately such errors are more often attributed to the limitations of radiography than to their usual source, faulty technique.

While not as important, there are many other desirable features to be sought in sinus films. Exposures made in the upright position will frequently show fluid levels of exudative pathology, especially in the antra. When opaque oils are injected, upright exposures are indispensable. Maximum contrast and detail must be obtained to detect the shadows of polyps and other chronic changes in the mucus membrane of these bony cavities. The technique must be adapted to exact duplication if comparative films are to register slight changes in density such as those resulting from congestion of temporary occlusion.

To obtain such films requires the aid of the Potter-Bucky diaphragm to minimize scattered radiation originating in the skull. Interposing a small diaphragm between the X-ray tube and the skull helps to eliminate radiation from points in the tube outside the focal spot. A small focal spot increases detail as does also a long target film distance. The head must be held rigid in a comfortable position and prolonged exposures must be avoided if motion is to be eliminated. No apparatus will successfully eliminate tremors. These frequently occur when the patient is uncomfortable or the required suspension of respiration is continued longer than a few seconds.

Any radiographic technique is a compromise of technical factors and sinus exposures are no exception. Using new double intensifying screens, of the type furnished naval activities, good sinus exposures may be obtained in the average male adult Caucasian skull with a radiator Coolidge tube drawing 30 milliamperes at a 30-inch target film distance. Using the Bucky diaphragm with modern mechanical rectification, 72 kilovolts peak for the baseline exposure and 82 kilovolts peak for the 23° exposure, will give good film contrast in 7 seconds time. The best results are obtained by varying the

tube voltage about 4 kilovolts peak above or below the average according to estimated skull thickness. The average tube voltage, of course, will vary somewhat with different X-ray equipment.

It is not necessary to compromise with the most important technical factor, i.e., the proper direction of the central beam. Many types of apparatus have been designed which accomplish this fairly well. Most of them are too complicated and expensive to justify their purchase except for the larger hospitals. The following device is inexpensive and is designed to be used with a common radiographic table of the tilt type installed in several naval hospitals and dispensaries.

Apparatus

The device in use at the naval hospital, Canacao, P.I., is made of monel metal and consists of two flat strips carrying perpendicular posts to which are clamped horizontal rods engaging the ears of the patient.

The flat strips are clamped to the table top in the grooves intended for the foot or head rest. The clamping is accomplished by $\frac{3}{16}$-inch threaded bolts equipped with lugs (to fit the table slots) and butterfly nuts (fig. 1B). Two bolts, with centers 20 inches apart, are sufficient for each strip. These strips measure 46 inches in length, 1½ inches in width, and are three sixteenths inch thick. A barely perceptible bend is placed in each strip about 15 inches from the perpendicular post. This eliminates the spring of the metal when the concave side is bolted to the table. A few turns of adhesive plaster around the free end prevent scratching the table top and assist in lining up the ear clamps. This metal strip should have no twist lest the upright post be thrown out of the perpendicular.

The perpendicular post is welded in the center line of each strip at a point 2 inches from the free end. It consists of a rod one half inch in diameter and 6½ inches in length. The measuring scale of an ordinary foot ruler is marked on one side (fig. 1). A narrow longitudinal groove is cut on the opposite side to receive the sliding pin of the double clamp.

The ear piece consists of a rod sliding horizontally at a right angle to the perpendicular post with a ball fitted at one end. This ball measures three eighths inch in diameter and is covered with gauze held in place by rubber bands which provides a clean ear piece for each patient. Sufficient gauze is added to make a snug fit for the external auditory meatus. The sliding rod is one fourth inch in diameter and 10 inches in length. It carries a measuring scale marked on its surface.

The double clamp serves to hold the perpendicular and horizontal rods together in the desired adjustment. It is fitted with two thumb

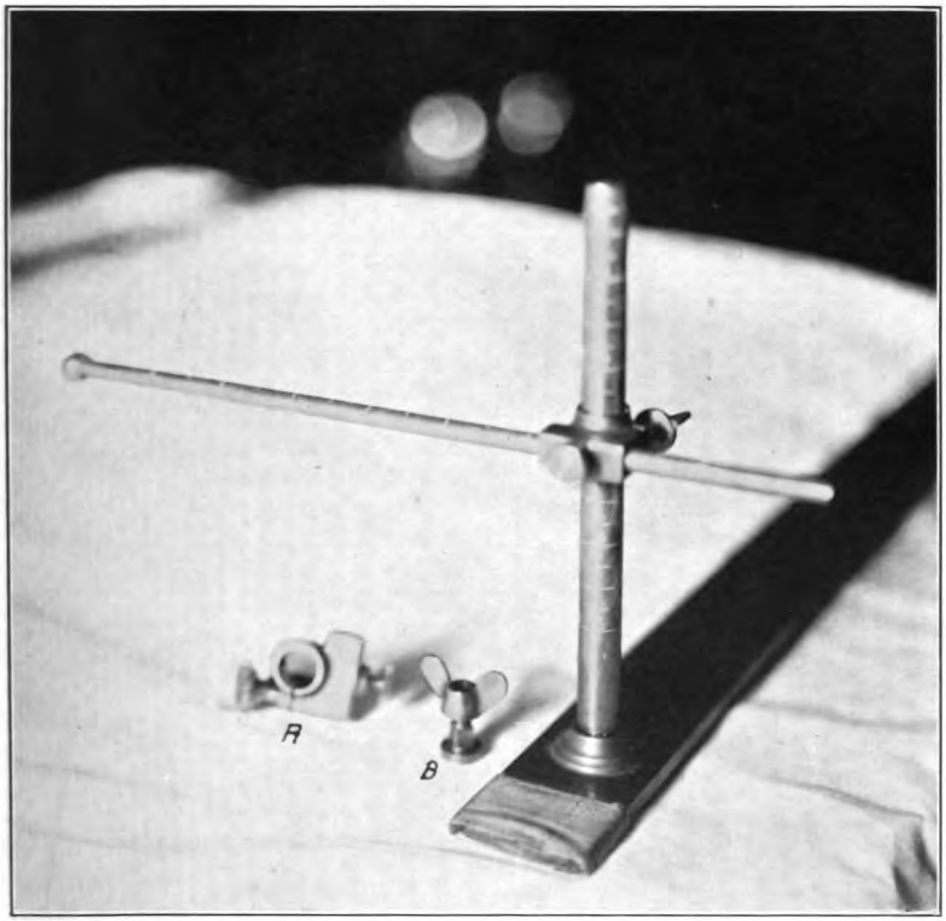


FIGURE 1.—LEFT SIDE OF HEAD-HOLDING DEVICE.

A, Double clamps shown again in the assembled parts. *B*, Bolt with butterfly nut used for clamping lower ends of flat strips to table top.

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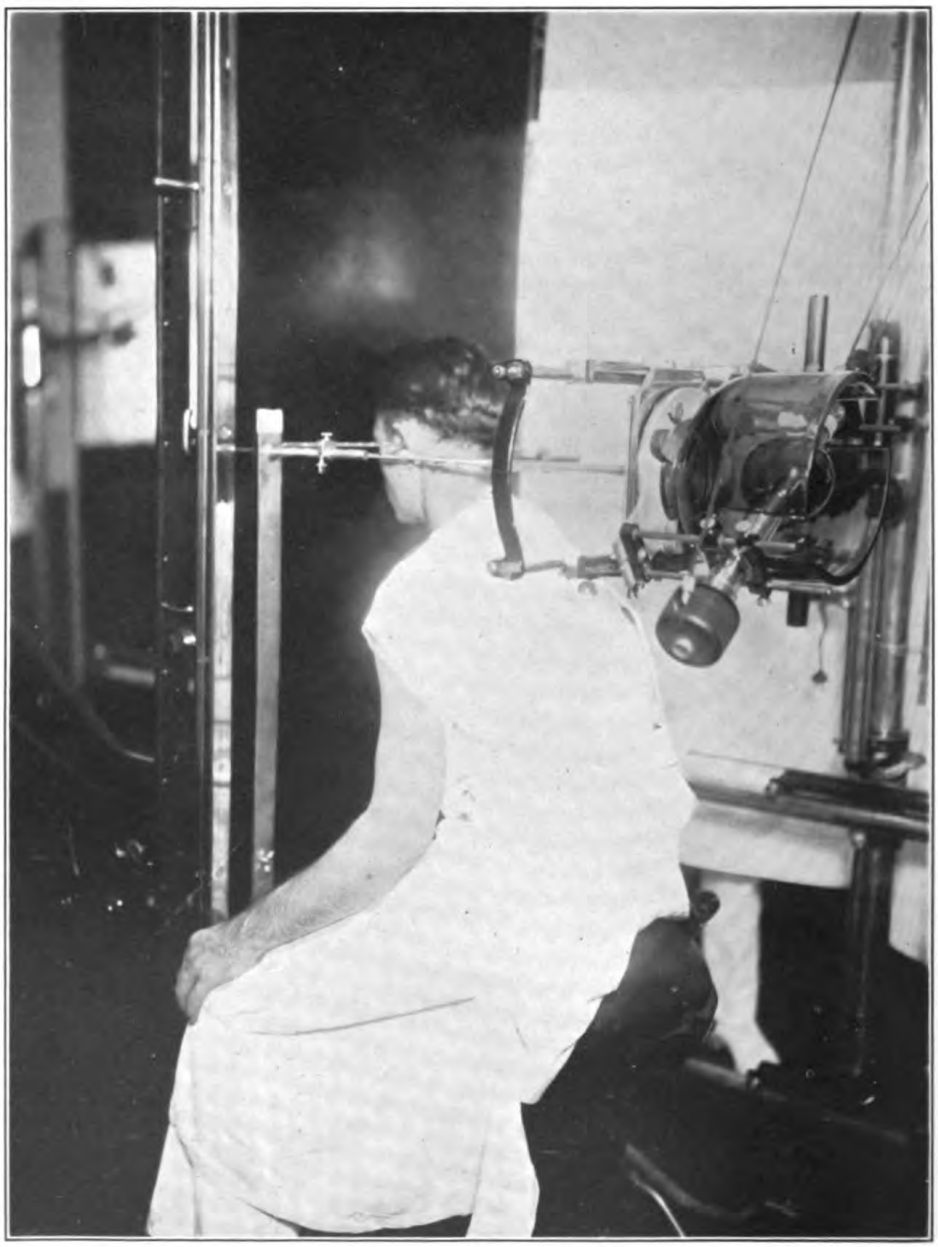


FIGURE 2.—THE HEAD HAS BEEN FIXED IN THE EXACT SAGITTAL PLANE; THE POINTER INDICATES THAT THE CENTRAL BEAM WILL PASS PARALLEL TO THE BASE LINE AND ONE HALF INCH BELOW IT.

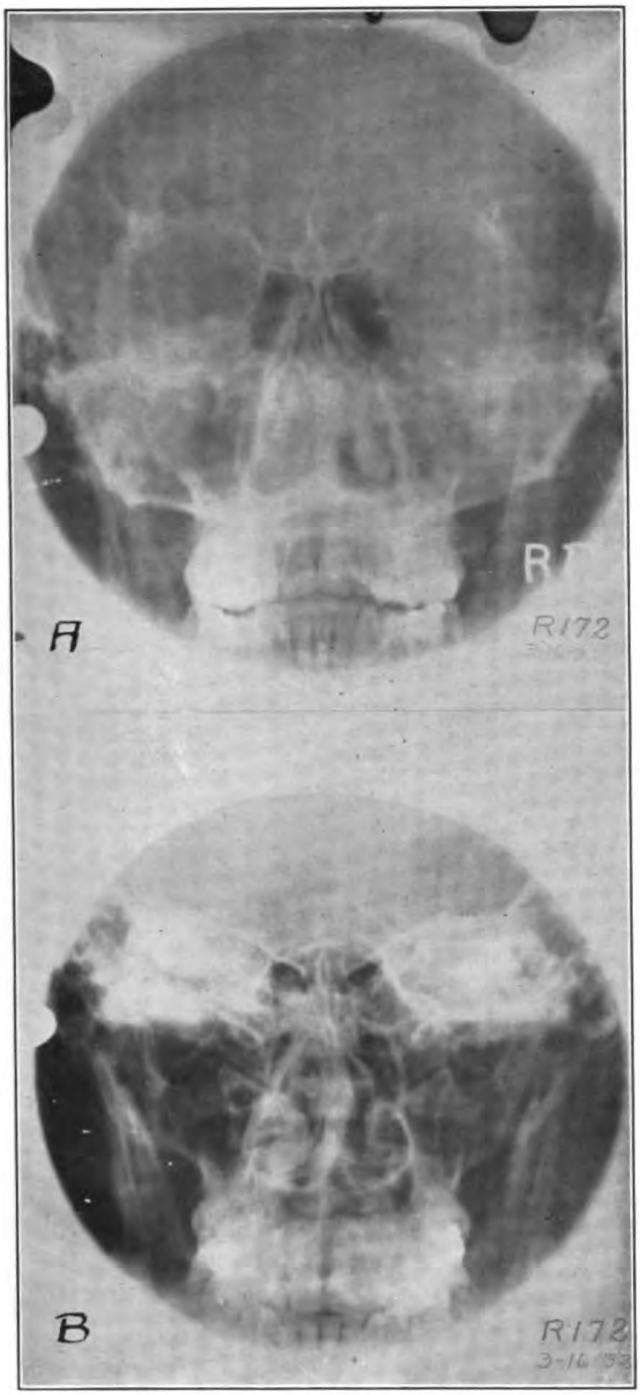


FIGURE 3.—A. 23 DEGREE EXPOSURE. B. BASE LINE EXPOSURE. SUFFICIENT BONY LANDMARKS ARE INCLUDED TO DETECT THE SLIGHTEST ROTATION OF THE HEAD. PRESERVING THE CONE SHADOW MAKES IT EASY TO DETECT IMPROPER CENTERING.

screws for this purpose (fig. 1A). A small pin, referred to above, prevents the turning of the clamp around the perpendicular post.

An old dental chair, from which the foot and back rests have been removed, completes the special equipment. This chair is placed at the foot of the table. When the table is tilted upright for sinus exposures, the chair (with the base properly prepared) slides easily into position. An ordinary operating stool may be used but is not as easily adjusted to the desired height with patient seated.

PROCEDURE

The method of procedure is as follows:

1. Clamp the long, flat metal strips to the table top and secure the double clamps at the 3¾-inch mark on each perpendicular post.
2. Assemble the horizontal rods and see that the ear pieces meet in the midline of the table. Draw ear pieces apart to the 4-inch mark on each side.
3. Place a loaded 8 by 10 cassette under the center of the Bucky diaphragm and clamp in position. It may be necessary to place a flat wooden block in the tray to hold the weight of the cassette when the table is raised.
4. Raise the table to the exact perpendicular and slide dental chair into position.
5. Set the tube stand at 30-inch Bucky distance by mark on side rail. Tube target must be in center of protecting lead bowl.
6. Tilt tube to 90 degrees and center on the midline of table top with central beam indicator. (The centering will not be the same as that for horizontal work unless the tube stand is very rigid.)
7. Remove central beam indicator and seat patient on dental chair with his hands resting on his knees. The nose and forehead should touch the midline of the table top. Adjust height of chair and patient's posture until the ears are approximately opposite the ear pieces. (This step will be facilitated if the ear pieces have previously been clamped as directed above.)
8. Again adjust the head in the nose forehead position. The tip of the nose should be slightly flattened against the table. By fine adjustments of chair height, head rotation and double clamps, the head is gradually worked into exact position where it is held by the ear pieces.
9. Check readings of marks on horizontal rods and perpendicular posts, comparing right with left. When the readings are exactly duplicated, the head is in the correct position. In other words, the sagittal plane of the skull is perpendicular to the film (and table top) which it bisects. (The ear pieces must fit snugly and yet not be painful. The patient is more comfortable in the correct position

and will not attempt to twist his head unless he is roughly handled. Care must be exercised, especially when changing casettes, that jarring is avoided. Such vibrations are transmitted through the ear pieces to the bony auditory canal and are very unpleasant.)

10. Draw a straight line with a skin pencil and ruler connecting the outer canthus of the eye with the external auditory canal. This represents the plane of the base of the skull and is referred to as the base line.

11. Replace the central beam indicator and adjust the central beam so that it passes parallel to the base line and one half inch below it. This step is simplified by sliding the tube on the horizontal bars until the extended pointer clears the side of the face (fig. 2). At this time it is convenient to raise or lower the Bucky diaphragm until its center is on a level with the pointer. Remove the pointer and slide the tube back to its correct central position.

12. Apply compression band over back of neck if patient does not cooperate.

13. Set Bucky diaphragm, or other timing device, at the desired exposure time (about 7 seconds) and place cone in position. (Good results are obtained by using the no. 1 cone furnished with the table. If a smaller diaphragm, measuring 1½ inches in diameter, is substituted for that attached to this cone, the quality of the negative is improved. The long cylindrical cone may be used if desired, but valuable bony landmarks will be sacrificed in the negative.)

14. Make the base line exposure. (The exposure technique given above may be used as a guide. Avoid overpenetration if contrast is desired. This position gives a clear symmetrical projection of the antra. The overshadowing by the spine is easily recognized. (See fig. 3B.))

15. Tilt tube downward 23° from its base-line position. (Since there is considerable variation in the topography of different skulls, the base line is not always exactly perpendicular to the tabletop when the nose and forehead are in contact therewith. Hence the reading on the tube tilt indicator may vary above or below the 90° mark. If 23° is subtracted from the angle of the base-line exposure, the result will be the correct angle for the 23° exposure.)

16. Raise the tube 9 inches above its base-line position. (With 30-inch target film distance, raising the tube 9 inches and tilting it 23° downward direct the central beam through the midpoint of the bridge of the nose. This assures a symmetrical exposure of all the nasal accessory sinuses.)

17. Change cassette and lower Bucky diaphragm 2 inches. (This procedure brings the center of the cassette into line with the central beam.)

18. Make the 23° exposure. (The exposure factors are the same as those for the base-line exposure except for a raise of 10 kilovolts in penetration. This angle projects the shadows of the petrous portions of the temporal bones over those of the antra and gives clear shadows of the frontal locules and ethmoidal cells. (See fig. 3A.))

19. Make the lateral exposure. (No special instruction is necessary for the lateral exposure other than that given in standard techniques. It is made in the upright position after moving the ear clamps out of the way. The head is secured in the true lateral position by a compression band and a padded block which fits the curve of the mandible.)

Modifications.—The above technique is easily modified for stereoscopic views. When these are used it is important to shift the tube in the sagittal plane. The Waters or nose chin position is easily obtained. The technique for oblique exposures of the ethmoids can be followed by placing one earpiece lower than the other. These ear clamps can also be used to hold the head for exposures of the base and occiput. With tables so arranged that clamps would not interfere with the Bucky diaphragm or compression band, the device could be modified to clamp on the sides of the table. This would eliminate the long flat metal strips used as supports for the perpendicular posts.

Summary

1. The importance of true sagittal exposures for accurate interpretation of the shadows of the nasal accessory sinuses is emphasized.

2. The construction and operation of a device to produce true sagittal exposures in the upright position at exact angles is described. Other desirable film qualities are obtained with this technique.

3. This device is inexpensive since it utilizes the Bucky diaphragm and tube stand of an ordinary radiographic table.

4. By standardized procedure, the technique has been made practically mechanical and produces excellent results in the hands of technicians with limited experience.

CLINICAL NOTES

COARCTATION OF THE AORTA

WITH REPORT OF CASE

By R. R. GASSER, lieutenant commander, Medical Corps, United States Navy

Coarctation of the aorta is a congenital defect characterized by a localized narrowing of the aorta of greater or lesser degree at or near the insertion of the ductus arteriosus, which may or may not remain patent. Two types of coarctation are recognized, e.g., the infantile and the adult. This paper will deal only with the adult type.

As to the cause of this defect two theories are advanced. First, that the adult type is due to an extension after birth into the wall of the aorta of the obliterative endarteritis by which closure of the ductus is accomplished or by traction of the ductus, or by both. Second, that the adult type had developed in utero before the end of the second month of gestation, by which time the primitive arches have assumed their final form and the right dorsal aorta should have disappeared, the closure or constriction of the aortic lumen representing a primary arrest in development. This second theory being substantiated by Blackford's case 12, which showed obliteration of the aortic lumen at the insertion of the ligamentum arteriosum and from the costo-cervical trunk arose a tortuous artery 14 millimeters in circumference and 7 millimeters long which passed behind the esophagus to open into the aorta just above the origin of the third intercostal artery by a bulbous aneurysmal dilatation.

All grades of coarctation are found, from that so slight it is scarcely seen at post mortem to complete aortic obliteration. Coarctation of the adult type is three times more frequent in the male than the female.

Coarctation is essentially a condition of childhood, but Abbott's analysis shows a maximum age at death of 92 years and a minimum of 33 months, with a mean age of 33 years.

Associated with coarctation may be other congenital abnormalities. Abbott in a series of 200 collected cases with autopsy found 47 of complete atresia, 108 of extreme and 45 of moderate stenosis of the descending arch of the aorta at or near the insertion of the ductus arteriosus; in 126 of the cases a collateral circulation had

been observed, and in 150 there was more or less generalized hypertrophy of the heart, though in only 63 instances was an associated chronic valvular lesion reported; the ductus arteriosus was patent in 17, the aortic valve was biscupid in 51 and there was congenital hypoplasia of the ascending aorta in 21, with dilatation of this vessel in 101 cases and a dissecting aneurysm in 39 cases. Spontaneous rupture of the aorta was recorded 33 times, of the heart 2, and of the descending aorta 5 times. Cerebral death had occurred in 26, death from mycotic endocarditis in 14 and cardiac death in 67, which in 17 instances was sudden and in 50 by failing compensation.

The results of coarctation on the circulation are of much interest and depend upon the degree. The lesser degrees producing little effect while the more pronounced obliteration causes dilation above and often narrowing below. A collateral circulation develops to carry blood to the lower part of the body through the dilated internal mammary, scapular, intercostal, and deep epigastric arteries. The heart enlarges and may become greatly hypertrophied and dilated.

The insidious and early course and frequent latency as regards symptoms of this defect, owing to the gradual slow development of an adequate collateral circulation in the upper part of the body, and its frequent occurrence in athletic and apparently healthy male subjects of a high degree of intelligence combined only too commonly with its abrupt termination in the full flood of early adult life by one or other of the catastrophes enumerated above, makes this one of the most startling dramatic pictures in the whole history of disease.

The knowledge of the peril which it may carry in its train should impel the physician to analyze carefully hypertension in young subjects and especially when difference in blood pressure in the upper and lower extremities is present, with other vascular signs, to keep in mind the existence of the condition and of the necessity for avoidance of sources of infection and laborious occupations involving undue physical strain.

There are no particular symptoms of coarctation of the aorta and often no signs, or such slight or misleading signs that the defect escapes notice during life. With higher degrees of coarctation there are a number of important signs: (1) inequality of blood pressure between the upper and lower extremities, the brachial systolic pressure being much elevated; (2) evidence of collateral circulation between the upper and lower parts of the body through the internal mammary, intercostal, scapular and deep epigastric arteries, which may be much dilated and visibly pulsating; (3) long systolic murmurs heard not only over the precordium and back but along the course of the dilated vessels and sometimes accompanied by palpable thrills; (4) decrease or absence of the shadow of the aortic knob

by roentgen ray, frequently dilatation of the ascending aorta and first part of the arch; (5) enlargement of the heart and sometimes signs of failure, due in part to frequently complicating heart lesions and in part to the associated hypertension caused apparently by the stenosis of the aorta.

The following case report well illustrates several of the classical cardinal signs, the insidious course, lack of symptoms, variability of signs, and the possibilities of easily overlooking the condition.

Case Report

F. R. O'B., midshipman, age 20, was admitted to the United States naval hospital, Annapolis, Md., on November 1, 1932, with the diagnosis of hypertension, arterial.

Patient stated that he had no complaints but to the contrary had never felt better in his life.

Family history.—Father and mother living and well. One brother living and well. None dead. No history of heart, lung, kidney, or other chronic disease.

Personal history.—Measles, chicken pox, and whooping cough. Tonsillectomy at the age of six years. No sickness other than minor athletic injuries since admission to the Naval Academy in June 1930. States he was always considered a normal healthy child, was always able to play and keep up with other children without any discomfort. Has never noted shortness of breath nor cyanosis while engaged in strenuous exercise. Played football and lacrosse during his plebe year and last year made the varsity lacrosse team. Always finished a game without trouble. No shortness of breath, cyanosis, headache, or dizziness. Patient states that last year at the time of the annual physical examination he was told that his blood pressure was high.

Record of examination in June 1930 shows that the patient had a tachycardia, pulse before exercise 120, after exercise 128, after rest 102. Blood pressure, 118 systolic and 89 diastolic.

At the annual physical examination in October 1932 patient was found to have a brachial blood pressure of 216 systolic and 106 diastolic. The blood pressure readings were checked periodically over a period of several weeks and the systolic pressure was found to be consistently above 190.

Physical examination on admission.—A large, well-developed, well-nourished, healthy-appearing, young adult male. Color good. Pupils equal and active. Nose and ears negative. Tonsils removed. Teeth in excellent condition. Thyroid normal. In the supra-sternal notch, along the course of the carotids and above both clavicals and extending into the axillae is seen a marked systolic pulsation. Palpation over these areas reveals no thrill. Above the left clavicle the vessel feels much enlarged, is firm, and does not relax during diastole. Thorax is large and well-formed. Expansion is good and equal. Percussion shows normal lung resonance. Breath sounds are normal throughout. Heart: apex palpable slightly within the midclavicular line. Impulse is strong, regular. No thrill is felt. Percussion shows an apparent slight enlargement to the right of the sternum. Auscultation shows action regular. Over the entire precordium is heard a loud systolic murmur, which is transmitted into the vessels of the neck and the axillae. The murmur is also heard in the back. The murmur is loudest over the aortic and pulmonic areas and least loud over the apex. Blood pressure left arm, three readings in a half hour, systolic 208, 190, 210; diastolic 116, 118, 120. Abdomen flat and muscular. Liver,

spleen, and kidneys not palpable. No masses. No areas of tenderness. Abdominal aorta not palpable. Genito-urinary organs normal. No glandular enlargement. Pulse in lower extremities doubtfully palpable. Feet moist and cool. No cyanosis. Blood pressure in the lower extremities is impossible to record except in the right popliteal artery where, after many difficulties, a systolic pressure of 94 was recorded. This pressure was rechecked and verified during consultation. Radiographic measurements of the heart (2-meter plate).

	Centimeters
Width of aorta	8.0
Width of left ventricle	11.8
Width of right ventricle	5.2
Width of right auricle	4.6
Width of left auricle	7.0
Transverse of heart	17.6

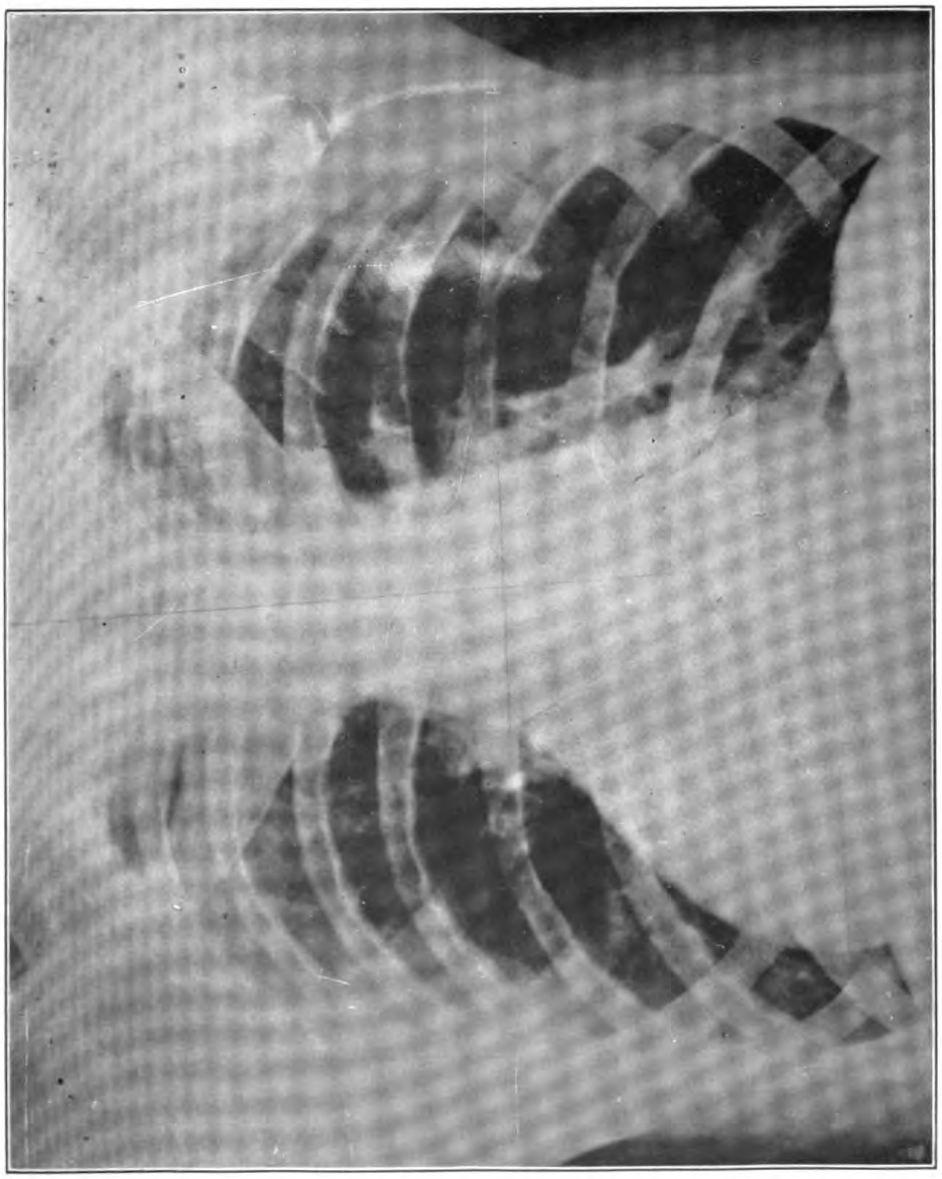
X-ray and fluoroscopic examination of chest and heart.—Heart central; right border prominent; ascending arch broadened and prominent; arcus absent; there is a small pointed appearance where the arcus is usually seen; left border convex; apex is at the midclavicular line. The inferior margins of the ribs (subcostal groove) are notched and irregular in outline, suggesting pressure destruction. A later view of the chest shows an apparent increase in the transverse diameter of the heart. The ascending limb of the aorta appears dilated. The transverse portion of the arch is not definitely outlined. There is a faint shadow which suggests a narrow arch. The oblique views, right and left, show what appears to be a much narrower descending arch with some irregularity in contour.

Electrocardiographic study.—Rate 60; rhythm regular except for sinus arhythmia; P waves upright in all leads; P is notched in lead 2 and 3; P-R interval 0.16 second; QRS occupies 0.08 second; contour is normal; diphasic in lead 1; T waves upright in lead 1 and 2, diphasic in lead 3. Conclusion: (1) tendency to right axis deviation; (2) sinus arhythmia.

Progress note.—This patient was under observation in the hospital for a period of 60 days. During this period of rest the brachial blood pressure settled down to between 170 and 200 systolic and the diastolic became fairly well fixed at 110. At no time was a definite pulse felt in the lower extremities nor was the blood pressure recorded other than in the right popliteal artery. The pulse rate usually ran about 80 but during sleep was often found to be as low as 40, slight exercise would cause a rise to 120. The systolic murmurs showed a marked variation from day to day and posture had its effect. The murmur at the apex was the most variable, at times it would be loud and long, again there would be a reduplication of the first sound, again the murmur would be entirely absent and two perfectly good sounds present, again it would be short and very faint. The murmurs at the aortic and pulmonary areas were next variable. At times loud and long, again scarcely audible and very short. The murmur at the tricuspid area was the most constant, varied less in intensity and was always audible.

Comment

With the above findings, e.g., a young male adult, high brachial pressure, low femoral pressure, enlargement of the heart, a systolic murmur heard over the entire precordia and in the back and the radiographic findings showing erosion of the ribs, dilatation of the ascending arch, absence of the aortic knob and suggestive narrowing



COARCTATION OF THE AORTA.

Showing notching and irregularity of the inferior margins of the ribs due to pressure destruction. (Gasser.)

of the aorta below the arch it is felt the diagnosis of coarctation of the aorta is justified.

Summary of Outstanding Points

1. Athletic, well built, apparently healthy young adult male.
2. Total lack of symptoms. Insidious course.
3. Hypertension in vessels of upper extremities; hypotension or inability to record pressure in lower extremities.
4. Enlarged, firm, pulsating vessels at the base of the neck and in the axillae. Evidence of collateral circulation as evidenced by rib erosion.
5. Precordial systolic murmurs transmitted into the vessels of the neck and axillae and to the back.
6. Variability of the murmurs.
7. X-ray evidence of dilatation of the ascending arch, absence of the aortic knob and suggestive narrowing of the aorta below the arch.

The writer wishes to thank Comdr. W. W. Hargrave, Medical Corps, United States Navy, United States Naval Medical School, who saw this case in consultation, for his great interest and most valuable assistance.

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ACUTE EPIDURAL ABSCESS OF THE SPINAL CORD

Report of Case

By H. L. Puckett, lieutenant (junior grade), Medical Corps, United States Navy and E. M. Harris, lieutenant, Medical Corps, United States Navy

Epidural abscess is a rare disease, there being only about 16 cases of the metastatic form on record (1). The acute stage of the disease is usually associated with furunculosis and is characterized by symptoms of infection and evidence of spinal compression. The case reported below is of especial interest because of its fulminant nature, its diagnostic problem, and its pathological anatomy.

Case Report

A white male, aged 38, was admitted to the hospital at 10 p.m. August 22, 1932, complaining of an inability to pass his urine during the preceding 12 hours. He was in good health until August 18, when he strained his back while attempting to stop a team of runaway horses which he was driving. The pain in the back grew more severe and was followed by a slight numbness in the legs. The following morning he was incapacitated to such an extent that he remained in bed. The condition remained unchanged then, until he developed the urinary retention.

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On physical examination at 10:30 a.m., August 23, his temperature was found to be 100.4° F. and his pulse 80. His gait seemed to be impaired by pain and hyperextension of the lumbar region. Tenderness was present over the lower lumbar vertebrae, sacrum, and ischium. The extremities were negative, except for furuncle about 3 centimeters in diameter on the right wrist. The prostate was normal in size and consistency, but the secretion was loaded with clumps of pus. The cystoscopic examination was negative. The neurological examination was also negative. The white blood count was 19,400, with 77 percent polymorphonuclear leukocytes and 23 percent lymphocytes.

The afternoon following the examination, the patient became slightly delirious. The next morning, August 24, it was noted that his abdomen was distended, and that he had developed a hyperactive left-knee jerk. A spinal puncture was done and a small amount of thick, yellow pus was obtained, from which a pure culture of *Staphylococcus aureus* was grown.

On August 26 the patient was obviously much weaker. The pupils were equal and regular, but were rather sluggish in their reaction to light. There was a sustained horizontal nystagmus. His abdominal reflexes and knee jerks were absent. The cremasteric reflex was sluggish. He was able to move the right leg, but not the left. The muscle strength in both was diminished. The pain sense was slightly impaired in the legs. X-rays of the spine were negative. The spinal puncture was repeated and thick, yellow pus was again found which revealed *Staphylococcus aureus* on culture.

In view of the rapidly progressive localizing signs of a spinal-cord lesion with definite evidence of the inflammatory nature of the condition, an exploratory laminectomy was decided upon. At 3:45 p.m., August 26, the spines and laminae of the third and fourth lumbar vertebrae were removed. Thick pus exuded from the epidural space, which was found to extend subdurally through a small opening in a dark friable dura. The wound was left open and packed with vaseline gauze.

Shortly after the operation the temperature arose from 101° F. to 106.4° F. The pulse mounted from 96 to 134, and the respirations from 34 to 48. By the next morning the temperature had dropped back to 101° F. and varied from that to 104° F. during the next 2 days. The abdominal distention and paralysis remained unchanged, while his delirium and general weakness became more pronounced. On August 30, 8 days after his admission, he died from an intercurrent broncho-pneumonia.

Necropsy

At autopsy the general appearance of the body was that of a normally developed, well nourished, white male. The recent surgical incision was present over the third and fourth lumbar vertebrae, open and packed with vaseline gauze. There was considerable foul pus on the gauze and dressing. The anterior border of the right wrist revealed a perforating ulcer of the skin about 3 centimeters in diameter, containing a large amount of purulent material. The cavity penetrated the superficial fascia and extended down to the transverse carpal ligament, which was intact, as were the sheaths of the neighboring flexor tendons.

On opening the skull no abnormal adhesions of the dura were noted. However, the blood vessels of the dura were engorged and a plastic exudate was found in the subarachnoid space. There were no abnormal adhesions between the arachoid and pia mater. The blood vessels in the latter were engorged and petechial hemorrhages were numerous over its surface. Section of the brain revealed no gross abnormalities.

The spinal cord and vertebrae were examined by exposing the cord from the site of the recent laminectomy to the eighth cervical vertebra. There was no demonstrable lesion in the adjacent soft tissue, nor was there evidence of fracture or dislocation of the vertebrae. The epidural space contained a large amount of thick, yellow, foul pus concentrated between the tenth thoracic and fourth lumbar vertebrae and traces of this could be seen as high as the eighth cervical vertebra. The blood vessels were engorged in the dura, the surface of which was dark grey in color. An opening, about one half centimeter in diameter, was found in the dura at the level of the third lumbar vertebra. The subarachnoid cavity contained a small quantity of turbid fluid with a reddish tint. The cord was not grossly abnormal.

Smears and cultures taken from the pia mater of the brain and the spinal canal were typical of *Staphylococcus aureus*.

The remainder of the autopsy was not remarkable except for an extensive broncho-pneumonia. A final diagnosis of epidural abscess of the spinal cord, purulent meningitis, cellulitis of the right wrist, and broncho-pneumonia was made.

Comment

The etiology of the epidural abscess in this case was probably metastatic from the cellulitis of the right wrist (1). However, the evidence would have been much more conclusive had several blood cultures been taken, as well as a smear and culture of the lesion on the right wrist. The history of trauma in this case is very important. The strain on the wrists in the attempt to stop the runaway horses was probably the main factor in the metastasis. The back strain evidently had little to do with the course of the disease, for there was no fracture, dislocation, or osteomyelitis of the vertebrae.

For a discussion of epidural abscess as a disease entity, the reader is referred to the excellent work of Dandy, who reviewed the literature on the subject at that time (2).

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MULTIPLE NEUROFIBROMATOSIS

(VON RECKLINGHAUSEN'S DISEASE)

With Report of a Case

By R. J. Leutsker, lieutenant commander, Medical Corps, United States Navy and J. H. Ward, lieutenant (junior grade), Medical Corps, United States Navy

This striking malady, originally described by von Recklinghausen in 1882, is a disease of uncertain etiology, probably neurogenic in origin, shows a strong hereditary tendency and the classical case com-

bines several outstanding features; multiple cutaneous fibromas, pigmentation of the skin and formation of large, loose cutaneous tumors (pachydermatoceles).

There has been considerable disagreement as to the histogenesis of the fibromas. Stewart and Copeland (1) state that "The whole scope of von Recklinghausen's disease is not yet defined nor is there agreement as to the nature of the cellular constituents making up the tumors which constitute the most prominent manifestation of the malady." Histologically the tumors are fibrous but contain a varying proportion of nerve cells arising from the epineurium, perineurium and endoneurium of the nerve trunks and that their origin is from the mesoderm is now generally accepted.

Pathologically are found:

(*a*) Few or numerous pedunculated or sessile, hard or soft tumors in the skin and subcutaneous tissue, varying greatly in size, sometimes following the distribution of a cutaneous nerve, sometimes well distributed over the entire body.

(*b*) Patchy, coffee colored pigmentation of the skin which frequently has a "bathing trunk" distribution.

(*c*) Superimposed on this picture are sometimes found large tumors of loose, wrinkled, thickened skin.

The disease is frequently associated with other stigmata of degeneracy, such as mental underdevelopment and epilepsy; and with such developmental defects as spina bifida, meningocele, and hypospadias.

There appears to be a definite connection between the tumors of von Recklinghausen and the solitary, malignant sarcomas of neurogenic origin. Stewart and Copeland (1) hold that, "The solitary, very cellular, anaplastic, rapidly metastasizing, fatal neurogenic sarcoma is but one link of a chain leading through varied clinical pathologic entities up to fully developed von Recklinghausen's neurofibromatosis with all its diverse related manifestations." They "acknowledge the multiple cutaneous neurofibromas, plexiform neuromas, multiple or solitary malignant neurosarcomas, elephantiasis neuromatosa, and ganglionic neuromas, alone or in association with other tumor processes, as part of the von Recklinghausen syndrome."

A large majority of the cases of von Recklinghausen's disease pursue a rather benign clinical course, however, a certain number estimated variously from 8 percent, Ewing (2), to 13 percent, Hosoi (3), are subject to changes and complications which seriously endanger life. Hosoi reports a fatal case of spindle cell sarcoma of neurogenic origin which occurred in a patient who presented the typical von Recklinghausen neurofibromatosis. Of 466 cases of von Recklinghausen's disease collected up to 1927, 13 percent showed

malignant transformation. Hosoi states that "In all those cases where sarcomas were found associated with multiple neurofibromas, the authors concluded that one of these neurofibromas had undergone sarcomatous change. It has been clinically observed that partial removal or any operative trauma of a neurofibroma may activate the tumor into a sarcoma." These secondary sarcomas are usually well walled off and metastasis is not frequent. However, "once malignancy has supervened, the prognosis is generally bad. The patients succumb to cachexia resulting from the many recurrences and operations."

Cutaneous neurofibromatosis may be asociated with neurofibromas of the deeper nerve structures accompanied by symptoms of pressure on the neighboring tissues. Hunt and Woolsey (4) report a case of extradural neurofibroma at the fifth cervical segment, complicating generalized neurofibromatosis (von Recklinghausen's), with lancinating pains in the shoulder followed in 6 months by leg symptoms of compression of the spinal cord. Bassoe and Nuzum (5) report a fatal case of central and peripheral neurofibromatosis. At autopsy there was found a neurofibromatous tumor in the left middle fossa and one in each cerebello-pontine angle and numerous small nodules connected with the roots of the spinal nerves.

The fact that melanomous tumors and neurofibromas have a neurogenic origin in common and the association of the melanotic pigmentation of the skin in von Recklinghausen's disease causes speculation as to whether or not the von Recklinghausen tumors are subject to melanous transformation. Stewart and Copeland report "a very extensive, hairy, bathing trunk nevus associated with neurofibromas. The patient died of melanosarcoma." Ewing states that "Melanoma has important relations to neurofibromatosis."

Heuer and Bell (6) present a series of four cases of von Recklinghausen's disease in each of which large pachydermatoceles were present. In case I there were recurrent spontaneous hemorrhages into the pachydermatocele, each one serious enough to threaten the life of the patient. At operation the source of the bleeding could not be found. In all, four operations were performed on this patient for hemorrhage. In case II there occurred hemorrhage into a large pachydermatocele following a fall in which the tumor was bruised. About a month later the patient was readmitted with a streptococcic infection of the tumor. After a rather prolonged, severe illness and a month's convalescence the entire tumor was removed at operation. Two years later he appeared with a large tumor, which proved to be sarcoma, associated with the sheath of the sciatic nerve. A year after its removal, following local recurrences, a high thigh amputation was done. After another year he returned with

local recurrences and generalized sarcomatous metastases. He died. In case III there was spontaneous hemorrhage into a large pachydermatocele. Medical help was not immediately available, and the patient died from shock and hemorrhage. In case IV there was spontaneous hemorrhage into a neurofibroma in the left temporal region. Operation failed to reveal the source of the bleeding.

Brooks and Lehman (7) report a series of seven cases of von Recklinghausen's neurofibromatosis in which the bone changes were marked. The changes included scoliosis, intermedulary bone cysts, subperiostial tumors which proved to be neurofibromata, spina bifida, and lengthening of long bones. The authors conclude that "von Recklinghausen's neurofibromatosis is a condition effecting bone as well as skin and nerve."

Case Report

P.S.A., age 38, Hebrew, Veteran's Administration patient, carpenter, was admitted to the San Diego Naval Hospital, August 9, 1932. His complaints on entry were recurrent bitemporal headaches, pain in left shoulder and left arm, freckling, pigmentation, and tumor masses about trunk, back, and legs. His past history was quite uneventful except for an appendectomy in 1912 and two plastic operations on his nose. Family history, father died at 55, cancer of the throat. He has been married for 8 years and has one normal male child of 6.

Onset of all complaints, which have been persistent, dates to service overseas in 1918.

(*a*) Attacks of "sick headaches" began after heavy firing. These are characterized by scintillating spots before the eyes accompanied by nausea and followed by severe persistent bitemporal headaches. These attacks recur almost at weekly intervals.

(*b*) Recurrent attacks of pain in left cervical region, left shoulder, and left arm; not usually incapacitating and rarely so severe he can't raise his arm.

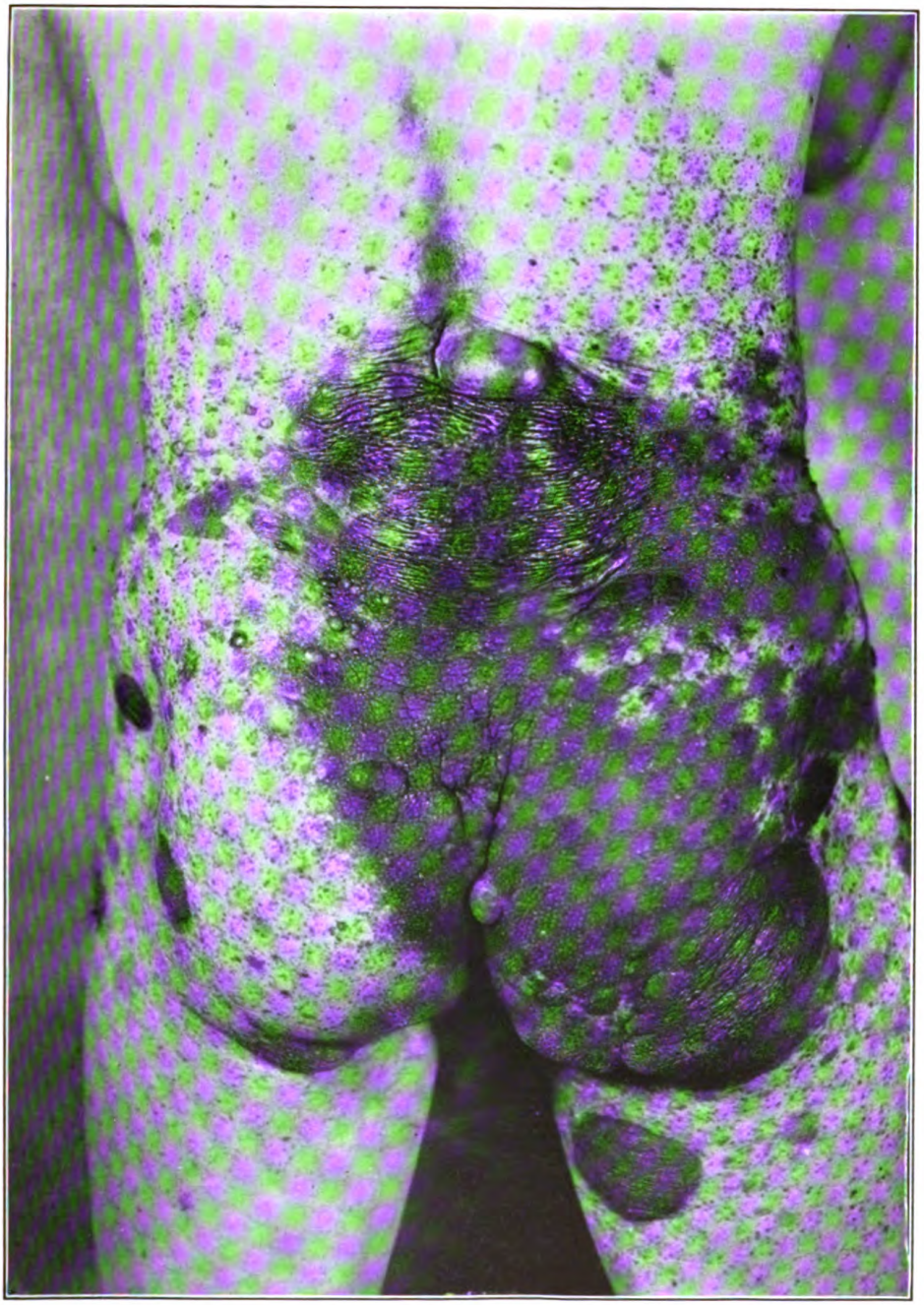
(*c*) While having a stool in a shell hole, the patient noted burning and stinging over buttocks and back which he attributed to burning from mustard gas. Eight months later, flat, not elevated, brown bloches appeared about groins and back. This assumed a bathing trunk distribution. One year later small tumor masses began to appear under the skin over the same area mentioned above. Shortly after this, a larger tumor in the small of his back suddenly increased in size; from this there was superficial bleeding. (This incident has never recurred.) Tumor masses and pigmentation spread up the back and down the legs until 5 years ago and since then the condition has been practically stationary. The tumors are painless except when bumped or bruised and then pain is severe and occasionally persists 2 or 3 days.

There has been no operative interference.

It might be well to note that prior to service patient had never noted any skin blemish other than a small mole on the right arm that he had carried from birth. This has never increased in size.

Physical examination revealed a slender, small, intelligent patient in no distress other than areas of pigmentation, tumors, etc. The examination was essentially negative.

The area involved is that of the "bathing trunk" variety. It is from the course of the ninth costal nerves, where there is a sharp line of demarcation,



CASE OF MULTIPLE NEUROFIBROMATOSIS.

(Leutsker and Ward.)

down to 3 inches above the knees (although there is a small patch of "freckles" on the left shoulder and another patch on the lateral aspect of the right calf). There are numerous tumor masses (cutaneous and subcutaneous, some firm, others soft and pliable varying in size from a pinhead to that of a walnut; the smaller ones are occasionally pedunculated, the larger ones sessile); large blochy pigmented areas about the groins and buttocks; areas of "freckling"; larger masses composed of loose folds of tissue covered by wrinkled and deeply pigmented skin thrown into folds, the largest, 5 by 10 centimeters, is in the small of the back, several similar but smaller masses are about the buttocks and thighs.

Laboratory and X-ray examinations were negative. These included routine blood and urine, blood chemistry, and X-rays of skull, spine, all long bones, and chest.

Diagnosis: (1) Von Recklinghausen's disease, (2) migraine, (3) neuritis, left shoulder.

SUMMARY

A case of von Recklinghausen's disease is presented in which:

(*a*) All the principle visible features are present and marked.

(*b*) The factor of heredity does not appear.

(*c*) It has been impossible to demonstrate any of the stigmata of degeneracy or any other developmental defects.

(*d*) There are no associated bony changes.

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HEMOCHROMATOSIS [1]

REPORT OF CASE

By G. A. CANN, lieutenant (junior grade), Medical Corps, United States Navy

Hemochromatosis is sufficiently rare to warrant reporting a case in which the clinical diagnosis was substantiated by post-mortem pathological examination. The case is of especial interest because of the long duration of the disease prior to the onset of diabetes and its consequent confusion with Addison's disease.

Regarding the pathogenesis of hemochromatosis, knowledge is far from complete. Von Recklinghausen in 1889 (1) showed the pigmentation of the skin and viscera to be due to the deposition of an iron-containing pigment, hemosiderin, and a non-iron-containing pigment, hemofuscin, and pointed out the similarity of these products

[1] From the medical service, United States naval hospital, Puget Sound.

to the blood pigments, which he further indicated by describing the disease under the name of hemochromatosis. He believed the pigment was produced by a destruction of the red blood cells.

Rous (2) supported this view by producing an identical condition in rabbits by repeated transfusions of blood, so that large amounts were being constantly destroyed. But in the cases of hemochromatosis reported, there has been no obvious anemia, no special activity of the blood-forming organs which would, of course, occur to anyone were a destruction of blood the source of so much iron-containing pigment.

Sprunt (3) in his summary of our knowledge of the disease concludes that there is no evidence of abnormal blood destruction, and that hemochromatosis is a primary disorder of metabolism, "implicating many of the body tissues, and manifested by a change in the chromogenic groups of the proteid molecule with the deposition of pigments."

Mallory (4) has advanced the theory that chronic poisoning with copper may be the cause of hemochromatosis, and has submitted as proof work on experimental animals, which were made to ingest and inhale copper with the production of lesions similar to those found in hemochromatosis.

Although the etiological agent giving rise to the disturbance in pigment metabolism which occurs in hemochromatosis is still a matter of dispute, it has been definitely demonstrated that the pathological changes are dependent upon the pigment deposition. Opie (5) in 1899 gave an admirable description of the pathology of the disease, to which little has been added since. The hemofuscin and hemosiderin, presumably derived from the hemoglobin, are deposited in the Kupffer's cells of the endothelium lining the sinusoids and in the parenchymatous cells of the liver. After the liver cells have taken up as much pigment as they can hold, it then begins to be deposited in the other organs and tisues, especially in the pancreas, cortex of the adrenal glands, lymph nodes in the upper abdomen, the heart, thyroid, skin, mucosa of the stomach, etc. The deposited pigment apparently acts as a foreign body, leading to much new growth of encapsulating fibrous tissue, resulting in tissue degeneration and necrosis.

The disease is a degenerative disease and hence one of middle age. Of the cases on record very few occurred in women. Dr. Maude Abbott's patient was known as "Blue Mary."

The clinical features, aside from the pigmentation of the skin which varies in color from a dark brown to a leaden or bluish black and is the thing which usually causes the patient to seek medical attention, are dependent upon the degenerative processes which may

produce myocardial insufficiency through fibrosis of the myocardium, parenchymatous degeneration of the kidneys with clinical nephritis, cirrhosis of the liver and pancreas with diabetes, or any combination of these. The diabetes is usually severe, and the course of the disease rapidly fatal after its onset.

Treatment is directed toward the specific pathological conditions clinically manifested, i.e., the myocarditis, nephritis, hepatic cirrhosis or diabetes, and toward the upbuilding of the general health.

As the end approaches, the measures are chiefly supportive and palliative.

Case Report

J. H. W., aged 56, Veterans' Administration patient, single, carpenter by trade, was admitted to the United States naval hospital, Bremerton, Wash., on May 9, 1932, with a diagnosis of Addison's disease.

Complaints on admission.—(1) Stomach cramps, gaseous distention, belching of gas and sour eructations; (2) chronic constipation; (3) dark discoloration and roughness of skin of entire body; (4) inability to perspire; (5) generalized weakness and loss of strength; (6) excessive thirst and excessive urination.

Family history.—No history of tuberculosis or diabetes.

Past history.—"Typhoid-malaria fever" when 14 years old. Pneumonia when 21. Mumps in 1920, complicated by a bilateral orchitis. "Cholera morbus, diarrhoea, and ptomaine poisoning" while in the Army between 1898 and 1902. All teeth extracted. No history of venereal infection. Has had no sexual desire or activity for 15 years.

Present illness.—The patient noticed his skin becoming dark in color 15 years ago. Thought at first he was being tanned by the sun, but the condition did not clear up in winter. His skin then became dry, rough, and scaly and the discoloration became progressive. His general health was unimpaired and he did not seek medical attention until 1925, when he had an attack of unexplained fever. Since that time has had several hospitalizations with no improvement in his condition. He was treated in this hospital for 2 weeks in October 1930 as a case of Addison's disease. At that time the blood pressure was recorded as systolic, 110 and diastolic, 80; the basal metabolic rate as minus 2; and the blood sugar as 93. The symptoms of excessive thirst and urination only manifested themselves about 2 months prior to this admission. No history of having taken any metallic preparations—silver, arsenic, or copper.

Physical examination.—The most striking feature of the examination was the grayish-leaden discoloration of the entire skin surface. The patient was fairly well developed and well nourished, but appeared rather senile. The body contour was masculine, but the breasts tended towards a feminine development, and the body was quite free from hair. The axillary hair was soft and fine, the pubic hair feminine in distribution and the beard sparse. Asthenia not marked.

Skin: there was a diffuse grayish-leaden pigmentation of the entire skin surface. The skin was very finely wrinkled (senile), dry, rough, and covered with fine furfuraceous scales. Scaliness was more pronounced on extremities.

Head and neck: hair thin and fine, turning gray; scalp very dry and scaly. Sclerae and conjunctivae had a slight grayish discoloration; moderate arcus senilis; pupils equal, regular, react normally; vision, both eyes, 20/100;

examination of fundi showed early arteriosclerotic changes. Nasal mucosa atrophied and crusted. Ears negative. Lips bluish. Edentulous; gums very atrophic; did not wear dental plates. Buccal mucosa and gums free from pigmentation. Thyroid not palpable.

Heart: no enlargement; heart sounds faint; rhythm regular; rate rapid (100 per minute); no murmurs or thrills. Blood pressure: Systolic, 94 and diastolic, 70.

Lungs: negative.

Abdomen: ptotic; slightly distended. Moderate tenderness on pressure in epigastrium and right hypochondrium. Liver palpable two fingerbreadths below costal margin, firm, smooth. Spleen and kidneys not felt.

Genitalia: Both testicles atrophic.

Rectal examination: negative.

Reflexes: normal.

Temperature, 99.2° F., pulse, 100; respiration, 20.

Urine: acid. Specific gravity, 1.042. Albumin, trace. Sugar, positive (heavy reduction). Microscopic: rare finely granular cast; much mucus; 3 to 6 leucocytes per high-power field; many red blood cells. Acetone and diacetic acid negative.

Blood: erythrocytes, 3,790,000; hemoglobin, 80 percent; leucocytes, 8,800; polymorphonuclears, 63 percent; lymphocytes, 30 percent; endotheliocytes, 4 percent; eosinophiles, 3 percent. Blood Kahn, negative. Blood chemistry: nonprotein nitrogen, 32 milligrams; urea nitrogen, 15 milligrams; uric acid, 3 milligrams; creatinine, 1.3 milligrams; blood chlorides, 398 milligrams. Blood sugar, 272 milligrams. Icterus index, 9.

Gastric analysis (Ewald test meal): free hydrochloric acid 18°; combined acids, 6°; total acidity, 24°. Bile, mucus and lactic acid, negative; occult blood, trace.

Basal metabolic rate: plus 11.

Electrocardiogram: rate, 91 per minute. Rhythm regular. P-R Interval, 0.18 second. QRS complex of low amplitude throughout; inverted in leads II and III; slightly notched. T wave flattened in all Leads. Conclusion: chronic myocarditis. Left axial deviation.

X-Ray of abdomen: no calcification in adrenals. Moderate degree of hypertrophic arthritis of spine.

Clinical diagnosis.—(1) Hemochromatosis; (2) chronic myocarditis without cardiac decompensation; (3) endocrinopathy (thyreo-gonadal-adrenal).

Course of disease.—The patient was placed on a diabetic diet of carbohydrate 52, protein 32, fat 66 (calories, 930), and within 2 days became sugar free. The diet was gradually increased to carbohydrate 84, protein 61, fat 94 (calories, 1,426), when it became necessary to administer insulin units V three times daily to keep the urine sugar free. By June 1, 1932, the patient was up and about, receiving a diet of carbohydrate 84, proteins 78, fat 132 (calories, 1,836), and insulin units V three times daily, and was symptom-free. The patient was discharged June 3. 1932, upon his own insistance, after teaching him to test his urine for sugar, to administer his insulin to himself, and giving him written dietary instructions. His weight upon discharge was 150½ pounds and his blood pressure was systolic, 100; diastolic, 76.

On June 15, 1932, the patient was readmitted to the hospital with a recurrence of his gastro-intestinal symptoms. The patient's condition was much the same as on his previous admission with the exception that he appeared very depressed mentally and convinced that a diabetic diet did not agree with him.

His weight was 141 pounds; blood pressure—systolic, 90; diastolic, 64; temperature, 99.4° F.; pulse, 96; and respiration, 20. His blood sugar was 375 milligrams per 100 centimeters, and his urine strongly positive for sugar, but negative for acetone and diacetic acid. The blood count on this occasion showed 4,300,000 erythrocytes and 85 percent hemoglobin, the white and differential count being essentially the same as previously.

The patient was placed on a diabetic diet of carbohydrate 64, protein 44, fat 83 (calories, 1,179), and insulin units XV three times daily.

By June 30 the diet had been increased to carbohydrate 109, protein 66, fat 110 (calories, 1,771), and the insulin to units XX—XV—XX. The urine remained sugar free, but the patient remained mentally depressed and had very little appetite, within the ensuing week the patient took a decided down-hill course with progressive weakness and refusal to eat. The blood pressure remained low and the pulse weak. On July 7 the patient was given 1,000 cubic centimeters normal saline and 10 cubic centimeters eschatin (Pfiffner and Swingle preparation of adrenal cortical extract) intravenously and was put on strychnine sulphate hypodermically.

The patient's condition became progressively more critical with coma developing, although the urine was kept free from sugar, acetone, and diacetic acid by glucose solution and insulin intravenously. Normal saline and eschatin were also given by vein daily. Death occurred on July 13, 1932.

Autopsy Report

Skin: There is a leaden grayish discoloration of the skin, especially noted over face, neck, fore arms, hands, and ears. The skin is very dry and scaly. There is a slight edema of the inferior extremities.

Heart: there is a thick yellowish pericardial effusion of 300 cubic centimeters. The visceral pericardium shows on the anterior surface a white irregular plaque three fourths by 1 inch. The remainder of the pericardium shows much scarring and innumerable small petechial areas of deep red coloration. The myocardium is slightly hypertrophied with much scarring and a marked softening of the muscle. All valves show a slight amount of sclerotic change and on the mitral valve is seen recent soft multiple vegetations of pale color. Ante-mortem and post-mortem clots seen in cavities of heart. Right side of heart is moderately dilated.

Liver: the liver is slightly enlarged. It is granular in appearance on section and has an ochre discoloration.

Pancreas: there is a dusky red discoloration of the entire pancreas. The organ is much softer than normal.

Kidneys: moderate chronic passive congestion and cloudy swelling.

Adrenals: the adrenals are slightly smaller than normal. Contour normal. The glands are very soft. There are no areas of necrosis visible, but there is a deep brown discoloration of the medullary portion.

Gastro-intestinal tract: the colon shows chronic passive congestion to a marked degree. The stomach is moderately dilated.

Lungs, spleen, bladder and prostate: negative.

Gross pathological diagnosis.—(1) Chronic myocarditis; (2) subacute pericarditis; (3) chronic hepatitis; (4) chronic pancreatitis; (5) parenchymatous degeneration of kidneys; (6) parenchymal degeneration of adrenals; (7) pigmentation of the skin.

Histopathological examination.—(The following report was submitted by Dr. R. L. Benson, pathologist, United States Veterans' Administration hospital,

Portland, Oreg.:) Liver: there is dense infiltration of dark brownish pigment granules in the liver cord cells, in the epithelium of the bile ducts and, to some extent, in the connective tissue. The connective tissue is considerably increased with formation of thick septa which in some places inclose islands of tissue consisting of one or more lobules.

Kidney: there is advanced degree of parenchymatous degeneration of the tubules, especially in the loop of Henle, together with focal atrophy of the tubules.

Adrenal: there is considerable brown pigment infiltration of the epithelial cells in the outer zone of the cortex.

Skin: very little, of any, pigment changes are seen.

Pancreas: the epithelial cells of the acini are densely infiltrated with brown pigment granules. In places there is degeneration of the pancreatic parenchyma with loss of the acini. There is also fibrosis and scarring. It is difficult to distinguish any islets because of degenerative changes.

Heart: there is moderate fibrosis of the myocardium. Some of the muscle fibres are diffusely infiltrated with brown pigment granules.

Testicle: there is a considerable atrophy of the seminiferous tubules with areas of complete hyaline necrosis.

Summary: (1) probable hemochromatosis with pigmentation of liver, adrenals, myocardium and pancreas (to be checked with stain for hemosiderin later); (2) secondary cirrhosis of liver; (3) chronic pancreatitis with considerable loss of acini and of islets of Langerhan's; (4) Parenchymatous degeneration and atrophy of renal parenchyma; (5) parenchymatous degeneration of seminiferous tubules of testes.

Supplementary report of Doctor Benson (sections stained with Mallory's ammonium sulphide stain for iron): the liver cells are found to contain black stained granules which in places are numerous and dense. Presence of iron granules is also confirmed in the pancreas, adrenal, myocardium and a little is found in the kidney and skin.

Diagnosis: Hemochromatosis. Chronic pancreatitis with degeneration of islets.

Discussion

The diabetes in this case provided the clue to the correct diagnosis. To differentiate between Addison's disease and hemochromatosis before the onset of diabetes is not an easy matter. Where hemochromatosis is suspected, the removal of a piece of skin and the demonstration of the presence of iron-containing pigment may establish the diagnosis. A lowered glucose tolerance in the prediabetic stage provides confirmatory evidence. In this case the points in favor of Addison's disease were the pigmentation, low blood pressure, gastro-intestinal symptoms, and weakness. Against Addison's disease were the duration of the disease, lack of marked asthenia, the hyperglycemia, normal blood chemistry, high basal metabolic rate, and lack of response to eschatin. However, it is to be remembered the adrenals were included in the degenerative changes produced by the disease, so that a certain degree of hypoadrenia appeared in the clinical picture.

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REPORT OF A CASE OF SEPTICEMIC PLAGUE IN WHICH PRACTICALLY THE ONLY SYMPTOM WAS AN ELEVATED TEMPERATURE

By F. H. JOHNSON, lieutenant (junior grade), Medical Corps, United States Navy

CASE REPORT

The U.S.S. *Palos*, patrolling the Yangtze River, left Shanghai on June 1, 1932, for an upriver trip; made 3 or 4 short stops at different ports and arrived at Kiukiang, China, on June 14.

C. E. T., seaman, second class, age 19 years, went ashore June 19, for the first time since the ship had left Shanghai. He was ashore only a short time. According to his companion, the two of them went into a native store, made a few purchases, and then returned to the ship.

At 9 a.m. June 22, the patient reported to the sick bay complaining of feeling "hot" and of having a slight headache. His temperature was 101.2° F.; pulse, 90; and the pharynx appeared slightly inflamed. Otherwise the physical examination was negative. He was put to bed; soft diet and forced fluids were ordered. During the afternoon his temperature rose to 102.2° F.; pulse, 110.

The next morning, June 23, patient stated he had no complaints whatever except that he felt "hot." The headache was entirely gone, he said. Temperature, 101.4° F.; pulse, 90. He appeared cheerful and talkative. White blood count, 7,800; polymorphonuclears, 47; lymphocytes, 51; eosinophiles, 2. During the afternoon his temperature went to 103° F.; pulse, 120.

On the morning of June 24, he again had no complaints. His temperature ranged from 101° F. in the morning to 101.8° F. in the afternoon. He laid in his bunk and read books and magazines, talked and joked with the pharmacist's mate. He did not appear very ill.

He was observed at 9 p.m. and at 1 a.m. by the medical officer; on both occasions he was apparently asleep. The pharmacist's mate slept in a bunk just beneath the patient's and heard nothing from him during the night.

On arising at 6:45 a.m. the pharmacist's mate found the patient to be dead. He was lying on his right side. His knees, hips, and neck were in moderate flexion; the bed clothes were tucked neatly about his body.

An autopsy was performed to determine the cause of death. The findings were as follows:

The body is that of a white male about 20 years of age, well nourished and bearing no signs of external violence. The skin is clear except for extensive purplish-red discoloration of the dependent right side of the body. Upon opening the thorax dense fibrous adhesions between the parietal and costal pleura are seen on the left side. The lower lobes of both lungs are blackish-red in color; blood exudes from the cut surface. The lungs, however, crepitate throughout. The middle lobe and both upper lobes appear normal. About the

hili of both lungs are many calcifications, the largest of which is about the size of a navy bean.

The heart and great vessels appear normal.

The liver is moderately enlarged; is dark red in color and on cut section is dark blood-red in color. The gall bladder is mottled, grayish-green in color. It is larger than usual. Upon opening it a thick yellow fluid is found. This material is odorless.

The pancreas is larger than usual. It is quite firm in consistency. On cut section numerous bright red areas about the size of a pin head are found.

The spleen is more than twice the normal size. It measures 22 by 13 centimeters. It is reddish-black, very soft; and on cut section the pulp is reddish-black, friable and mushy.

The kidneys are somewhat larger than usual; on cut section the tissue is dark red. The cortical markings are very pronounced.

Upon opening the stomach about one half cup of unclotted blood is found. The wall of the stomach seems very thin and flabby. The mucous surface throughout is dull brick-red in color. The duodenum and jejunum are very soft, thin and flabby. The serous surfaces are purplish red in color, the mucous surface is dull brick-red in color.

There are numerous masses, ranging in size from a pea to a lima bean, dark red in color, in the mesentery of the small gut. The masses on cut section are grayish red in color. They are of about the same consistency as kidney tissue.

The blood in the large vessels is unclotted. No clotted blood has been seen during the entire autopsy.

Smears were made from the cut surface of the spleen for bacteriological examination. On microscopic examination of the stained smears, short, thick, bipolar, Gram-negative bacilli in large numbers were found. Diagnosis: *Bacillus pestis.*

Anatomical diagnosis:

1. Chronic fibrous pleurisy.
2. Healed hilar tuberculosis with calcification.
3. Hypostatic congestion, lower lobes of both lungs.
4. Passive congestion of liver and kidneys.
5. Enlarged, congested spleen.
6. Empyema of gall bladder.
7. Hemorrhage into walls of stomach, duodenum and jejunum.
8. Hemorrhage into tissue of pancreas.
9. Multiple, enlarged mesenteric lymph glands.
10. Septicemia, the causative organism of which is the *B. pestis.*

Comment

This case is remarkable on account of the absence of the usual clinical symptoms of prostration, delirium, etc. Dangerous illness was not evident. The diagnosis was suggested at autopsy by the enlarged lymph glands in the mesentery of the small gut and by the absence of clotted blood.

Sudden hemorrhages, occurring simultaneously, in the tissues of numerous vital organs, seem a reasonable explanation of the immediate cause of death.

NAVAL RESERVE

MEDICAL CORPS

APPOINTMENTS, FIRST QUARTER, 1933

Name	Rank	Appointed
Crutchett, William L	Lieutenant (junior grade), MC-V(G)	Nov. 29, 1932
Harden, Wyman W	do	Dec. 16, 1932
Hummel, Merwin L	do	Nov. 16, 1932
Person, Edward C	do	Nov. 17, 1932
Wheeler, Howard L	Lieutenant, MC-V(S)	Dec. 8, 1932
Gilshannon, Bernard J	do	Jan. 3, 1933
Glidden, Henry S	Lieutenant (junior grade), MC-V(G)	Jan. 13, 1933
Hankins, Franklyn D	do	Jan. 15, 1933
Icks, Karl	do	Dec. 28, 1932
Steen, William B	do	Dec. 8, 1932
De Milia, Alfred F	do	Feb. 14, 1933
Langley, Robert W	Lieutenant, MC-V(S)	Dec. 21, 1932
Shinn, Adam L	Lieutenant (junior grade), MC-V(G)	Feb. 14, 1933
Willis, Park W., Jr	Lieutenant, MC-V(S)	Dec. 28, 1932

PROMOTIONS

Name	From—	To—
Gelber, Maksymiljan Robert.	Lieutenant (junior grade), MC-V(G)	Lieutenant, MC-V(G).
Hockett, Verden E	do	Do.
Smalzried, Elmer W	Lieutenant (junior grade), MC-F	Lieutenant, MC-F.
Leete, Edward D	Lieutenant, MC-V(G)	Lieutenant commander, MC-V(G).
Losli, Ernest J	Lieutenant (junior grade), MC-V(G)	Lieutenant, MC-V(G).
Sala, Roland O	Lieutenant (junior grade), MC-F	Lieutenant, MC-F.

DENTAL CORPS

APPOINTMENTS, FIRST QUARTER, 1933

Name	Rank	Appointed
Barth, Jesse B	Lieutenant (junior grade), DC-V(S)	Jan. 21, 1933
Curry, Roy C	Lieutenant (junior grade), DC-V(G)	Jan. 28, 1933
Gibson, Henry L	do	Mar. 16, 1933
Molt, Frederick F	Lieutenant commander, DC-V(S)	Mar. 11, 1933
Rose, Clifford E	do	Jan. 7, 1933
Salman, Irving	Lieutenant (junior grade), DC-V(G)	Feb. 7, 1933
Smith, William A	do	Mar. 16, 1933
Walker, Alfred S	Lieutenant commander, DC-V(S)	Jan. 16, 1933
Weeden, Joseph B	Lieutenant (junior grade), DC-V(G)	Mar. 11, 1933

PROMOTIONS

Name	From—	To—
Tatum, Laurice A	Lieutenant (junior grade), DC-V(G)	**Lieutenant DC-V(G).**
Schneider, Wilber J	do	**Do.**

NOTES AND COMMENTS

NEW STANDARD BOOK LIST FOR FISCAL YEAR 1934

Effective July 1, 1933, a new standard list of books will form the supply table list for all Medical Department activities except hospitals. Requisitions from hospitals, as in the past, will not be limited to any particular list.

The new list appears below. Asterisk indicates changes made since the list published in the July 1932 BULLETIN.

The bureau always welcomes suggestions from medical officers as to the desirability of retaining any of these books on the list, or of replacing them by others.

Textbook of Anatomy, Cunningham, 6th edition.
* Regional Anesthesia, Labat, 2d edition.
Practical Bacteriology, Blood Work and Animal Parasitology, Stitt, 8th edition.
Chemistry, Inorganic Pharmaceutical, Rogers, 1930.
* Conduction, Infiltration, and General Anesthesia in Dentistry, Nevin and Puterbaugh, 3d edition.
Dental Histology and Embryology, Noyes, 4th edition.
Dental Formulary, Prinz, 4th edition.
Bacterial Infection, Appleton, 1925.
Modern Dental Materia Medica, Pharmacology, and Therapeutics, Buckley, 5th edition.
Dental Pathology and Therapeutics, Buchard and Inglis, 7th edition.
Dental Roentgenology, Ennis, 1931.
Textbook of Exodontia, Winter, 2d edition.
Dental Dictionary, Ottofy, 1923.
Operative Dentistry, McGehee, 1930.
Periodontal Diseases, Merritt, 1930.
Prosthetic Dentistry, Nichols, 1930.
Medical Diagnosis for the Student and Practitioner, Greene, 6th edition.
* American Illustrated Medical Dictionary, Dorland, 16th edition.
Diseases of the Skin, Andrews, 1930.
Dispensatory, Wood and La Wall, 21st edition.
Drill Book for Hospital Corps, United States Navy, 1920.
The Nose, Throat, and Ear, and Their Diseases, Jackson and Coates, 1929.
Food Analysis, Typical Methods and the Interpretation of Results, Woodman, 3d edition.
Fractures and Dislocations, Speed, 2d edition.
Urology, Eisendrath and Rolnick, 2d edition.
Gonorrhea in the Male and Female, Pelouze, 2d edition.
Hospital Corps Handbook, United States Navy, 1930.
Naval Hygiene, Pryor, 1918.

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Manual of the Medical Department, United States Navy, 1927.
Medical Compend for Commanders of Naval Vessels, 1923.
Textbook of Medicine, Cecil, 2d edition.
National Formulary, 5th edition.
Diseases of the Nervous System, Jelliffe and White, 5th edition.
Principles and Practice of Nursing, Harmer, 2d edition.
Manual of the Diseases of the Eye, May, 13th edition.
Pharmacopoeia of the United States, 10th edition.
Practice of Pharmacy, Arny, 3d edition.
Preventive Medicine and Hygiene, Rosenau, 5th edition.
Modern, Surgery, General and Operative, Da Costa, 10th edition.
Modern Clinical Syphilology, Stokes, 1926.
Principles of Therapeutics, Hare, 21st edition.
Diagnostics and Treatment of Tropical Diseases, Stitt, 5th edition.
Modern X-ray Technic, Jerman, 1928.

CORRECTION

In the article on A Study of the Convulsive Seizures Caused by Breathing Oxygen at High Pressures, by Lt. (Jr. Gr.) C. W. Shilling and Lt. Comdr. B. H. Adams, Medical Corps, United States Navy, which was published in the April 1933 number of the Naval Medical Bulletin, the second sentence of paragraph 2 under Discussion (page 119), beginning with the words, "In spite of, etc." should be changed to read. " In spite of this, were the submarine at a depth of 200 feet, the pressure of oxygen in the 'lung' might be approximately 5.25 atmospheres absolute, or 64 pounds gauge, and in order to avoid the possible hazard of breathing such a tension of oxygen, the 'lung' should not be breathed from for a period longer than is necessary for the adequate testing of the adjustment of the appliance preparatory to escape."

FORWARDING OF SPECIMENS FOR BOTH CHEMICAL AND HISTOPATHOLOGICAL EXAMINATION

Abdominal viscera have been received at the Naval Medical School in a frozen state for both chemical and histopathological examination. Paraffin sections, made after tissue has been frozen and thawed, show considerable postmortem change which tends to obscure the histopathological picture.

It is recommended that the use of cold as a preservative be limited to those tissues for chemical examination and that small cubes of tissue for histopathological examination be placed in 10 percent formalin to assure proper fixation.

In addition to the above suggestion, it may be stated that chapter 22, manual of the Medical Department, contains a number of practical points concerning the collecting, preserving, and forwarding of pathological, bacteriological, and other specimens.

CARBON MONOXIDE FROM PAINT IN SEALED COMPARTMENTS

In the Journal of Industrial Hygiene of January 1933, S. F. Dudley, F. G. Edmed, and R. C. Frederick, Royal Naval Medical School, Greenwich, report the results of further research on the production of carbon monoxide from paint in sealed compartments.

A previous communication concerning this investigation, which appeared in the same journal, was commented on in the July 1932 number of the Naval Medical Bulletin.

In discussing their work, the authors state:

When a sealed compartment contains drying paint the oxygen is absorbed and carbon monoxide is evolved. It would seem that, in the British Navy, under the usual routine of painting and, later, examination of such sealed compartments, the absorption of oxygen generally proceeds to such an extent as to produce an irrespirable atmosphere by the time the compartment is reopened for examination. And it is deficiency of oxygen, rather than the presence of carbon monoxide, which is most often the danger to be feared by anyone entering such a compartment. Nevertheless as was shown in the previous communication, under some conditions the atmosphere may contain enough oxygen to support life long enough for a man or animal to obtain a dangerous or lethal dose of carbon monoxide. In the former paper this inference was supported by no direct oxygen analyses. In this communication, further experiments have shown directly that considerable quantities of oxygen may exist in a sealed space in the presence of a dangerous quantity of carbon monoxide. For example the gas in a can which had been sealed for a month contained 12 percent of oxygen and 0.36 percent of carbon monoxide. A similar can opened after three months contained only 3 percent of oxygen and 0.39 percent of carbon monoxide. A man entering an atmosphere of the first composition might breathe long enough to acquire a fatal dose of carbon monoxide, while in the second case it is certain that he would "drown" before the carbon monoxide could poison him. One notes in this experiment that after 1 month there was little less carbon monoxide than after 3 months, whereas there was four times as much oxygen at 1 month as at 3 months. It would appear from this that the absorption of oxygen is a relatively slower process than the evolution of carbon monoxide—that is to say more time is required for the former reaction to reach its "end-point." Hence probably there is often a critical period when there is enough oxygen to support respiration in the presence of a dangerous concentration of carbon monoxide. Later more oxygen is absorbed, and the atmosphere becomes irrespirable.

* * * two factors, quantity of paint (or perhaps rather the surface area exposed) and time the compartment has remained sealed, determine whether the atmosphere in the compartment will "drown" an animal, or poison it with carbon monoxide. In practice it seems probable that the quantity of paint used, and time the compartments are left sealed before reopening, is generally sufficient to exhaust practically all the oxygen. It is most likely therefore that deaths from carbon monoxide poisoning are due to incomplete ventilation of such compartments after they have been opened for inspection. The original atmosphere may have been too deficient in oxygen to support life, but by partial ventilation becomes diluted with normal air to an extent sufficient to maintain respiration, but insufficient to reduce the carbon monoxide below the danger level. * * *

Summary.—It is shown that any composition such as paint, containing linseed oil will in a confined space such as a sealed compartment of a ship, not only give rise to dangerous concentrations of carbon monoxide but will also, through absorption of oxygen during drying, produce an atmosphere seriously, and in some cases almost completely deficient in oxygen.

METHYLENE BLUE AS ANTIDOTE FOR CYANIDE AND CARBON MONOXIDE POISONING

According to J. C. Geiger (Journal of the American Medical Association, Dec. 3, 1932, p. 1944) and Matilda M. Brooks (Journal of the American Medical Association, Jan. 7, 1933, p. 59), methylene blue (methylthionine chloride, U.S. Pharmacopoeia) has proved a successful antidote in cases of cyanide poisoning.

These writers also suggest that the dye may be useful in cases of carbon monoxide poisoning.

Dr. Geiger cites four cases of cyanide poisoning—three in which methylene blue was not administered, all terminating fatally; and one in which 50 cubic centimeters of a 1 percent sterile aqueous solution of methylene blue was administered intravenously, resulting in complete recovery in 15 minutes.

Dr. Brooks, likewise, mentions a case of cyanide poisoning which was successfully treated by the methylene blue method.

A STANDARD CLASSIFIED NOMENCLATURE OF DISEASE

Since March 22, 1928, various national and governmental organizations, including the Medical Department of the Navy, have been collaborating through the National Conference on Nomenclature of Disease, which they created, in the preparation of a standard national nomenclature of disease.

The first complete draft of the Standard Classified Nomenclature of Disease was approved in 1931, and a preliminary trial edition was issued in April 1932 for the purpose of detecting and correcting errors and omissions.

The official edition was released in January 1933 under the title A Standard Classified Nomenclature of Disease.

As compared with previous nomenclatures, it is stated that "this book is much more inclusive and takes into systematic account, as most other nomenclatures do not, both the etiology of the disease and the part of the body affected. It is the first classified nomenclature to be developed by national rather than local interests."

Twenty-two national organizations, including the American Medical Association, American College of Surgeons, and the Association of American Physicians, have approved either the whole nomenclature or the sections with which they are particularly concerned.

ACTUATION OF THE INERT DIAPHRAGM BY A GRAVITY METHOD

Under the title above, Frank C. Eve, in the Lancet for November 5, 1932, describes what appears to be a very practical method of artificial respiration.

The method consists of laying the patient on a stretcher, which is pivoted about its middle on a trestle, and rocking up and down rhythmically so that the weight of the viscera pushes the flaccid diaphragm alternately up and down.

Experiments on a normal subject showed 1,500 cubic centimeters of air expired when the rocking stretcher method was used, as compared with 1,000 cubic centimeters with Schafer's method.

Successful results with the method were obtained in a case of post diphtheritic diaphragmatic paralysis and in a case of Landry's paralysis.

For respiratory failure in spinal anesthesia by the heavier fluids, the author suggests "that the rockings need be only 5 or 6 per minute, of which 8 to 10 seconds should be in the feet-down position and only 2 seconds in the head-down posture. Thus gravity should help to keep the heavy fluid away from the medulla. Presumably Schafer's method would be tried first, and the rocking apparatus only if prolonged resuscitation proved necessary."

The author also states "If used with the patient face downwards, it offers, in my opinion, many important advantages over the ordinary methods in use for resuscitation after drowning or gassing or anesthetics and the like."

REDUCTION OF DISLOCATED SHOULDER: A NEW METHOD

Lt. (Jr.Gr.) J. W. Shumate, Medical Corps, United States Navy (Journal of the American Medical Association of December 24, 1932), reports a new method for the reduction of a dislocated shoulder. The method is as follows:

Several thicknesses of 3-inch gauze roller bandage are passed over the left shoulder and under the right arm of the operator and then about the semiflexed arm of the patient at the elbow. The hands of the operator are then placed against the chest wall of the patient in such a manner that the thumbs can palpate the head of the humerus. Traction is afforded by the operator leaning backward strongly and pushing with his hands against the chest wall of the patient, while, with the thumbs, the head of the humerus is pushed into its socket. Care should be taken to keep the bandage short enough so that the arm of the patient is held against the chest of the operator, that sufficient traction may be obtained. Morphine not only lessens the pain of the operation but is of primary importance in reducing the tension of the shoulder muscles. He is of the opinion that this

method of reduction possesses many advantages over those commonly employed, chief of which are:

1. It is much less painful and more effective than the method of Kocher.

2. It is simplier and easier than the traction method advocated by Wilson and Cochran and requires only one operator.

3. It is much more effective than the commonly used "foot in armpit" method and is safer in that there is less danger of injury to the soft parts.

According to the author, in approximately eight cases reduced in this manner, no difficulty was experienced and each dislocation was reduced easily, quickly and with minimum pain to the patient.

FUMIGATION OF SHOES WITH FORMALDEHYDE AS A MEANS OF TREATMENT IN FUNGOUS INFECTION OF THE FEET

Writing on this subject in the October 1932 number of the Archives of Dermatology and Syphilology, Yandell Henderson concludes that—

> When shoes are left for from 8 to 60 hours in a closed tin box containing a small dish of formaldehyde, the vapor effects sterilization even at room temperature. Leather absorbs considerable amounts of formaldehyde vapor which it gives off again for many hours afterward. When shoes so treated during the night are worn during the day, a distinct amelioration or disappearance of infection of the skin may result after a time. Incidentally, the feet are also protected from reinfection from the shoes.

Two cases are cited in which the fungous infection cleared up completely.

BIOPSY IN BREAST TUMORS

The following editorial entitled "Biopsy in Mammary Cancer", by Dr. James Ewing, appeared in the January 1933 number of the bulletin of the American Society for the Control of Cancer.

The extent and severity of the radical operation of mammary cancer calls for a positive diagnosis in every case. Since women are now coming earlier for diagnosis of mammary disease, and often before the characteristic clinical symptoms of established cancer have developed, the diagnosis of these conditions has become more difficult and biopsies are more frequently required.

The practice of removing apparently benign nodules from the breast in a doctor's office and waiting 2 or 3 days for a report from a distant pathologist often leads to serious situations, and, in the opinion of some surgeons, may imperil the patient's chances for a cure even by a radical operation. The medical trauma from such a biopsy may well dislodge cancer cells and cut across and loosen

cancerous lymphatics, while the delay of some days gives opportunity for the dislodged cells to reach the distant lymph nodes. The hyperemia of the inflammatory process may also stimulate tumor growth and facilitate the local growth and even the dislodgement of more active tumor cells. There have been some observations which indicate that these undesirable events actually occur and it is reasonable to assume that they do occur. Therefore the conservative surgeon will not remove a tumor nodule from the breast except in a surgical operating room where he is prepared to have an immediate diagnosis made and the proper operation performed at the same time.

There is a difference of opinion regarding the best method of performing the operation for a biopsy of the breast. Some surgeons prefer to cut directly into the tumor, make the diagnosis on the gross appearance which is usually specific, or cut out a piece of the tumor for frozen section. If the tumor proves to be cancer, the wound is closed over a sponge soaked in 10 percent formalin. They then discard the instruments and gloves used in the exploration, prepare the skin anew, and proceed with the operation indicated. This is a very direct and expeditious method. It avoids much trauma inevitable in a local excision which requires cutting on all sides of the tumor nodule. In the case of bulky tumors it may be the best method.

In the case of small tumors I think it is safer to remove the whole tumor, together with a wide area of normal breast tissue, using extreme care not to squeeze or roughly handle the cancerous mass. This procedure avoids cutting into cancerous tissue, and if it is done with extreme care not to squeeze the tumor, cancer cells should not be dislodged.

An experienced surgeon or pathologist should be able to recognize the great majority of malignant tumors of the breast by gross examination of the cut surface of the tumor. Unless he can do this it is obvious that the tissue chosen for microscopic section may not contain the malignant tumor. Therefore great importance attaches to the gross diagnosis, which should be relied upon wherever possible. The extent of the disease also can be told only by gross examination. The cicatricial character, resistance, opacity or translucency, and the chalky streaks of carcinoma are generally specific. Frozen section is therefore often unnecessary but should be made in all cases which are in any respect doubtful to the particular surgeon or pathologist concerned. This diagnosis should be made at the operation and the appropriate procedure carried out immediately.

There are some lesions in the breast in which it is difficult for any surgeon or pathologist to state positively whether the condition is malignant or benign. Hence the surgeon must not assume that

by obtaining a microscopic diagnosis he has secured positive information. In such cases the clinical data, age of patient, extent and duration of the disease, condition of lymph nodes, and especially the gross characters of the lesion should be given much importance in the decision. Under these circumstances some surgeons would err on the side of caution and perform the radical operation. I believe it is unfair to the patient to perform a radical mastectomy unless the diagnosis of carcinoma is positive. There are many precancerous and suspicious lesions in the breast which are clinically benign, while a true carcinoma is nearly always obvious to a pathologist of adequate experience. When a substantial doubt exists about the nature of a microscopic section of a breast tumor, it is generally not cancer.

THE DIVISION OF PREVENTIVE MEDICINE

O. J. MINK, Captain, Medical Corps, United States Navy, in charge

MOSQUITO CONTROL IN HAITI[1]

It has been stated that there is no definite rainy season in Haiti. For the country as a whole this is true, but during the calendar years 1931 and 1932, the rainy season has been quite sharply defined in the vicinity of Port au Prince. Starting in April and terminating in December, the rainfall has been unusually heavy during the 2-year period mentioned above. For this reason, mosquito control, always a very important part of any public health program in the tropics, has required an extraordinary amount of attention, work and materials.

Mosquitoes, larvae or adults, found in or near Port au Prince during the year are listed below with a brief statement of their habitat as noted by Dyar (1928) and places in which they have been found as noted by Drs. S. S. Cook and J. E. Henry, United States Navy, and others:

1. *Aedes aegypti.*—Species wholly domesticated, the larvae breeding in water in artificial containers about human dwellings. Prevalent in coastal plains of Haiti. Found in pit latrines Port au Prince, January 1931. Carries yellow fever and dengue.

2. *Aedes mediovittatus.*—Larvae occur in tree holes and occasionally in artificial receptacles. Found Berley elevation 1,900 feet, 1929, Gallois Springs, 1930, and Port au Prince, 1931 and 1932. Not a disease carrier.

3. *Aedes seapularis.*—Larvae occur in temporary rainpools. Found Bailey's Beach, 1929, Petit Goave, 1929, La Victoire, 1931, Port au Prince, 1932. Not a disease carrier.

4. *Aedes sollicitans.*—Larvae occur in the most salty shore-pools filled by high tide, storm or rain, and a brood emerges when the pools are thus filled, provided conditions are favorable. If pools dry out too soon, the larvae perish and if the tide rises high enough to admit fish the larvae are devoured. Often appear in swarms which may travel inland for 40 miles or more. Found, Plain of the Cul de Sac, 1931, Port au Prince, 1932. A great nuisance, but not a disease carrier.

[1] From the annual report of the director of the American Scientific Mission to Haiti for the fiscal year 1931–32.

5. *Aedes taeniorhynchus.*—Larvae occur in pools in coastal marshes which while not necessarily salty are not far removed from the coast. Adults may fly many miles. Where marshes are extensive may appear in swarms. Tolerated in Panama as a temporary plague. Adults soon disappear. Not a disease carrier. Found, Cayes, 1918, and Trou Camian, Cote Boeuf, and Chapel Trail, 1932.

6. *Aedes tortilis.*—Larvae occur in temporary pools. Found, Petit Goave, 1929, Marigot, 1931, Port au Prince, 1931 and 1932.

7. *Anopheles albimanus.*—Larvae occur in ground pools but not ordinarily found in dense shade. A dangerous carrier of malaria. The only known carrier of malaria in Haiti. Quite prevalent below elevation of 2,500 feet.

8. *Anopheles grabhamii.*—Larvae occur in various ground pools. Adults bite readily. Probably do not carry malaria. Found, Cayes, 1928, Port au Prince, 1931, and Petionville, 1932.

9. *Culex nigripalpus.*—Larvae live in clear ground pools of a swampy or permanent nature. One of the most abundant tropical *Culex*. Species not troublesome. Adults do not enter houses to any extent. Found, Cayes, 1928, Petit Goave, 1929, Cape Haitien, 1930, Port au Prince, 1931 and 1932.

10. *Culex quinquefasciatus.*—Larvae occur largely in the water in artificial receptacles, being only rarely found in ground pools. The species is domesticated and infests houses, biting at night. Very troublesome. In Haiti breeds especially in foul water, latrines, and septic tanks. Carries filariasis. Found Cayes, 1928; Port au Prince, 1931 and 1932.

11. *Deinocerites cancer.*—Crab-hole breeders. Not a nuisance. No economic importance. Found Port au Prince, 1929, 1931, and 1932.

12. *Psorophora pygmaea.*—Larvae occur in temporary rainpools. Cycle, egg to adult rapid; 3 to 4 days. By instinct, female deposits where water is sure to collect. Found Cayes, 1928; and Cape Haitian and Gonaives, 1930, Petit Goave, 1931; Port au Prince, 1931 and 1932.

13. *Uranotaenia sapphirina.*—Larvae occur in semipermanent ground pools such as commonly harbor anopheline larvae. In Haiti, commonly found in small streams, but at times it breeds in large pools. Full-grown larvae seldom seen. Bailey's Beach, 1929, Port au Prince, 1931.

14. *Wyeomyia mitchellii.*—Larvae occur in water in leaf bases of epiphytic Bromeliaceae. If given opportunity adults will bite man. Not a disease carrier. Found, Gallois Springs (biting adult), 1930; Quartier Morin, Aux Cayes, and Petit Goave, 1931; and Port au Prince, 1931 and 1932.

The most common anopheline as well as the carrier of malaria in Haiti is the *Anopheles albimanus*. In the abatement of the mosquito

menace or nuisance, the primary requisite is the capture and identification of adults or larvae. To do so at first will avoid unnecessary work and duplication of effort.

Certain pest mosquitoes, notably *Psorophora pygmaea* and *Aedes taeniorhynchus*, are very abundant at times on the outskirts of Port au Prince, in the flat country close to the shore and in the salt marshes. They are prolific breeders, fierce biters, and under favorable circumstances, capable of flights of 4 to 5 miles. Usually anophelines breed with or near these pest mosquitoes so that efforts against *Anopheles* affect them to some extent also.

The greatest source of these three mosquitoes in Port au Prince undoubtedly has been the Audain fields, the cane fields of the Haitian American Sugar Company, and the Cote Boeuf area—all described later. Before these areas came under control heavy flights of *P. pygmaea* and *A. taeniorhynchus* into the city occurred at intervals during the rainy season of this year. Some *Anopheles* would be seen at these times and also young anopheline larvae would appear along the down-town water front and at other favorable places inside the city limits. A knowledge of the habits of these mosquitoes and the manner of their appearance in the city led to the discovery of their main breeding grounds in the vast flat areas along the coast northwest of Port au Prince. Partial control was established there and gradually extended during the last rainy season with the result that the incidence of *anopheles* and malaria in the city gradually dropped as the rainy season advanced instead of the reverse which is usually expected. Whether the decrease will continue throughout the rainy season remains to be seen. The control and drainage projects in these areas are still incomplete but it is expected that the next year will see them finished.

A letter from Mr. W. H. W. Komp, sanitary engineer of the United States Public Health Service on duty in the Panama Canal Zone, on the habits of certain mosquitoes in Panama confirms our observations here.

He states that the flight habits of *Aedes taeniorhynchus* are well known and thoroughly established and that this mosquito will fly long distances (up to 40 miles and possibly more) from its breeding places. The Canal Zone is periodically infested, at the beginning of the wet season in the spring, with these mosquitoes in flight from down the Pacific side. The length of the life cycle from eggs to adult is so short (from 5 to 6 days) that little can be done toward control. Oil or the moist-sand paris-green mixture may be used against the larvae, however, when the source of the mosquitoes is known.

While Mr. Komp has had no personal experience with the *Psorophora pygmaea* mosquitoes, he noted while engaged in some rice-field

investigations at Stuttgart, Ark., that the *Psorophora columbiae*, a closely related species of similar habits, would fly long distances—that is, from 2 to 3 miles—into the town from newly flooded rice fields and thinks there is no reason why *pygmaea* should not do the same. It has been his experience, in general, that any mosquito which takes human blood will, if produced in excessive numbers, fly much farther than is normally considered the flight range of that particular mosquito, in order to secure a blood meal.

To a more limited extent, the same is true of *A. albimanus*. Undoubted flights of over 2 miles have been recorded in the Canal Zone and it is suspected that this insect is capable of much longer flights, up to 3 or 4 miles, if produced in sufficient numbers. It is not known, however, just how dangerous such flights are with regard to the production of epidemics of malaria, but it is thought that these mosquitoes are less dangerous than mosquitoes bred nearby; that is, the danger is in inverse proportion to the length of the flight.

A further illustration of the value of the knowledge of the habits of mosquitoes in locating breeding places and in planning drainage is found in the effect of sunlight on the larvae of *A. albimanus*. It has been stated that these larvae thrive only in water reached by sunlight. Observations in Haiti confirm this statement. It has been noted again and again while dipping along ditches, streams, and stagnations for *Anopheles* that when ditches were shaded breeding grew less and less and did not occur in the heavy shade of thickets and dense cane fields but reappeared when the streams or ditches emerged to sunlight and algae again. The deeply shaded ground of cane fields, however, should not engender a false sense of security. In several instances, Dr. J. E. Henry, United States Navy, found pools in irrigation ditches where sufficient sunlight percolated through small openings in dense cane to support the growth of algae. There were also many thriving anopheline larvae.

Old deep ditches with high shaded sides, if not obstructed and allowing free flow for storm water, could safely be left unmolested. Streams through heavy forests, vines, and undergrowth were not troublesome but where areas were cleared along such streams in getting wood or for farming there usually was trouble. This principle has been borne in mind in planning drainage, particularly in the flat country areas. Thus unnecessary work has been avoided as well as the opening up of harmless areas to sunlight conditions favorable to *Anopheles* breeding.

Last year an outbreak of malaria occurred in the south or Portail Leogane section of Port au Prince. As stated by the undersigned in the annual report of the Service d'Hygiene for the fiscal year 1930–31, the excessive rains of the spring and summer of the year 1930

rather suddenly raised the level of the subsoil water and caused the formation of large swamps and bogs in this section. In some places, land that had been dry enough for pasture was turned into impenetrable muck and mire. Under such conditions the production of *Anopheles* mosquitoes became tremendous. Within a short period of time more than 500 cases of malaria were discovered by public-health nurses working in this section.

Assuming that for each known case of malaria several other individuals would be infected and become missed or untreated cases and carriers, the outlook for Portail Leogano at the beginning of the rainy season was not good. The results of several completed filling and drainage projects together with careful control in this locality exceeded our greatest expectations and but few cases were reported. When it is remembered that the Fete of St. Aune occupied the week ending July 16, 1932, and that during this time the streets were filled both day and night with crowds which were composed of as many as 5,000 persons all without mosquito protection of any kind, these results may be described as little short of marvelous. As a matter of fact the local press called attention to previous experience which was to the effect that this fete has always been followed by an epidemic of malaria.

Conditions, however, were different in the opposite or north side of the city. On May 3, 1932, the colony of the Haitian-American Sugar Co., located just beyond the aviation field, reported dense clouds of mosquitoes some of which were later caught and identified as *P. pygmaea*. No such flights had previously been reported in this section. It is probable that the mosquitoes had increased in number during the extraordinarily wet season of the preceding year and had attained a concentration sufficient for long flights with the beginning of the rainy season this year. During the next few days specimens of these mosquitoes were captured in various sections of Port au Prince, along the Chapel Trail, and as far away as the upper part of Petionville. Anophelines undoubtedly took part in this flight, for about 2 weeks later cases of malaria began to occur among marines stationed at and near the aviation field, students at the Ecole Militaire, and the population of this part of the city.

In searching for the source of the mosquitoes, the extremely bad areas known as Belle Fleur, Belle Fleur Saline, and Chancerelle were discovered and placed under control. It was thought that these areas were the source of *Psorophora* and *Anopheles* mosquitoes which had migrated toward the city. It was also thought that the wide expanse of the tidal mud flats of Chancerelle would effectually protect the city from flights of mosquitoes from the extensive low-lying country beyond. Nevertheless, on May 13 an inspection trip

was made through the section known as Cote Boeuf. Many Anopheline larvae were found in the railroad ditch at the edge of the saline. This area was promptly controlled. At Cote Boeuf, a few ponds and some *Psorophora* larvae were noted but, in general, the ground was dry. There had been no rain for several days. The area along the shore was not visited and the real menace was missed. On July 29 an inspection trip was made along the National Railway and many Anopheline larvae found in a ditch about a quarter of a mile long on the Port au Prince edge of Brouillard estate. Control was immediately started. The same evening another flight of mosquitoes occurred into Port au Prince. On August 11, a second inspection was made of the Cote Boeuf area. The picture was entirely changed—and what a picture! Practically the entire surface of the ground was covered with a thin sheet of water and extensive ponds were numerous. Hoofprints were everywhere on the soft mud and every collection of water whether large or small was teeming with *Psorophora* larvae. The smaller collections were solid with larvae and in the larger, the larvae were so thick in spots as to make the water seem black, even from a distance, as though there was a heavy film of black crude oil on the surface. At no time has the writer ever seen larvae so thick. A hurried survey left no doubt as to the source of the two flights of mosquitoes. The cycle from egg to adult of the *P. pygmae* is only from 3 to 4 days. The larvae were nearly full grown and about to pupate. No time could be lost if another flight was to be avoided. Control was accordingly started the same afternoon with a gang of 20 men. During the afternoon Chief Pharmacist's Mate, C. E. Jones, United States Navy, who was in immediate charge, happened to walk toward the shore in his inspection of the work and discovered the large fresh-water marsh. His description is as follows: "While walking through the woods, I suddenly came out upon a large open space extending to the shore. Upon investigation I found a large fresh-water marsh fed by two streams. Most of the marsh was covered with about 6 inches of water. Walking was difficult on account of the soft mud and water. A dense growth of reeds occupied the center where the ground was so soft that water reached the top of a pair of rubber boots. Numbers of aquatic birds and animals were feeding. Hoofprints of the latter added to the problem. There were many small fish. Shallow water was alive with *Psorophora* larvae while farther out there were plenty of Anopheline larvae. The fish were eating the larvae and the birds were eating the fish." It may be stated here that tadpoles have been accredited as efficient destroyers of mosquito larvae. Experience here has been to the contrary. In many instances tadpoles and larvae of various species have been found in large numbers

in the same pool apparently living happily together. The entire Cote Boeuf area has been controlled once or twice a week as necessary, until the area dried up in December. Within 2 weeks after control started the incidence of malaria and mosquitoes in Port au Prince began to diminish. Anopheline larvae have been found in this area repeatedly but the timely application of paris green or oil prevented their development. Anopheline control was started about the same time on the Drouillard and Audain estates where the larvae were abundant.

Of great assistance in the control of open ditches which require constant supervision and maintenance is the so-called "Panama Whale." These are made of native rope with a 30-pound weight bound inside the lead end. Saturated with oil it smoothes and grooves a ditch in one operation. Sufficient oil is left to kill existing larvae. All open ditches in or near the city are so treated once each week.

Septic tanks in this city are problems. In most instances they are not true septic tanks at all but simply covered leaching cesspools which usually contain water. Because of cracks in the masonry and ill-fitting covers they are frequently found to be producing a pest type of mosquito. A temporary correction is made by the use of oil. Following this the owner is given instructions to make proper repairs. Pit latrines are a constant problem due to faulty construction. Water enters because of seepage from drainage canals in close proximity, emptying of water into them, and natural seepage where latrines are situated on the sides of hills or slopes. After observing many tests of various combinations of oil for use in latrines it has been found that a mixture of 5 percent crude cresol, 10 percent kerosene, and 85 percent Diesel engine oil atomized in the latrines is most suitable from an economical standpoint as a larvacide and deodorant.

During the year 417,939 latrines were inspected, 3,471 of which were found insanitary. Corrective measures were instituted. Conditions are not perfect today; but by oiling latrines and controlling vast areas in and near the city, pestiferous mosquitoes and malaria-carrying mosquitoes have been noticeably reduced.

Last year the complaints of individuals were too numerous to investigate, 10 to 20 in a day not being unusual. During recent months they averaged about one in 10 days. Last year there was an epidemic of malaria; this year a few scattered cases.

During the fore part of 1932, efficient control was extended to the 2-mile limit along the south shore, and north to include Chancerelle. Control has been extended to the 2-mile limit north, with ever-increasing efficiency, which today might be termed fair from Fond Chancerelle to the north.

Control gangs have been trained, large amounts of material used, but most important are the drainage projects which either dry out areas or make them more easily controllable. The Haitian-American Sugar Co. has cooperated heartily in projects within their territory. The Audain properties to the north of town are becoming more controllable through work done by this service. Cote Boeuf is under control in the area west of the National Railway from Fond Chancerelle to the 2-mile limit.

Materials used in suburban control have been oil where pestiferous mosquitoes threatened emigration into the city; or when the water was clear, 1 percent paris green mixture. Where only anopheline control was desired, 3 percent paris green in lime was used. Oil was also used in large ponds.

During the past year 13,457 gallons of mosquito oil, 435,750 pounds of paris green and sand mixture, and 4,000 pounds of paris green and lime mixture were used in mosquito control, as compared with 5,416 gallons of mosquito oil, 53,350 pounds of paris green and sand mixture used during the previous year.

The following table is an index of mosquito control in Port au Prince during the fiscal year 1931–32:

	Number of properties inspected	Properties on which mosquito larvae were found	Percent of properties inspected having larvae	Number of receptacles found containing larvae
October 1931	35,273	2,453	6.9	2,647
November 1931	35,446	2,200	6.2	2,386
December 1931	37,413	2,232	5.9	2,346
January 1932	40,257	1,958	4.8	2,119
February 1932	36,666	1,458	3.9	1,568
March 1932	38,474	1,668	4.3	1,861
April 1932	37,941	1,820	4.7	2,144
May 1932	33,945	2,023	6.1	2,440
June 1932	35,655	2,164	6.0	2,386
July 1932	34,786	2,074	5.0	2,356
August 1932	34,743	1,989	5.4	2,730
September 1932	34,829	2,116	6.0	2,896
	435,428			27,879

Drainage.—This interesting subject has occupied to a considerable extent the interest and the resources of the mission. Port au Prince and the environs offer practically every type of problem in the respect that could be desired such as the following: Rocky, sandy, pervious soil with ample grade and the same type of soil in which it is difficult to obtain sufficient grade or pitch and where the absolute limit of depth must be reached for drainage to be successfully accomplished; new rubbish fills on soft mud flats where a foundation must be provided for and drainage established; old rubbish fills resembling a wet sponge where water tends to drain in most unusual direc-

tions; vast areas in which the problem is complicated by irrigation projects; areas of occasional springs; and areas where springs flow the year round.

There are two types of drains, namely, surface and subsurface drains. These may be further subdivided as follows:

Surface drains:

(*a*) Surface ditches.
(*b*) Sectional concrete ditches.
(*c*) Fixed or massive concrete ditches.

Subsurface drains:

(*a*) Rock drains.
(*b*) Cigarette drains.
(*c*) Tubular drains.
1. Metallic pipe.
2. Glazed tile.
3. Wooden pipes.
4. Unglazed tile.
5. Cement tile.

Double-deck drains (combination of above two types).

Surface drains.—Surface drains entail a minimum amount of excavation and are open to inspection at all times. If not properly and constantly maintained, pools may be formed by slight obstructions and produce mosquitoes.

(*a*) Surface ditches constitute the most inexpensive and at the same time most temporary form of drainage. They may be shallow or deep. Initial cost is low but upkeep is expensive. These are simply ditches excavated on the surface of the ground. They may be readily installed, changed, or filled to suit the needs of the moment. They are largely employed in rural projects, to drain temporary pools, and to study problems before installing more costly types of drainage.

(*b*) Sectional concrete drains, when properly installed, require less upkeep than surface ditches but are more expensive. They cost as much or more than fixed concrete drains but are removable. Sectional drains are used in projects that have been partially or thoroughly solved; where fixed drains cannot be properly constructed with available labor; and especially where the supporting ground is soft or liable to permit settling. In one instance, a massive fixed drain built by private interests has settled so much in the central section that it collects rather than drains water. Sectional drains permit water to enter at the joints and unless cemented, water or other contained material may leak into the surrounding ground. If contained liquids are objectionable, a nuisance may be created.

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(*c*) Fixed or massive concrete drains are costly to install but maintenance is low. This type should be constructed only on a solid foundation and after mature study. When once built they obviously cannot ordinarily be changed or moved.

Subsurface drains.—Subsurface drains are practical only when the conducting material is at least 30 inches below the surface. Maintenance is practically nil and, if properly constructed, they never produce mosquitoes. They are useful in intercepting springs and subsurface streams. When near the surface they drain water rapidly from above and a short distance on either side and may act to some extent as intercepting ditches. Deep drains are costly to install and drain surface water more slowly than the above. When once constructed, however, they are less liable to become obstructed by root growth, dry the ground to a greater distance on either side, lower the ground water to a greater extent, are less liable to suffer injuries, and are much more efficient as intercepting ditches, which brings to mind the dictum of Sir Malcolm Watson, "To drain an acre of land dig the ditch around it and not through it."

(*a*) Rock drains are simply deep ditches partially or completely filled with rock. An excellent method is to place rock or large gravel on the bottom of the ditch to a depth of 1 or 2 feet, then a layer of rubbish, grass, branches, or similar material and then fill the ditch with earth. In some instances abundant supplies of rock may be at hand while tile may be difficult to obtain. Rock drains function perfectly when they are short or not over 100 feet in length. A considerable pitch, however, is required.

(*b*) Cigarette drains, so herein termed, are similar to rock drains except that a bundle of sticks or timbers is encased end to end in the center of the rock fill. The same considerations apply as for rock drains.

(*c*) Tubular drains, if constructed of metallic pipe or glazed tile are used to remove obnoxious material, have sealed joints and do not drain the ground they are imbedded in. Unglazed or cement tile on the other hand, not only conducts water but drains the ground they pass through. Closed at the upper end, it is not possible for debris to be carried into them. They are constructed in the same manner as cigarette drains, the tile taking the place of the wood. Wooden pipes or tubes, if and when available, may be used in place of tile. With a slight pitch, tubular drains may be extended for great distances. In one instance, a cement tile drain 2,000 feet in length, is functioning as perfectly as when first constructed some 6 months ago. Manholes should be constructed at intervals to permit inspection and cleaning if necessary. Columns of rock may also be constructed at intervals to permit the easy ingress of surface water. Silt may, however, be

introduced in this manner. Rock columns are not successful when used in connection with household wastes as the rock soon becomes coated and clogged with a gelatinous substance similar to that which occurs in the sprinkling filter beds of sewage purification works.

Double-deck drains.—Double-deck drains are combinations of surface and subsurface drains. They are useful in spring areas to provide for the removal of storm water and at the same time lower the level of ground water. Practically the only disadvantage is the deposit of silt in the tile. This is especially liable to occur if the tile are not deeply placed or porous material is interposed between the tile and the surface.

Discussion.—In drainage installed by or under supervision of the mission during the year under review all of the above varieties of drains have been used except the wooden tubular type. Having in mind the close connection between drainage and mosquito control and particularly the control of the anopheline or malaria-carrying mosquito, the objective has been to make the latter as nearly automatic or as easy as possible. This has been accomplished by installations which will readily permit mosquito larvae and pupae to be washed out to sea or into the larger and more permanent ditches, where they are speedily assimilated by the fish.

Whenever practical, and especially within the city limits, the closed subsurface drain has been the type used by preference for the following reasons: It is the most desirable; interferes with nothing; leaves the surface intact and dry; and, if not disturbed, constitutes as permanent an improvement as anything in tropical sanitation can be. Closed drains, to be permanent, however, must be placed at least 30 inches below the surface and have a minimum pitch of 0.5 percent. A firm foundation of rock should be placed in the bottom of the ditch, then concrete tile embedded in one foot of large gravel up to 3 inches in size over which a layer about 6 inches thick of grass, reeds, branches or trash is placed and the ditch filled with earth. Ordinarily, tile having a diameter of 6 inches are preferred as they cost but little more than smaller sizes and are much more efficient. Such a drain should last for years. The layer of grass or trash prevents loose earth from being carried into the tile with the first rains and lasts until after the earth becomes firm. If sufficient depth cannot be secured it is better to construct open ditches. Tile by the hundreds were laid from 6 to 12 inches below the surface in the Martissant during previous years. When taken up in 1930 and 1931 they were found to be plugged solidly with earth and roots and of course were of no value for the purpose originally intended. They were not deep enough to intercept water and probably were closed soon after being laid. A transit, a cheap one will do, should be used

in this work as grades and depth cannot be accurately judged otherwise. Some of the tile placed at a depth of 6 inches were found in an area where a depth of 4 to 6 feet has since been obtained. It is also a saving of labor to know just how deep to dig. Otherwise it is necessary to construct the ditches and wait for a rain to know whether or not water will flow before placing the tile.

Surface ditches are desirable if water is to be conducted a long distance and if the contour of the ground prohibits a depth below root growth and also in any case as a temporary measure to effect immediate drainage with a view perhaps of converting them later into closed drains. They should have at least a 1 percent pitch to be really effective, but in very flat country this may be difficult or even impossible to attain. In such instances a very slight pitch is better than nothing if the ditches will remove the bulk of the water and permit the surrounding ground to dry. Some stagnant water in a ditch is an improvement over a stagnant area and much easier to control.

Open ditches require constant supervision, maintenance, and control. Drainage and mosquito control are intimately associated. The latter is described in a separate section. It may be stated here, however, that the implement known as the "Panama whale" is of great aid in the care of ditches and in mosquito control. Here the "whale" is made of native rope and when ready for use has a 30-pound weight fastened in the forward end. Saturated with oil and pulled by man power along a ditch, it smooths and grooves a ditch in one operation. Sufficient oil remains to kill existing larvae. All open ditches in or near the city are so treated once each week. Occasionally grass is trimmed from the sides, but this is of minor importance. The bottom of the ditch is kept clear of grass.

MEASLES EPIDEMIC IN GUAM, 1932

By J. B. Mears, captain, Medical Corps, United States Navy

An epidemic of measles in Guam began in June 1932, the first to occur on the island in about 5 years. Old records show that measles has been introduced at irregular intervals since 1861, when a severe epidemic developed after the visit of a trading schooner. Another epidemic of a milder type developed in 1888, following the visit of a Spanish steamer from Manila. The next epidemic of importance was introduced in April 1913 by an Army transport, and continued until October 1913. This epidemic apparently was of a mild type and low death rate, but the infection was widespread, with about 56 percent of the native population developing the disease, and there were 6,887 known cases. Infected transports again introduced the

disease in 1920, 1921, 1923, 1924, 1925, 1926, and 1927. Except for the years 1924 and 1927, the infections apparently did not spread beyond the transport passengers.

The 1932 epidemic began as a mild type of infection, but later developed into a more virulent form, the virulence becoming attenuated toward the end of the epidemic. The exact source of infection is unknown, as all ships entering port had given clean bills of health. Pratique was granted to transports upon certificates of the medical officers that there were no contagious diseases on board. The crews and passengers of all ships entering port without a medical officer were examined by the quarantine officer before granting pratique, and in no case was measles suspected until actual cases began to develop in Agana in June 1932.

On April 28, 1932, a native with dengue was isolated at the hospital as a measles suspect. He was discharged as negative on May 14, and no suspects developed in his contacts. On June 14 a native child was admitted to the hospital from the San Antonio district of Agana with a mild case of measles. The father stated that another child of his family had just gotten over a similar rash of about 3 days duration. A third child later developed the disease. The father could give no clue as to the exposure of his children to measles, but stated that he had worked as a stevedore loading and unloading the U.S.S. *Chaumont* on May 26, 1932.

Quarantine of this house and section of Agana was established, but other cases of measles soon developed in the immediate neighborhood, and in July cases began to appear in other sections of Agana and in the outlying districts. New cases continued to develop until the latter part of August, when it appeared that the outbreak had reached its peak. However, the lull was only temporary, and the number of cases markedly increased during September and October in a more virulent form.

The health department was taxed for working personnel during the height of the epidemic, as parents seldom reported measles voluntarily, and many would take their children to their ranches when they knew or suspected an inspection visit from the health department. The Guam chapter of the Red Cross employed 4 graduate native nurses for public health work, and a request was made to the Navy Department for 2 additional Navy nurses for hospital duty. These were supplied from the naval hospital, Canacao. The services rendered by these extra nurses were invaluable, and with their assistance the epidemic was more readily brought under control. The two Navy nurses were returned to Canacao on November 15, and the native Red Cross nurses were discharged in November, when the epidemic had declined to such an extent that conditions could be handled by the regular station personnel.

All cases of measles in this epidemic were natives except two Americans. One of these was the child of a chief pharmacist's mate, and the other was an adult, the wife of an officer who was admitted to the hospital during the epidemic, for confinement, and developed the disease after the birth of the baby. The baby was given convalescent serum and escaped infection.

Convalescent serum was used to some extent in the beginning of the epidemic, for treatment and prophylaxis, but had to be discontinued for lack of personnel to do the work. The serum was not used in enough cases to give an accurate picture of its value, but in the cases used the result was not entirely satisfactory in either treatment or prophylaxis. Suitable serum was difficult to obtain as so many of the natives give positive Kahn reactions from yaws infection. Most Chamorros have large families and live in small houses in close contact, and with their habits and mode of living, quarantine was difficult to enforce and was of little value. Anemia, malnutrition, ascaris, and hookworm infection no doubt helped the mortality rate, but otitis media and broncho-pneumonia were the principal complications and the latter was the principal cause of death.

Very few adults contracted the disease, and it would appear that they were immune from some previous epidemic, as most of them gave a history of measles in 1924. Children in the age group from 1 to 5 years were attacked most, next from 5 to 12 years, comparatively few from 12 to 16 years, and only occasionally was a case found over 16 years old. The oldest case was a man 38 years of age. There were only two cases to develop in the service personnel; both of these were natives in the insular force.

The total number of known cases of measles on the island from the beginning of the epidemic to December 31, 1932, was 2,054, and the number of deaths for the same period was 152. This is an annual admission rate per 1,000 for the period of 106.42 and the annual death rate per 1,000 was 7.87. There were 528 measles patients admitted to the hospital during the epidemic, with a hospital mortality of 58 or 11 percent of those admitted. This is a higher per centage of deaths than occurred on the outside. This was due to many cases being brought in in a moribund condition and to the development of complications prior to admission, so that on the whole the severer cases were admitted to the contagious ward. There were 1,526 cases that were not admitted to the hospital, with 94 deaths, giving 6 percent death rate. Total percentage of deaths to total cases was 00.74.

In previous epidemics of measles on the island all schools were closed in an effort to control the outbreak, but apparently without much success, as contact with those most susceptible to the disease was lost immediately. During the present epidemic all schools were

allowed to continue as usual, and with the pupils under daily observation, cases of measles were more easily found than they otherwise would have been. All schools furnished the health department with a list daily of all absentees and these were visited at once to determine the cause. Nurses visited the schools daily and worked with the school teachers in segregating active or suspicious cases. It is believed that this system was better than the closing of schools for an indefinite period, as actual cases and contacts were known at once and a better check was possible on children of preschool age than otherwise would have been.

The epidemic is now apparently over, only sporadic cases occurring during the last month; however, an occasional sporadic case may be expected for awhile longer.

ROAD ACCIDENTS

The April issue of the United States Naval Medical Bulletin contained an analysis of automobile accidents among the personnel of the battle force. The author pointed to the striking correlation between the age of the driver and the percentage of accidents. In this connection the following study of this class of injuries in Great Britain is of special interest. The report is taken from the Journal of the American Medical Association, December 24, 1932:

> The National "Safety First" Association in cooperation with the Ministry of Transport is conducting a research into the circumstances of road accidents. During the 2 months July and August of the present year there were in England and Wales 1,017 fatal road accidents, in which 1,059 persons were killed—an average of 17 a day. Less than 2 percent of the accidents are classed as unavoidable, and over 85 percent are ascribed to human failure, or errors of judgment, as distinct from road or vehicle defects or other causes. Pedestrians were the victims in 38 percent, as compared with over 50 percent in the corresponding 2 months of 1929. A classification of the persons killed shows that 453 were motorists, 406 pedestrians, 193 pedal cyclists, and 7 horse users. Most persons were killed on Saturdays, for which the average was 24. Over 70 percent of the deaths occurred in cities or built-up areas, and 29 percent in the open country. Greater London had one fifth of the total number of accidents. Half of the motor drivers involved had over 5 years' experience. Two thirds were between the ages of 16 and 19, which has given rise to the criticism that these boys should not drive. One eighth of the accidents occurred on road bends and one fourth at road junctions. For children the most dangerous age is between 4 and 5 years, and the casualties are almost entirely due to running into the roadway. For children between 13 and 16 the accidents are almost all due to cycling. In children, precipitate action is the principal cause of the deaths. Pedestrians might be expected to be safe on the footpath, but 24 fatal accidents occurred on the pathway or the verge of it, including cases in which some part of the vehicle projected over the path. These accidents resulted in 26 deaths, of which 21 occurred to pedestrians.

CAUSES OF REJECTION IN RECRUITS 50 YEARS AGO

It is believed that some interest may attach to a comparison of the causes of rejection in recruits in 2 years separated by a period of half a century. In comparing the most numerous physical disabilities leading to rejection in 1881 and 1931, only those disabilities causing at least 1 percent of rejections have been listed. Many of the differences are of great interest. The fact that hernia was three times as prevalent in 1881 is easily explainable when we realize that the Bassini's operation appeared about 1888.

1881		1931	
Recruits examined	8, 807	Recruits examined	149, 512
Recruits rejected physically	2, 750	Recruits rejected physically	51, 275
Percent of recruits rejected physically	31	Percent of recruits rejected physically	34
CAUSE OF REJECTION		CAUSE OF REJECTION	
Visus deterior	418	Defective teeth	10, 420
Debilitas	309	Errors of refraction	4, 125
Morbi valvularum cordis	153	Flat foot	3, 688
Caries dentium	148	Defective physical development	2, 560
Haemorrhoids	148	Underweight	2, 375
Varix	118	Color blindness	2, 118
Syphilis	115	Heart affections	1, 735
Statura corporis defecta	96	Deformities	1, 479
Mensura thoracis defecta	90	Abnormalities of teeth	1, 327
Hernia	84	Varicocele	1, 146
Omnes mensurae defectae	90	Pyorrhea	1, 064
Morbo pulmonale praedispositio	72	Skin diseases	720
Achromatopsia	69	Tonsillitis	634
Myopia	69	Curvature of spine	572
Gonorrhea	65	Underheight	533
Tonsillitis chronica	53	Hernia	511
Palpitatio	49	Defective hearing	489
Phimosis	40	Overweight	404
Erupto cutis	36		
Cor irritabile	32		
Deformitas thoracis	29		
Alcoholismus	27		
Catarrhus nasalis	27		

HEALTH OF THE NAVY

The general admission rate, all causes, based on returns for October, November, and December 1932 was 556 per 1,000 per annum, as compared with 565, the rate for the corresponding months of 1931. The median rate for the fourth quarter, as indicated by the records of the preceding 5 years, is 557. The admission rate from disease was 502 per 1,000 per annum. This rate is 8.6 percent lower than the

rate for the preceding quarter, but slightly greater than the expected rate, 483. The admission rate from accidental injuries was 54 per 1,000 The median rate for the corresponding quarter of the preceding 5 years is 61.

The incidence of acute respiratory diseases was a little greater than the experience of the corresponding months of the last 3 years. This was due largely to the increased numbers of cases notified as catarrhal fever among naval personnel ashore. Influenza and catarrhal fever were occurring in epidemic form in many cities in the United States during November and December which probably influenced to some extent the general prevalence and spread of these diseases in the Navy. A total of 1,008 cases of catarrhal fever and 136 cases of influenza were reported by shore stations in the United States during the quarter, of which 613 and 103 cases, respectively, were notified in December. The United States naval training station, Norfolk, Va., reported 343 cases of catarrhal fever during the quarter. There were 21 cases in October. In November, a few scattered cases appeared throughout the month, but on the 28th, 15 cases suddenly appeared. New cases were admitted at the rate of about 15 to 30 per day until the 13th day of December, when the number of cases rapidly diminished. This disease started among the recruits and gradually spread until all activities on the station were involved. The United States naval training station, San Diego, Calif., notified 29 cases of catarrhal fever in October, 97 in November, and 72 in December. The United States naval training station, Great Lakes, Ill., reported 7 cases of catarrhal fever in October, 49 in November, and 33 in December. The medical officer of this station stated that the slightly more than seasonal incidence of this disease occurring the last few days of November, was no doubt due to variable weather conditions. The United States naval training station, Newport, R.I., reported satisfactory health conditions. Only 41 cases of catarrhal fever occurred at this station during the quarter. Other shore stations in the United States reporting 25 or more cases were as follows: air station, Anacostia, D.C., 51; air station, Norfolk, Va., 39; navy yard, Washington, D.C., 29; air station, Pensacola, Fla., 26; Marine Corps base, San Diego, Calif., 26; and Marine Barracks, Washington, D.C., 25.

The admission rate, all causes, for forces afloat was 481 per 1,000 per annum. The median rate for the fourth quarter of the preceding 5 years is 524. There were 1,213 cases of catarrhal fever reported by all ships of the Navy during October, November, and December. This was a 58 percent decrease from the number of cases notified for the preceding quarter, but the total number of cases reported is about normal expectancy for this season of the year. However, small outbreaks occurred on board a few of the

ships during December. Ships reporting 25 or more cases during the quarter are as follows: U.S.S. *Texas*, 65; U.S.S. *West Virginia*, 51; U.S.S. *Lexington*, 34; U.S.S. *Pensacola*, 32; U.S.S. *Dobbin*, 30; U.S.S. *Mississippi*, 30; U.S.S. *Maryland*, 28; and the U.S.S. *Idaho*, 25. The U.S.S. *New Mexico* reported 45 cases of influenza during December, and the U.S.S. *Detroit*, 41 cases in October.

Three cases of typhoid fever were reported by ships in Asiatic waters during the quarter. The U.S.S. *Guam* received a case in September from the U.S.S. *Oahu* with "diagnosis undetermined." Diagnosis of "typhoid fever" was established November 10. The health record states that the source of infection was probably in Nanking, China, where the patient had been on liberty during the time of exposure. He had received a course of staight typhoid prophylaxis in September and November of 1931. The case was moderately severe and the patient returned to duty the last of November. The U.S.S. *Luzon* reported one case of typhoid fever in December. The history of this case is not available. The third case, reported by the U.S.S. *Rochester*, was fatal. The probable place of infection was Shanghai, China. Death occurred from intra-abdominal hemorrhage on the twenty-fifth day of the disease. The patient received a full course of typhoid prophylaxis 2 years and 3 months before death occurred. The U.S.S. *Rochester* also notified a fatal case of cerebrospinal fever in October. The case was originally admitted from the U.S.S. *Black Hawk*. Death occurred in the Shanghai Municipal Isolation Hospital, Shanghai, China, 14 days after onset of the disease. Two cases of scarlet fever were reported by the U.S.S. *California* in November, and one in December. The U.S.S. *Richmond* and the U.S.S. *Brazos* notified one case each in December. The U.S.S. *Henderson* reported receiving a patient with acute anterior poliomyelitis from expeditionary forces, Nicaragua, and transferring him to the Marine Barracks, Quantico, Va., in December. He was returned to duty after 59 sick days from the first date of admission to the sick list.

TABLE No. 1.—*Summary of morbidity in the United States Navy and Marine Corps for the quarter ended Dec. 31, 1932*

	Forces afloat	Forces ashore	Marine Corps	Entire Navy
Average strength	70,850	38,997	16,691	109,847
All causes:				
Number of admissions	8,514	6,755	2,810	15,269
Annual rate per 1,000	480.68	692.87	673.42	556.01
Disease only:				
Number of admissions	7,655	6,134	2,594	13,789
Annual rate per 1,000	432.18	629.18	621.65	502.12
Communicable diseases, exclusive of venereal disease:				
Number of admissions	2,238	2,897	986	5,135
Annual rate per 1,000	126.35	297.15	236.30	186.99
Venereal diseases:				
Number of admissions	2,602	931	695	3,533
Annual rate per 1,000	146.90	95.49	166.56	128.65

TABLE No. 1.—*Summary of morbidity in the United States Navy and Marine Corps for the quarter ended Dec. 31, 1932*—Continued

	Forces afloat	Forces ashore	Marine Corps	Entire Navy
Average strength	70,850	38,997	16,691	109,847
Injuries:				
Number of admissions	858	618	215	1,476
Annual rate per 1,000	48.44	63.39	51.52	53.75
Poisonings:				
Number of admissions	1	3	1	4
Annual rate per 1,000	0.06	0.31	0.24	0.15

TABLE No. 2.—*Deaths reported, entire Navy, during the quarter ended Dec. 31, 1932*

		Navy			Marine Corps			
		Officers	Midshipmen	Men	Officers	Men	Nurse Corps	Total
Average strength		9,378	1,779	81,503	1,181	15,510	496	109,847
CAUSE—DISEASE								
Primary	Secondary or contributory							
Alcoholism:								
Acute	None					1		1
Chronic	do			1				1
Aneurysm, abdominal aorta	Hemorrhage intraabdominal.				1			1
Aneurysm, aortic arch	None	1						1
Angina, Ludwig's	Septicemia			1				1
Appendicitis, acute	Peritonitis, general, acute			1		1		2
Carcinoma:								
Bladder	Pneumonia, broncho-			1				1
Caecum	Peritonitis, general, acute			1				1
Kidney and ureter	None	1						1
Lung	Hemorrhage, lateral ventricles.			1				1
Neck	Metastases, viscera				1			1
Pancreas	Hemorrhage, stomach			1				1
Cerebrospinal fever	None			1				1
Dysentery, amoebic	Abscess, amoebic, liver			1				1
Encephalitis, acute, hemorrhagic.	Psychosis, unclassified			1				1
Endocarditis, acute, ulcerative (malignant).	None			1				1
Endarteritis, coronary artery	Pneumonia, lobar			1				1
Gonococcus infection, endocardium.	None			1				1
Hemorrhage, cerebral	Arteriosclerosis, general			1				1
Influenza	Pneumonia, broncho-			2				2
Nephritis, chronic	None			1				1
Pneumonia, lobar	Abscess, lung					1		1
Do	Pleurisy, suppurative			1				1
Sarcoma, sacro iliac	None	1						1
Sinusitis:								
Frontal	Meningitis, cerebral	1		1				2
Frontal and ethmoidal	Abscess, brain			1				1
Syphilis	Encephalitis, acute			1				1
Do	Poisoning, neoarsphenamine, acute.			1				1
Teratoma, testicle	Metastasis, lymph nodes, abdominal and lungs.			1				1
Thrombosis, coronary artery	None	1		1				2
Do	Angina, pectoris	1						1
Do	Arteriosclerosis general	1						1
Do	Heart Block	1						1
Do	Myocarditis, chronic			2	1			3
Tuberculosis, chronic, pulmonary.	None			2				2
Do	Pneumothorax			1				1
Do	Tuberculosis, meninges			1				1

TABLE NO. 2.—*Deaths reported, entire Navy, during the quarter ended Dec. 31, 1932*—Continued

		Navy			Marine Corps		Nurse Corps	Total
		Officers	Midshipmen	Men	Officers	Men		
Average strength		9,378	1,779	81,503	1,181	15,510	496	109,847
CAUSE—DISEASE—Continued								
Primary	Secondary or contributory							
Tuberculosis, Pulmonary, acute pneumonic.	None			1				1
Tuberculosis, meninges	do			1				1
Typhoid fever	do			1				1
Ulcer, stomach and duodenum.	Hemorrhage, duodenum			1				1
Total for diseases		8		34	3	3		48
CAUSE—INJURIES AND POISONING								
Avulsion, cranium	None			1				1
Burn, multiple	do			1				1
Drowning	do			8		1		9
Do	Alcoholism, acute			1				1
Electric shock, injury from	None			1				1
Fracture:								
Compound, skull	do			2				2
Do	Gangrene (B. Welchii) gas			1				1
Do	Hemorrhage, intracranial			2				2
Do	Intracranial injury			1		1		2
Do	Rupture, traumatic, liver					1		1
Simple, skull	Hemorrhage, intracranial			1		1		2
Do	Intracranial, injury	1		1				2
Intracranial injury	None			1				1
Injuries, multiple, extreme	do		1	2		1		4
Do	Pneumonia, broncho-			1				1
Rupture, traumatic, spleen	None	1				1		2
Rupture, traumatic, liver and lung.	Hemorrhage, traumatic, intrapleural.					1		1
Wound:								
Incised, neck	Hemorrhage, traumatic, jugular vein.			1				1
Gunshot:								
Brain	None	1						1
Chest	do					1		1
Poisoning, acute:								
Carbon monoxide	do			1				1
Hydrocyanic acid	do	1						1
Phenol	do			1				1
Total for injuries and poisoning		4	1	27		8		40
Grand total		12	1	61	3	11	0	88
Annual death rate per 1,000:								
All causes		5.12	2.25	2.99	10.16	2.84	0	3.20
Disease only		3.41	0	1.67	10.16	.77	0	1.75
Drowning		0	0	.44	0	.26	0	.36
Injuries		1.28	2.25	.79	0	1.81	0	.98
Poisoning		.43	0	.10	0	0	0	.11

ADMISSIONS FOR INJURIES AND POISONING, FOURTH QUARTER, 1932

The following table, indicating the frequency of occurrence of accidental injuries and poisonings in the Navy during the fourth quarter, 1932, is based upon all form F cards covering admission in those months which have reached the Bureau:

	Admissions, October, November, and December 1932	Admission rate per 100,000, per annum	Admission rate per 100,000, year 1931
INJURIES			
Connected with work or drill	572	2,083	2,433
Occurring within command but not associated with work	466	1,697	1,649
Incurred on leave or liberty or while absent without leave	438	1,595	1,603
All injuries	1,476	5,375	5,685
POISONING			
Industrial poisoning	0	0	17
Occurring within command but not connected with work	4	14	35
Associated with leave, liberty, or absence without leave	0	0	22
Poisoning, all forms	4	14	74
Total injuries and poisoning	1,480	5,389	5,759

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	
	October, November, and December 1932	Year 1931	October, November, and December 1932	Year 1931	October, November, and December 1932	Year 1931
Percent of all injuries	38.7	42.8	31.6	29.0	29.7	28.2
Percent of poisonings	0	23.4	100.0	46.8	0	29.8
Percent of total admissions, injury, and poisoning titles	38.6	42.5	31.8	29.3	29.6	28.2

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.

The following cases, selected from October, November, and December 1932 reports, are worthy of notice from the standpoint of accident prevention:

"*What price carelessness?*"—To be neglectful or heedless of danger or inattentive to what is going on about you is to be careless and carelessness exacts a price.

The following cases show how this price was paid by the Navy in time lost from duty, and by the individual in suffering and injury as a result of careless practice:

Lack of eye protection.—A seaman, first-class, was not furnished goggles while chipping paint. A flying particle lodged in one eye and he was on the sick list 3 days.

A fireman, third-class, was not furnished goggles while chipping paint. A chip lodged in the cornea of one eye and he was absent from duty 6 days.

A seaman, second-class, was not furnished goggles while chipping paint. A flying chip lodged in the cornea of one eye and he was on the sick list 7 days.

While chipping paint with air hammer a seaman, first-class, not wearing goggles, was struck in one eye with a flying particle and was on the sick list 4 days.

A seaman, second-class, was chipping paint and not wearing goggles. A flying particle lodged in one eye, causing absence from duty for 42 days, 38 of which were on a hospital ship.

An electrician's mate, third-class, using an emery wheel, neglected to use goggles provided at the wheel. A small piece of steel lodged in the cornea of one eye and he was on the sick list 2 days.

A fireman, third-class, using a lathe without goggles in violation of ship's order, was struck in one eye by a steel chip and was on the sick list 3 days.

A marine private was struck in one eye by a flying chip of steel while operating a lathe. No goggles were provided and he was on the sick list 10 days.

While working near electric welding, and not wearing protective goggles which could have been used, the reflected light from the electric arc caused an actinic ray ophthalmia in a seaman, second-class, a machinist's mate, second-class, and a fireman, second-class. They were on the sick list 7 days, 1 day, and 1 day, respectively.

A fireman, first-class, while working as helper looked at the flame of an electric torch, causing an intense photophobia. The case was reported as due to "own negligence," as he failed to wear the protective goggles which were available. Loss of time, 4 days.

Burns.—A seaman, first-class, received burns on the buttocks when he accidentally backed against an uncovered steam pipe while bathing and scrubbing clothing in a crowded washroom. He was 10 days on the sick list. This case was reported as due to "lack of safety device."

A seaman, second-class, suffered a chemical burn on the right hand when a cloth saturated with sulphuric acid was given to him to clean the knife edge of a hatch coaming. He was 26 days on the sick list.

Due to the negligence of others, a radioman, second-class, suffered a burn of the foot when he stepped into a bucket of hot water which had been carelessly left outside of the washroom by a mess cook. Loss of time 7 days.

Steam turned on in broken steam line on which a chief machinist's mate was working burned his left arm and his chest. Valves on line had not been wired and tagged. He was absent from duty for 4 days.

A gunner's mate, third-class, suffered a burn of the foot when he stepped into a bucket of hot water which someone had carelessly left in the passageway outside of the washroom. He was 17 days on the sick list.

Food-machinery hazards.—While feeding material into the hopper of a meat grinder with fingers instead of using the pushing device provided, a ship's cook, third-class, suffered a traumatic amputation of a finger due to his own negligence. Loss of time 11 days.

A fireman, second-class, pressing fruit into a meat grinder lost a finger when it was caught in the blade of the grinder. Absent from duty 36 days, 31 of which were in the hospital.

A ship's cook, third-class, used his hand to feed meat into power-driven food chopper, and part of one finger was amputated. On sick list 6 days.

While operating meat grinder, a ship's cook, first-class, lost part of one finger by amputation when his hand slipped into revolving knife. He was in the hospital 66 days.

Miscellaneous.—While descending a ladder and holding on to hatch coaming, a hatch cover which was improperly secured fell and caused compound fractures of all the fingers of an electrician's mate, third-class, and his absence from duty for 37 days.

While scrubbing paintwork near blower, a fireman, third class, struck his head against an uncovered revolving fan blade, sustaining a lacerated wound of the scalp for which he was on the sick list 1 day.

Due to his own negligence a seaman, second-class, while trying to drive a nail into a shoe with a bottle, cut the back of his hand when the bottle broke. On sick list 2 days.

STATISTICS RELATIVE TO MENTAL AND PHYSICAL QUALIFICATIONS OF RECRUITS

The following tables were constructed with figures taken from monthly reports submitted by naval training stations:

Cumulative data

	Number	Percent of recruits received	Percent of recruits reviewed
JANUARY TO DECEMBER 1931			
All naval training stations:			
Recruits received during the period	7,071		
Recruits appearing before board of medical survey	227	3.21	
Recruits recommended for discharge from the service	122	1.73	53.74
OCTOBER, NOVEMBER, AND DECEMBER 1932			
United States naval training station, Hampton Roads, Va.:			
Recruits received during the period	266		
Recruits appearing before board of medical survey	14	5.26	
Recruits recommended for discharge from the service	11	4.14	78.57

Cumulative data—Continued

	Number	Percent of recruits received	Percent of recruits reviewed
OCTOBER, NOVEMBER, AND DECEMBER 1932—Continued			
United States naval training station, Great Lakes, Ill.:			
Recruits received during the period	399		
Recruits appearing before board of medical survey	3	.75	
Recruits recommended for discharge from the service	2	.50	66.67
United States naval training station, San Diego, Calif.:			
Recruits received during the period	474		
Recruits appearing before board of medical survey	24	5.06	
Recruits recommended for discharge from the service	2	.42	8.33
United States naval training station, Newport, R.I.:			
Recruits received during the period	242		
Recruits appearing before board of medical survey	12	4.96	
Recruits recommended for discharge from the service	2	.83	16.67

The following table was prepared from reports of medical surveys in which disabilities or diseases causing the surveys were noted as existing prior to enlistment. The time which elapsed from date of enlistment to date of medical survey is noted in each case. With certain diseases, survey followed enlistment so rapidly that it would seem that many might have been eliminated in the recruiting office. The difficulty in establishing a diagnosis in nervous and mental cases is demonstrated by the time interval in the table. An exception in this group is epilepsy, which may or may not diagnose itself promptly. Certain groups, of course, present difficulties in diagnosis at the time of enlistment due to lack of equipment:

Cause of survey	Number of surveys	Number of days between enlistment and survey
Absence, acquired (incisor teeth)	1	1
Arterial hypertension	1	24
Blepharitis	1	7
Color blindness	1	8
Conjunctifitis, granular	1	6
Constitutional psychopathic inferiority	1	21
Constitutional psychopathic state, emotional instability	1	169
Do	1	114
Do	1	67
Deafness, bilateral	1	67
Deformity, acquired, left elbow	1	4
Deformity, acquired, right lower extremity	1	32
Enuresis	1	157
Epilepsy	1	276
Do	1	58
Do	1	15
Flat foot	1	32
Foreign body, both legs (no. 4 bird shot)	1	6
Gonococcus infection, urethra	1	7
Hematoma, traumatic	1	21
Hernia, inguinal, right	1	37
Do	1	2
Insufficiency, ocular muscle	1	9
Myopia, both eyes	1	4
Nephritis, chronic	1	125
Pleurisy, serofibrinous	1	11
Syphilis	1	48
Tuberculosis, pulmonary, chronic, arrested	1	149
Valvular heart disease, mitral insufficiency	1	11
Vincent's infection (oral)	1	6

VOLUME XXXI OCTOBER 1933 NUMBER 4

United States Naval Medical Bulletin

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

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

VOL. XXXI OCTOBER 1933 No. 4

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
LIEUTENANT COMMANDER LOUIS H. RODDIS
MEDICAL CORPS, U.S. NAVY, IN CHARGE

Compiled and published under the authority of Naval Appropriation Act for 1934, Approved March 3, 1933

UNITED STATES
GOVERNMENT PRINTING OFFICE
WASHINGTON : 1933

For sale by the Superintendent of Documents, Washington, D.C. - - - - - - See page II for price

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:

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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.

SUBSCRIPTION PRICE OF THE BULLETIN

Subscription should be sent to Superintendent of Documents, Government Printing Office, Washington, D.C.

Yearly subscription, beginning July 1, $1; for foreign subscriptions add 35 cents for postage.

Single numbers, domestic, 25 cents; foreign, 35 cents, which includes foreign postage.

Exchange of publications will be extended to medical and scientific organizations, societies, laboratories, and journals. Communications on this subject should be addressed to the Surgeon General, United States Navy, Washington, D.C.

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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.

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U.S. NAVAL MEDICAL BULLETIN

VOL. XXXI OCTOBER 1933 NO. 4

SPECIAL ARTICLES

COMPRESSION FRACTURE OF THE SPINE

By F. V. SUNDERLAND, Lieutenant, Medical Corps, United States Navy

Compression fracture of the spine may be defined as a fracture affecting one or more of the vertebral bodies in which impaction of the spongy bone occurs producing a characteristic wedge-shaped deformity. This deformity results when the upper and lower surfaces of the vertebra are crushed together, thus mushrooming its body, increasing its breath and lessening its height (1).

This is the most frequent of bony injuries to the spine and occurs most often in the weakest part of the spinal column, namely, the last two dorsal and the first two lumbar vertebrae which are located in the area in which the normal kyphotic curve of the dorsal vertebrae is reversed into the normal lordotic curve of the lumbar vertebrae (2). It may result from forceful hyperflexion or from a force acting in the long axis of the vertebral column when it is in a position of slight flexion. The most common causes may be listed as follows:

1. Automobile and motorcycle accidents.
2. A weight falling upon the shoulders.
3. A fall from a height landing upon the buttocks, feet, or head. A fall on the buttocks generally affects the lumbar, whereas a fall on the head affects the cervical vertebrae.
4. From practically negligible trauma such as a fall in a sitting posture.

The spinal arch generally escapes injury because it is composed of compact bone. The intervertebral disc escapes injury unless more than one vertebra is compressed. Lateral deviation of the spine occurs when one side of the fracture is higher than the other. A typical wedge-shaped deformity results because the component of force falls upon the anterior portion of the vertebral body causing it to be further compressed than the posterior portion. Complicating cord compression may arise from any one of the following factors:

1. Isolated portions of bone broken off and projecting into the spinal canal.
2. Protrusion of an intervertebral disc when the posterior ligament is torn.
3. From a bulging of the posterior portion of the injured vertebral body.

Early diagnosis is imperative because without proper treatment a traumatic spondylitis results, producing a prolonged or permanent

disability (1). We rely upon the cardinal symptomatology and repeated X-ray findings for diagnosis. The symptoms are:

1. A lame back resulting from an injury or fall.
2. *Deformity.*—Kyphosis produced by projection of the spinous process.
3. *Pain.*—Elicited by pressure on the spine of the injured vertebra, which is the spine immediately beneath the kyphosis. All movements of the spine are painful. The pain at the site of fracture is augmented by pressure upon the head or shoulders.
4. Limitation of motion of the spine.
5. Marked muscle spasm and tenderness in the region of the fracture.
6. Localization of symptoms in the midline of the spine.
7. Disturbed sensation in the region supplied by an injured nerve root aids in locating the level of the lesion. X-ray pictures should be taken in the lateral and anterior-posterior planes, the later being stereoscopic to enable thorough study. The lateral view gives the best conception of the wedge-shaped deformity resulting from compression fracture.

Compression fracture must be differentiated from two other conditions:

1. Tuberculosis of the spine without abscess formation. In Pott's disease the intervertebral disc is involved early, in compression fracture it generally escapes injury unless more than one vertebrae is involved.
2. *Hypertrophic arthritis.*—The late stage of healing in compression fracture is produced by ankylosis from the formation of large bony spurs which resemble hypertrophic arthritic changes but compression fractures have a wedge-shaped deformity. A careful history and examination are of assistance.

The diversity of treatments recommended can best be illustrated by the abstract of several papers published in the past few years. S. W. Boorstein (3), Fordham Hospital, New York, reported 108 cases of spinal fractures of which 49 were of the compression type. He prefers the conservative treatment. Operate early if there is bone pressing on the cord, otherwise do not operate on patients with fresh spinal fractures. With complete lesions of the cord an operation does no good. If the lesion is incomplete wait 2 to 3 weeks before operation. If the injury is in the cauda, operate, as its nerves can regenerate. H. E. Conwell (4), Employees Hospital, Fairfield, Ala., reported 215 cases of fracture of the spine, which included 100 cases of fracture of the vertebrae without cord injury. Conwell believes in conservative treatment. F. Crooks (5), of Nottingham, England, advocates reduction. He reported 12 cases of fracture of the spine seen several weeks after injury: good movements were restored; only 1 was free from pain and but 2 cases had returned to work. J. I. Mitchell, of Memphis (6), believes in strong hyperextension on the Rogers frame. He reported 13 cases treated in this manner; 8 were seen and treated immediately and X-rays have shown definite proof of correction of the compression deformity; the other 5 cases were seen from 1 to 2 months after injury but were able to secure partial correction.

It seems no more than natural to attempt correction of the deformity, but appliances and positions recommended to date have not proved entirely successful or satisfactory. It is conscientiously believed that conservative nonoperative or external fixation is the treatment of choice in all recent cases and that operative or internal fixation is indicated for late cases which have not received proper or adequate treatment.

Conservative treatment calls for immediate recumbent position on a firm board supported mattress or preferably a Bradford frame which prevents further deformity. Correction is thus instituted by the force of gravity and by the compensatory extension of the other vertebrae. As soon as full curve restoration at the site of injury is obtained a posterior half-shell plaster cast is applied from the neck to the mid-thigh in most cases. It is of utmost importance that this shell fit the patient snugly and firmly and thus hold the spine in the maximum correction position. The posterior shell keeps the fracture in fixation. The anterior half-shell plaster cast is made a few days later while the patient lies in his posterior or half-shell plaster cast. When the two shells are strapped together the patient may be turned over without disturbing his healing fracture. In cervical fractures apply traction to the head and later a similar plaster shell cast except that it extends up to the chin and occipital protuberance—called a Minerva type body cast. In fracture of the lumbar vertebrae with marked deformity apply 15 pounds extension to each leg. The time element is essential. Keep the patient in the recumbent position of fixation in a molded plaster shell for a period of 8 to 10 weeks. At the end of this time a plaster jacket may safely be applied and when thoroughly dry the patient may become ambulatory and the plaster jacket be left in place for 6 to 8 weeks. At the end of this period the plaster jacket is bivalved and the patient measured for a Taylor back brace or its equivalent. This Taylor back brace with crutch supports is worn for 3 to 9 months or until a period of 1 year has elapsed since injury.

Early operative treatment has been advised in which fixation by bone graft is employed. The advocates of this school believe that healing is more certain and disability time less than when treated by the conservative method only. The conservative school, however, believes that:

1. As good if not better results are obtainable by conservative treatment.
2. There is less risk to the patient.
3. That the operative treatment requires as long bed rest as the nonoperative.
4. There is a minimum area of ankylosis, whereas the operative treatment requires a more extensive ankylosis.
5. That operative treatment is for late cases which have not received adequate treatment.

The operation of choice at the United States Naval Hospital, San Diego, Calif., has been the modified Hibbs, using an osteoperiosteal graft from the tibia of sufficient length to fuse the spinous processes of the two vertebrae above and the two below to the injured spinous process and vertebra.

Report of Cases

Case 1.—L. A. M., white, age 20, admitted November 13, 1932, with contusion back. The final diagnosis was fracture, simple, compression, sixth dorsal vertebra.

Chief complaint and present illness.—Severe pain in the back in the midline at the level of the shoulder blades. The injury was sustained in an automobile accident on November 13, 1932. Shortness of breath. Sharp pains in his back when he moves but only a dull ache when he lies still. Pain in the left side radiating down to the left testicle and the left leg.

Positive physical and X-ray findings.—Marked tenderness over the spines of the fifth and sixth dorsal vertebrae. Soreness and splinting of the dorsal musculature. X-ray shows evidence of a wedge shaped compression fracture of the body of the sixth dorsal vertebra. No evidence of lateral displacement.

Treatment and complications.—Bed rest on a firm board supported mattress for 7½ weeks, then a plaster jacket. No complications.

Present status.—The patient is ambulatory wearing a bivalved plaster jacket and he is awaiting a Taylor back brace with crutch supports. He is to be recommended for medical survey out of the United States Naval Service.

Case 2.—S. E. P., white, age 43, admitted January 11, 1933, with contusion back, upper third. The final diagnosis was fracture, simple, compression, fifth dorsal vertebra.

Chief complaint and present illness.—Pain in the chest and back. The patient fell a distance of 15 feet from a tree, landing on his back across a bench. He was unconscious for several minutes. Severe lancinating pain was present in his back between the shoulder blades.

Positive physical and X-ray findings.—Tenderness elicited by palpation over the fourth, fifth, and sixth dorsal spines. No visible deformity. Muscle splinting was present in this area. X-ray shows a compression fracture of the fifth dorsal vertebra; no lateral displacement; obliteration of the intervertebral disk between the fourth and fifth dorsal vertebrae.

Treatment and complications.—Bed rest on a Bradford frame for 1 month. A posterior plaster shell cast was then applied. Routine treatment for benign tertian malaria. Benign tertian malaria with chills, fever, and malaise developed on the fifth day and later subsided under routine treatment.

Present status.—Bed patient lying in a posterior half-shell plaster cast on a Bradford frame. This patient will be recommended for a medical survey out of the United States Marine Corps Service at a later date.

Case 3.—K. W. O., white, age 29, admitted July 17, 1932, with undetermined (injury back). The final diagnosis was fracture, simple, compression, second lumber vertebra.

Chief complaint and present illness.—Severe pain and tenderness in the small of his back. The injury resulted from an automobile accident. The severe pain in the small of his back is aggravated by moving.

Positive physical and X-ray findings.—There is marked tenderness and pain elicited by palpation over the spines of the first, second, and third lumbar vertebrae. A slight palpable deformity and soreness and splinting of the musculature in this area were found on further examination. X-ray on July 18,

1932, shows a compression fracture of the second lumbar vertebra. No apparent lateral displacement. On December 22, 1932, a check plate showed slight tilting of the body of the second lumbar vertebra to the left carrying with it the body of the first as a result of an old compression fracture of the second lumbar vertebra; there is some new bone formation between the bodies of the first and second lumbar vertebrae left side.

Treatment and complications.—To bed on Bradford frame and posterior half-shell plaster body cast for 7½ weeks. Plaster jacket and then ambulatory for the next 10 weeks. Taylor back brace with crutch supports since November 23, 1932. There is a slight lateral displacement. No complaints.

Present status.—Discharged from the United States Naval Service on February 16, 1933, as the result of an approved medical survey, retained as a supernumerary patient.

CASE 4.—E. D. F., white, age 35, admitted November 22, 1932, with fracture, spine. The final diagnosis was fracture, simple, compression, first lumbar vertebra.

Chief complaint and present illness.—Pain in the small of his back. Difficulty in urination. Constipation. The injury was received on Aug. 14, 1932, in an automobile accident. The patient was treated in a civilian hospital for 1 week when he was sent home because there was "nothing wrong with him." He remained at home until November 8, 1932, when he was X rayed again at another institution and a fractured spine was diagnosed.

Positive physical and X-ray findings.—Tenderness is elicited on palpation over the spines of the first and second lumbar vertebrae. There is slight limitation of motion; no visible deformity. X-ray reading of plates on November 30, 1932 which the patient brought with him were in the anterior-posterior position only; rather marked lipping, occasional spur formation of the bodies; no plate evidence of bone injury involving the lower dorsal spine. Impression: Hypertrophic arthritis, moderate degree. On February 4, 1933, a lateral X-ray shows a slight apparent compression fracture of the first lumbar vertebra.

Treatment and complications.—To bed on Bradford frame with 15-pound extension on each leg for 8 weeks; Taylor back brace with crutch supports since that time. On admission to this hospital it was found necessary to catheterize this patient for a period of 6 days. His bowels moved only by enema during this period.

Present status.—Discharged to civil life on February 17, 1933, wearing a Taylor back brace with crutch supports which he was advised to wear for a period of 1 year. The patient was symptom free.

CASE 5.—C. D. C., white, age 23, admitted October 13, 1932, with curvature, spine (kyphoscoliosis). The final diagnosis was the same as on admission.

Chief complaint and present illness.—Stiffness in the small of his back on arising. He fractured his spine on January 1, 1932, in an automobile accident. His treatment at that time consisted of bed rest for 7 weeks and then a plaster jacket for 3 months. On June 19, 1932, he was returned to duty, where he was symptom free for a period of 1 month. Pain and stiffness lasting for 10 to 15 minutes on arising have been present for the past 2 months in the small of his back. He states that he has been able to do his duty.

Positive physical and X-ray findings.—Marked prominence of the spinous processes of the first, second, and third lumbar vertebrae. Kyphoscoliosis of the lumbar spine from an old compression fracture. X-ray on October 17, 1932, showed an old compression fracture of the first lumbar vertebra; no lateral displacement; definite bone bridge between the twelfth dorsal and first lumbar vertebrae.

Treatment and complications.—To bed with hyperextension of the spine for 5½ weeks. He was then placed in a half-shell posterior plaster cast. Operated December 8, 1932, modified Hibbs (spinal fusion) eleventh and twelfth dorsal and the first, second, and third lumbar spinous processes and vertebrae, using an osteoperiosteal graft from the tibia. No complications.

Present status.—Bed patient. A medical survey from the United States naval service has been recommended.

Summary

Conservative treatment is the treatment of choice at the United States Naval Hospital, San Diego, Calif. The Taylor back brace with crutch supports has been satisfactory and effective.

Case 1 is an example of the early ambulatory type of treatment: Bed rest on a firm mattress for 7½ weeks, and then a plaster jacket in which the patient has been comfortable and ambulatory.

Case 2 shows the etiological factor of a force acting in the long axis of the vertebral column when it is in a position of slight flexion, which is the position assumed in a fall.

Case 3 illustrates the use of a Bradford frame and a posterior half-shell plaster body cast for 7½ weeks; an ambulatory plaster jacket for 10 weeks; followed by a fitted Taylor back brace with crutch supports. The X-ray is significant of healing.

The advisability of repeated X-ray examinations when symptoms persist and of securing a picture in the lateral position is proved by case 4. During the first 10 to 14 days a slight compression fracture may not be observable.

Case 5 proves that service cases cannot be returned to duty short of 1 year without wearing a spinal brace. This patient was treated for 6 months in 1932 and returned to duty symptom free, but without wearing a spinal brace. He had no deformity when he was returned to duty but was admitted 4 months later with a marked kypho-scoliosis.

The course of healing can be followed by X-rays. When the bony bridges uniting the injured vertebra to the vertebrae above and below are present, the fracture is considered cured.

Conclusion

The early recognition of compression fracture of the spine occurring in the United States naval service is of the utmost importance. It has been observed that cases are not physically fit to be returned to active duty even after a period of 6 months' treatment; the minimum time required for a cure is 1 year. The patient could be returned to duty after a period of 6 months' treatment provided that he wore a Taylor back brace or its equivalent, but he would thus be unable to perform his duties or to dress neatly. Hence, every service case of a spinal compression fracture means that the patient must be surveyed

from the United States naval service, as he cannot be retained on a duty status for the required treatment and convalescent time, namely, 1 year.

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CARDIOSPASM

With Report of Case

By T. C. Anderson, Lieutenant Commander, Medical Corps, United States Navy, and J. L. Enyart, Lieutenant (junior grade), Medical Corps, United States Navy

Spasmodic affections comprise most of the motor disturbances of the esophagus. They occur under a variety of conditions. Tetanus, hydrophobia, epilepsy, chorea, and hysteria may be characterized by attacks of esophageal spasm. Many cases are of reflex origin. Organic disease of the esophagus itself may have superimposed a reflex spasm of the organ. Laryngeal tuberculosis, gastric disorders, and uterine disease have been recorded as reflex causes of spasm of the esophagus. Recently a case of cancer of the rectum was observed in which agonizing esophagismus was one of the chief complaints. A third group of cases is described as of psychic origin. Such patients are excitable, irritable, "nervous", and attacks of spasm of the esophagus may be precipitated by emotional upsets, such as shock, anger, fright, worry, etc. An idiopathic group must be added to include those cases where no cause is apparent. It is to this last class that cases of cardiospasm belong.

Any part or all of the esophagus may be affected. Spasmodic contraction of the entire organ is known as esophagismus, the attacks of which usually are brief in duration, subside spontaneously, but tend to recurrence. Localized contraction of the esophageal introitus at the level of the cricoid cartilage has been termed "cricoid spasm." Similar localized spasm may involve the esophagus at different levels lower down, that at the cardia, if acute, being known as spasm at the cardia; if chronic, cardiospasm.

Of these varieties, cardiospasm gives rise to the most serious esophageal obstruction. The other types are usually acute, temporary, or intermittent. They do not cause dilatation of the esophagus. Attacks may effectually be treated by eliminating psychogenic or reflex factors and passing esophageal sounds. Cardiospasm is a chronic disorder, causing marked obstruction, from the

symptoms of which the patient is never free, once the condition is fully established. A remarkable dilatation of the esophagus is a prominent feature. The passage of esophageal sounds does not relieve most of these patients. Stretching of the cardiac sphincter with a hydrostatic or pneumatic dilator is the most effective treatment.

Cardiospasm may be defined as chronic obstruction at the lower end of the esophagus, caused by tonic muscular contraction, and associated with dilatation of the organ and inflammatory changes in its mucosa.

This condition was first described by Purton in 1821. Fifty years later 17 cases were collected from the literature by Zenker and Ziemssen. The dilatation of the esophagus without organic narrowing of its lumen was described. Mikulicz suggested contraction at the cardiac sphincter as the initial change in cardiospasm and successfully treated four cases by dilating the lower esophagus from below through an opening in the stomach. Russell (1898) first treated cardiospasm by stretching the cardia with a hydrostatic dilator passed through the mouth. In 1903 Sippy devised a similar apparatus and later reported 60 cases treated and cured with his instrument. In 1908 Plummer described a hydrostatic dilator and in 1912 reported 91 cases treated with this instrument. Vinson, Friedenwald, and Verbryke have reported several hundred cases. The condition can no longer be considered rare. It should always be thought of in patients presenting symptoms of esophageal obstruction.

The cause of cardiospasm is unknown. Disturbance of the nervous regulation of the esophageal musculature is the generally accepted explanation. This disturbance has been regarded as psychogenic reflex, and neurogenic by different observers. Psychogenic factors are suggested in the histories of many patients by the prompt development of symptoms following some mental upset. In some of these cases, where relief has been obtained by dilating the cardia, recurrence of symptoms has followed further psychic trauma. Psychotherapy is not effective in relieving true cardiospasm and many cardiospasm patients give no history suggestive of psychoneurosis.

A reflex origin is suggested by the development of cardiospasm in patients suffering from disease elsewhere in the body. Verbryke has reported 23 cases of cardiospasm occurring in patients suffering from aortitis, aneurysm, and angina pectoris. Blows on the chest and epigastrium, the swallowing of irritating chemicals, and foreign bodies have been noted as exciting causes. Cardiospasm has been considered secondary to ulcer of the stomach, gall-bladder disease, and appendicitis. Removal of these conditions where this is possible seldom benefits the cardiospasm.

The neurogenic theory is based on the conception that the changes of cardiospasm are due to organic disease of the nerve supply to the

esophagus. Kraus reported degenerative changes in the vagus nerves in the post mortem examination of one case.

The initial change is a failure of the cardiac sphincter to relax its tone as part of the normal swallowing act. The increased pressure within the esophagus due to this obstruction results in a gradual dilatation of the organ. Retention of food and secretions produces inflammatory changes of the esophageal mucosa.

The most striking feature of the pathology is the dilatation of the esophagus. This is greater than occurs in any other condition. The organ commonly has a capacity 4 or 5 times the normal of about 100 cubic centimeters, and may be dilated to a capacity of 1,800 cubic centimeters. Hypertrophy of the cardiac sphincter has been described. There is no organic narrowing of the lumen or scar tissue about the cardia to account for the obstruction. The muscularis of the esophageal wall may be several times its normal thickness of about 2 millimeters.

The mucosa shows evidence of chronic inflammatory change—congestion, erosion, ulceration, and scarring.

The three cardinal symptoms of cardiospasm are pain, dysphagia, and regurgitation (Vinson). Pain, often the first symptom, may be present for a prolonged period without other evidence of the disease. It is usually localized to the region of the lower esophagus, is dull and aching, such as a normal individual may experience on swallowing a large food bolus too rapidly. The pain may radiate into the thorax, back, or neck and may be so severe as to suggest angina pectoris or gall-stone colic.

Difficulty in swallowing may be at first intermittent, the attacks becoming more frequent and severe until the patient is never free from a feeling of obstruction on swallowing. Soft foods are usually taken more readily than liquids, a symptom in sharp distinction to organic stenosis, where liquids enter the stomach more easily. Foods containing hard particles, uncooked fruits and vegetables, bread crust, give the most trouble.

Regurgitation indicates a more severe obstruction. The food is returned suddenly without nausea shortly after ingestion. As dilatation of the esophagus progresses, regurgitation occurs less frequently and the food is retained in the dilated gullet. Some of this retained material passes into the stomach. Much of it is regurgitated, either intentionally, when the patient irritates his throat with his finger, or when the esophagus is distended. Regurgitation during sleep is a common occurrence and patients learn to empty the esophagus before retiring. Aspiration of some of the regurgitated material may awaken the patient in a paroxysm of coughing. Chronic pulmonary infection is induced in many cases in this way.

Loss of weight is due to the limited amount of food the patient is able to swallow. In extreme cases, death may result from starvation. Usually a lowered weight level is maintained, the reduction depending upon the degree of obstruction.

In addition to the history, information of value in making a diagnosis may be obtained from an X-ray examination and from passing esophageal sounds.

In the examination with the X-ray, the dilated, angulated esophagus ending in a smooth funnel shape with a tip at the cardia is characteristic.

The introduction of a large esophageal sound over a silk thread, which the patient has previously swallowed, will locate the obstruction at the cardia. This may be overcome with moderate pressure and the sound passed into the stomach. The slight resistance to a large sound at the cardia in a patient suffering marked obstruction is strongly suggestive of cardiospasm.

The most common disease from which cardiospasm is to be differentiated is carcinoma at the cardia. The history in the latter is usually of shorter duration and liquids are ingested more readily than soft foods. In the roentgen examination, the constricted area is irregular and ragged in outline, and the dilatation of the esophagus less marked than in cardiospasm. The passage of a large sound encounters marked resistance at the stricture. In doubtful cases examination with the esophagoscope may be indicated.

Diverticula of the lower esophagus may cause obstruction by pressure when they become distended with food. The diagnosis depends upon outlining the rounded pouch at one side of the esophagus in the X-ray examination.

The most satisfactory results in the treatment of cardiospasm are obtained by stretching the cardia with a dilator of the Russell type. This instrument consists of a rubber bag, covered with a silk sac and attached to an introducer with a fenestrated olive tip. The silk sac is 5 inches long and 2½ inches wide. Its purpose is to preserve the cylindrical shape of the rubber bag when distended and to limit the stretching force applied to the cardia. The silk sac is constricted slightly at its middle in order to reduce the tendency of the dilator to slip out of the cardia during dilation. The upper end of the rubber bag is connected to a tube through which water or air may be forced to distend the dilator when it is in place in the lower esophagus.

The first step in the treatment is the introduction into the esophagus of a silk thread over which the drilled tip of the instrument may be passed. Such a guide reduces the danger of perforation of the esophagus and makes possible the more accurate location of the dilator in the cardia.

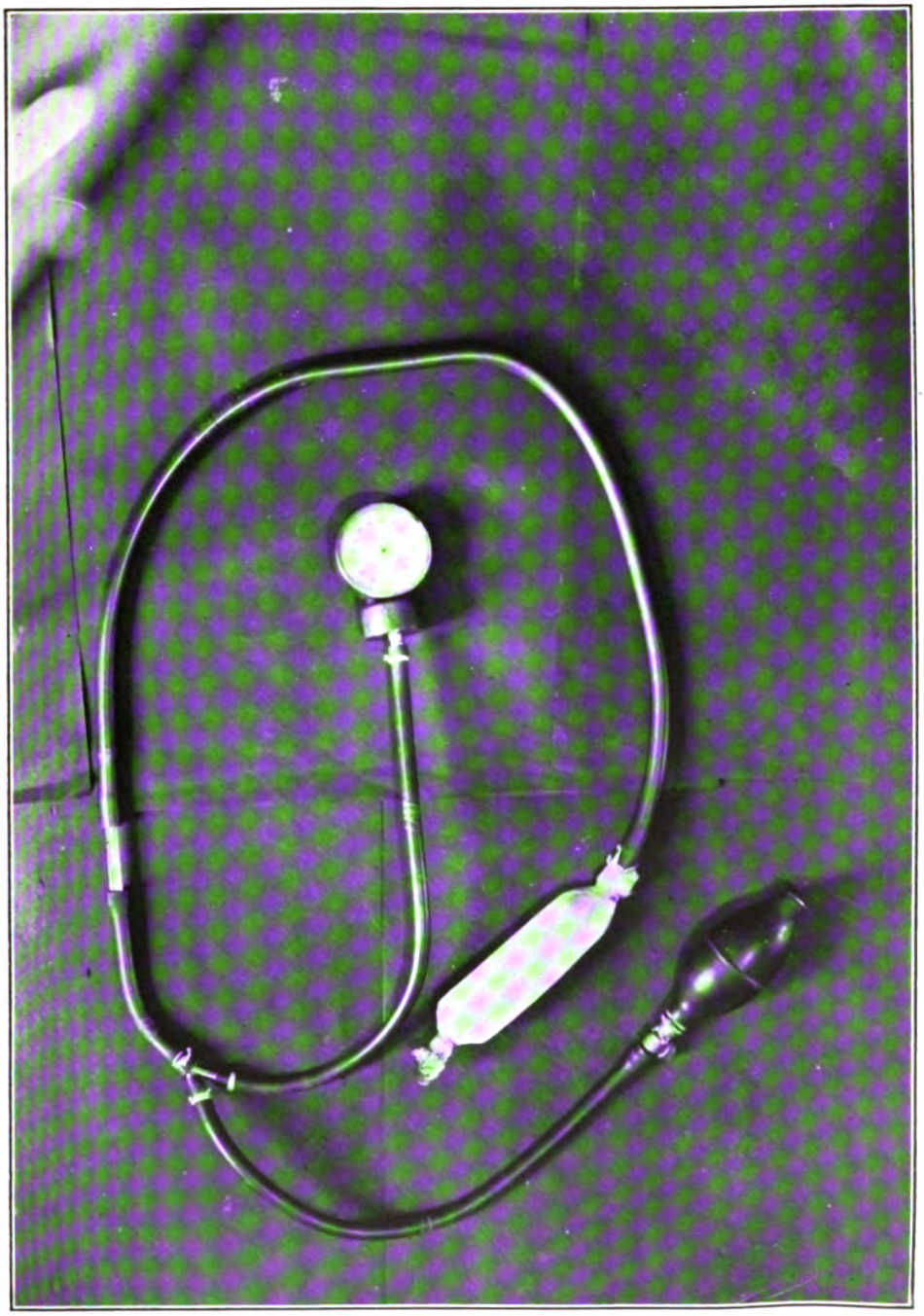

FIGURE 1.—APPARATUS ASSEMBLED.

A heavy buttonhole silk twist may be used. The patient begins to swallow the thread the day before the treatment, taking about 1 foot an hour until 5 yards have been swallowed. The thread finds its way through the cardia and the stomach into the small intestines where it becomes fixed in the coils of bowel. Tension may then be applied to the thread without its being withdrawn.

The esophagus should be emptied by inducing regurgitation. No anesthetic is required. The distance from incisor teeth to the cardia is determined by passing a large sound over the thread guide. The dilator is then introduced over the guiding thread and placed so that the bag projects a short distance into the stomach. Some operators prefer to locate the dilator with the aid of the fluoroscope. If water is used to distend the bag, the container may be elevated so that a pressure of 6 or 7 feet is applied. Air pressure equivalent to 150 millimeters of mercury measured on a sphygmomanometer gage is usually sufficient. When distended the bag becomes a firm cylinder, its circumference determining to what extent the cardia is stretched.

Thre greatest danger of the treatment is rupture of the esophagus. This accident is followed by a fatal infection of the mediastinum and is said to occur in about 1 percent of the cases treated. There is moderate pain during and after dilatation and slight bleeding from the traumatized mucosa may be noted on withdrawing the instrument.

Immediately following dilatation the patient should be able to swallow without difficulty. If not relieved or if there is recurrence of symptoms, the treatment should be repeated. Three fourths of the patients are permanently relieved by one good stretching of the cardia. Most of the recurrent cases can be cured by further dilatation.

In the treatment of the case described below, an apparatus on the principle of the Russell dilator was assembled. A small rubber bag is used and by cutting off the blind end, converted into an elastic rubber cylinder. This is drawn within a silk sheath of the dimensions mentioned above. A flexible staff is passed into a stomach tube without a funnel. This will give the tube sufficient rigidity to pass the contracted cardia. An opening is cut in the side of the stomach tube about 3 inches from the end. The silk-covered bag is then drawn over the tube and secured by silk ligatures above and below this opening. The ligature below should also make an air-tight closure of the lumen of the tube. The terminal and side openings at the end of the stomach tube may be used in introducing the apparatus over a thread guide. When in position within the cardiac orifice, the bag may be distended by air or water forced through the tube.

Case Report

J. G. C., Veterans' Administration patient, age 40 years, an architect, complained of pain over the upper epigastrium, difficulty in swallowing, vomiting (regurgitation), loss of weight, and cough with expectoration.

The patient is of a nervous temperment. He does not sleep well and suffers from "nightmares." He is given to worrying. He suffers from severe "colds on the chest" during the winter.

The present trouble began in 1918, when he had an attack of influenza. This was complicated by severe bleeding from the nose, for which packing of the nasal cavity was done. The patient suffered from vomiting during this treatment and following recovery vomiting persisted. He does not recall the details of his early symptoms. He remembers that he had difficulty in swallowing and that he vomited frequently, without being "sick at his stomach." Pain has been intermittent and has taken the form of "soreness" over the upper epigastrium.

He believes food does not get into his stomach, as he has often regurgitated undigested food eaten the previous day. He coughs much at night and finds his pillow wet with material he has "coughed up." He is 30 pounds under his normal weight of 152 pounds.

Physical examination showed an undernourished, white male adult 69 inches tall. There was some enlargement of the palpable lymph glands. Hearing was defective in the left ear. The tonsils were small and "ragged." General physical examination was otherwise negative. The X-ray examination disclosed symmetrical dilatation of the entire esophagus, ending in a smooth tip at the cardia, with some tortuosity above that point. A stomach tube could not be passed and was seen on fluoroscopic examination coiled in the esophagus.

With a stomach tube containing a flexible staff, little resistance was encountered at the cardia and the tube was passed into the stomach. The cardia was dilated on March 16, 1932, using the apparatus described above.

The position of the dilator in the lower esophagus was verified with the fluoroscope. Air pressure at 150 millimeters of mercury was employed. The patient suffered moderate pain and there was some blood streaked mucus on the dilator when withdrawn. The patient was discharged a week later able to swallow food without difficulty, but still complaining of some pain over the upper epigastrium. Seen 6 months later, he had gained 20 pounds in weight and was taking food without difficulty. He still complained of "soreness" over the upper epigastium.

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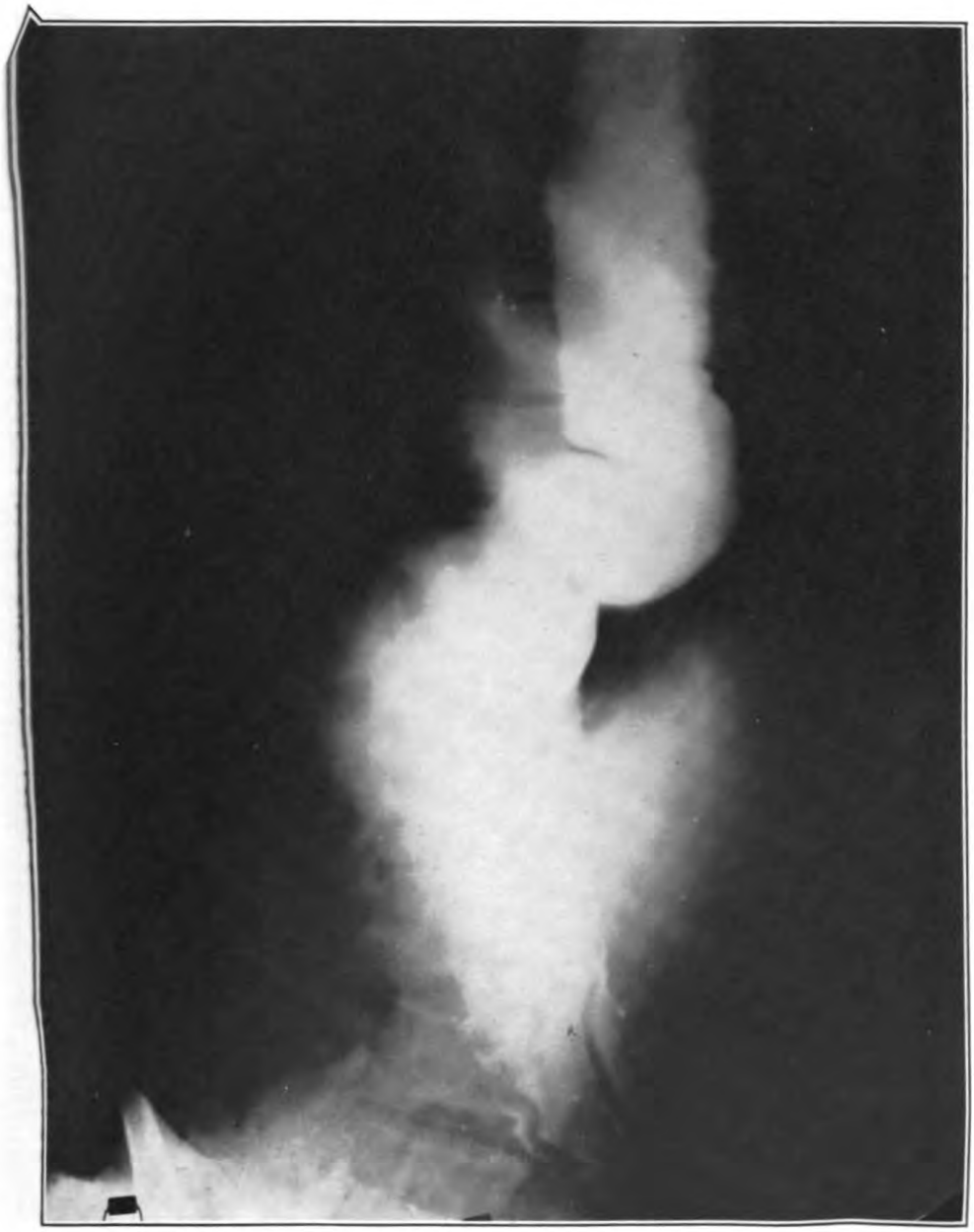


FIGURE 2.—ROENTGENOGRAM SHOWING DILATED ESOPHAGUS WITH SMOOTH TIP.

ATABRINE IN THE TREATMENT OF MALARIA

A Report of 53 Cases

By T. L. Morrow, lieutenant commander, Medical Corps, United States Navy, and W. G. Wieand, lieutenant (junior grade), Medical Corps, United States Navy

In the past, chemotherapeutic research in the treatment of malaria has added the drug plasmochin as an adjunct to quinine; its particular value being its action upon gametes. Further research has evolved the drug atabrine (formerly spelled atebrin) another synthetic product for use in the treatment of malaria. According to the statements of the manufacturer, atabrine was first called "Erion" and is described as the dichloride of an alkylamino derivative of acridine. It is a light-colored yellow powder, bitter to the taste and soluble to the extent of 7 percent in water.

The use of atabrine in Nicaragua was begun as soon as a supply could be obtained after reading the reports of its use by the medical department of the United Fruit Co., whose reports were favorable. Malaria is particularly prevalent in Managua during and after the rainy season. The benign tertian type is most common during the dry season and the early part of the rainy season, but the estivoautumnal type is predominant in the latter part of the rainy season and the months immediately following.

In collecting the data on this treatment, there was no selection of cases. It was begun on November 1 and was terminated on January 1 upon the evacuation. This is the period immediately following the rainy season and a great number of the early cases were estivoautumnal in type.

When a malarial suspect was admitted to the hospital, he was put to bed and thick blood smears made. These smears were stained with Giemsa stain and the same method used in all further blood work. When the diagnosis of malaria was made, treatment was started at once. The routine in use consisted of the administration of atabrine, grains 1½ (1 tablet) and calomel, grains 2. This was followed in 4 hours by magnesium sulphate. The next day the same dosage of atabrine was begun and continued until 15 doses had been given. If the case was estivoautumnal in type, in addition to atabrine plasmochin, grain ⅙, was given twice daily. The temperatures were taken every 4 hours and thick blood smears were made daily until the blood was negative for malarial parasites. Urinalyses were made every second day. Reports had mentioned the peculiar skin discolorations occurring in atabrine treatment, but due to the lack of equipment at the field hospital, liver function tests were not made. In tabulating the results, the days of fever following admission were carefully charted, and in the cases of benign tertian types the number of paroxysms was recorded.

In a small percentage of cases patients presented the symptoms of severe nausea and vomiting and could not retain any medication given by mouth, and it was necessary to give such cases quinine intramuscularly in order to control the extreme nausea. After this was controlled, atabrine was then given for 5 days. In the milder cases no quinine was given.

This report is the result of the treatment of 53 cases, it being necessary to end the series because of the evacuation of Nicaragua. Fifteen cases of this series occurred at aircraft squadrons and the data on those cases were furnished through the kindness of Lt. Comdr. L. E. Mueller, Medical Corps.

Results of Treatment

In the estivoautumnal group of cases the temperatures became normal and remained there on an average of 4 days after the beginning of treatment. The blood smears in these cases became negative on an average of 2½ days. In the benign tertian cases the temperatures fell to normal and remained there on an average of 2½ days after treatment was begun. The blood smears became negative in the same number of days, an average of 2½. In the benign tertian cases the number of paroxysms were recorded and the amount of treatment received when these occurred. In almost all cases there were no paroxysms after the patient had received as many as 7 tablets of atabrine of 1½ grains each. In 2 cases there were paroxysms after as many as 8 and 10 tablets, respectively, had been given.

Three cases of the series were listed as failures of this treatment. After 5 days the clinical symptoms persisted and quinine was then administered in doses of 30 to 40 grains a day, which resulted in immediate improvement. The blood smears became negative in all these cases in 5 days, but the symptoms persisted. All three cases were estivoautumnal in type.

The first case had a history of repeated malarial attacks in Nicaragua which were self-treated and inadequate quinine was taken. Atabrine failed to relieve the symptoms, but quinine did so immediately. The second failure also had a history of three previous attacks, but all were adequately treated. The patient had a history of amebic dysentery and was found to have the encysted forms in his stools constantly while in the hospital. The third case was in general poor health when admitted. He had had several attacks of unexplained diarrhea, which recurred while he was in the hospital. No causative organism could be found in this case.

No explanation is offered for the failure of treatment in these cases. Possibly the complicating conditions played a part but this cannot be stated with certainty.

Urine examinations during atabrine treatment showed nothing unusual. Albumin was found in several cases, but this occurred during and following high fevers which is a frequent finding in all malarial cases treated with quinine or untreated.

Skin discoloration was noted in 10 percent of the cases. This occurred usually about the fourth or fifth day and was similar to the yellowish tinge of a mild icterus, but somewhat lighter in color. This color was also noted in the urine. The blood serum was not tested in these cases, but some writers have attributed this discoloration to the dye of the drug itself. Green, in the Lancet of April 16, 1932, states that his cases did not show an increase of serum bilirubin. The discoloration persisted for 4 to 10 days after treatment was discontinued.

In four cases of the series, abdominal pain was noticed on the fourth day of treatment. Two had very severe pain and two were rather mild. All were estivoautumnal in type and had been taking plasmochin in addition to atabrine. The pains were situated in the epigastrium and were continuous. There was no associated nausea. The plasmochin was discontinued in these cases and the pains subsided in 48 hours. Although this symptom sometimes occurs in plasmochin overdosage, the doses in these cases were small and it was never noticed previously in our patients when the same dosage or larger doses of plasmochin were given with quinine. There were several reports of abdominal distress from cases not studied in this series and who had taken plasmochin with atabrine. The symptoms were very severe in three cases and even suggested a possibility of some intraabdominal surgical condition. These cases had taken a half or more grains of plasmochin daily. This complication should have further study in view of the fact that similar dosages of plasmochin with quinine do not cause these symptoms. There were no other untoward effects of atabrine noted.

No recurrences were found in this series. The distribution of the men after leaving Nicaragua made it impossible to follow all cases. However, 20 percent of the cases were under observation for 2 months and 50 percent for 1 month after discharge. The remainder of the cases were treated during December.

Although this series of cases is large, further study of the effects of the drug are necessary, it is believed that atabrine offers a definite value in the treatment of malaria especially if 5 days' treatment is curative and will prevent recurrence. Perhaps larger doses may be used or it may find an excellent place in conjunction with quinine. It is believed that quinine, especially when given intramuscularly, controls the clinical symptoms of malaria more quickly than atabrine, although no cases were analyzed with this in view. Perhaps after quinine has controlled the severe symptoms, atabrine may be admin-

istered and thus avoid the long much disliked follow-up course of quinine.

Conclusions

1. Atabrine has definite value in the treatment of malaria.
2. It is apparently more effective in controling the symptoms of benign tertian type and is best used with plasmochin in treating estivoautumnal cases.
3. Blood smears are usually made negative in 2 or 3 days.
4. Abdominal distress is sometimes noted when plasmochin is used with atabrine.
5. The drug is easy to administer and no untoward effects other than the above were noted.
6. No recurrences were found in a series of 53 cases.
7. Further study of the effects are recommended.

Number	Type	History	Treatment	Days fever	Positive smears	Results
1	B.T.	Malaria 2 weeks ago; inadequate treatment.	Atebrin, gr. 1½, t.i.d.	3	3	1 paroxysm after 6 tablets; normal recovery.
2	B.T.	2 paroxysms before admission.	Atebrin, gr. 1½, t.i.d.	1	2	No paroxysms after admission; normal recovery.
3	B.T.	Typical case; admitted in first paroxysm.	Atebrin, gr. 1½, t.i.d.	1	2	Recovery; no symptoms after admission.
4	B.T.	Mild case	Atebrin, gr. 1½, t.i.d.	1	2	Normal recovery after initial fever.
5	B.T.	Typical case; slight nausea on admission.	Atebrin, gr. 1½, t.i.d.	3	2	In a paroxysm on admission and 1 after 6 tablets.
6	B.T.	Recurrence; stopped quinine 4 days ago.	Atebrin, gr. 1½, t.i.d.	3	3	Recovery; 1 paroxysm after 5 tablets.
7	B.T.	Symptoms severe	Atebrin, gr. 1½, t.i.d.	4	4	1 paroxysm after 6 tablets; recovery.
8	B.T.	Paroysm on admission	Atebrin, gr. 1½, t.i.d.	1	2	6 tablets prevented second paroxysm.
9	B.T.	Admitted with general malaise.	Atebrin, gr. 1½, t.i.d.	3	3	1 paroxysm after 6 tablets; normal recovery.
10	B.T.	Typical case	Atebrin, gr. 1½, t.i.d.	4	3	A paroxysm after 3 tablets; another after a total of 8.
11	E.A.	Several previous attacks with inadequate quinine.	Atebrin and plasmochin.	12	2	Improved up to fourth day when symptoms recurred with severe nausea; quinine intramuscularly on sixth day with recovery.
12	E.A.	Mild case	Atebrin and plasmochin.	3	1	Normal recovery.
13	E.A.	Recurrence after irregular quinine treatment.	Atebrin and plasmochin.	3	2	Recovery normal; abdominal pain on fifth day.
14	E.A.	Mild case	Atebrin and plasmochin.	3	1	Normal recovery.
15	E.A.	Mild case	Atebrin and plasmochin.	4	3	Normal recovery.
16	Mixed	Several previous attacks	Atebrin and plasmochin.	3	3	No symptoms after second day of treatment.
17	E.A.	Mild case	Atebrin and plasmochin.	2	2	Normal recovery.
18	E.A.	Complicated with advanced pulmonary T.B.	Atebrin and plasmochin.	4	3	Severe abdominal pain on fifth day which subsided 2 days after treatment stopped.
19	B.T.	Woman 6 months, pregnancy.	Atebrin, gr. 1½, t.i.d.	1	2	Recovery from all symptoms 3 days after treatment began.
20	B.T.	Typical case	Atebrin, gr. 1½, t.i.d.; quinine, gr. 20, at onset.	2	2	High fever on second day with normal recovery; ambulatory case.
21	E.A.	General malaise	Atebrin and plasmochin.	3	2	Normal recovery after third day; slight diarrhea on third day.
22	E.A.	Mild case	Atebrin and plasmochin.	3	2	Normal recovery; patient ambulatory.
23	E.A.	Mild case	Atebrin and plasmochin.	4	4	Normal recovery; skin discoloration marked on fifth day.
24	E.A.	Mild case	Atebrin and plasmochin.	1	2	Normal recovery.

Number	Type	History	Treatment	Days fever	Positive smears	Results
25	E.A...	Severe symptoms for 3 days.	Atebrin and plasmochin.	4	4	Severe abdominal cramps on the fifth day; recovery followed.
26	E.A...	Several previous attacks; patient also had encysted E. histolytica.	Atebrin and plasmochin; quinine intramuscularly.	10	2	Resistant to treatment, with vomiting difficulty to control; quinine intramuscularly on the fourth day. Temperature normal after 10 days.
27	E.A...	Repeated attacks with loss of weight and anemia.	Atebrin and plasmochin.	2	3	Normal course; severe abdominal pain on the fifth day; pain disappeared in 2 days.
28	E.A...	Severe symptoms.....	Atebrin and plasmochin.	8	4	Slow response to treatment; weakness and emaciation pronounced.
29	Mixed	Typical case.....	Atebrin and plasmochin.	4	3	Marked chill after 7 tablets; recovery after the fourth day.
30	E.A...	Severe abdominal pain simulating appendicitis.	Atebrin and plasmochin.	1	2	No symptoms after 1 day of treatment.
31	B.T...	Mild case.....	Atebrin, gr. 1½, t.i.d.	3	4	Normal recovery.
32	B.T...	Mild case.....	Atebrin, gr. 1½, t.i.d.	2	3	Normal recovery.
33	B.T...	Symptoms severe.....	Atebrin, gr. 1½, t.i.d.	5	4	Recovery after fifth day.
34	B.T...	Mild case.....	Atebrin, gr. 1½, t.i.d.	4	4	Normal recovery.
35	B.T...	Mild case.....	Atebrin, gr. 1½, t.i.d.	1	2	Normal recovery.
36	B.T...	Mild case.....	Atebrin, gr. 1½, t.i.d.	3	3	Normal course.
37	B.T...	Mild case.....	Atebrin, gr. 1½, t.i.d.	2	2	Normal course.
38	B.T...	Typical paroxysm on admission.	Atebrin, gr. 1½, t.i.d.	1	2	Normal recovery.
39	B.T...	Usual sypmtoms.....	Atebrin, gr. 1½, t.i.d.	3	3	Normal recovery.
40	B.T...	Mild case.....	Atebrin, gr. 1½, t.i.d.	2	2	Normal recovery.
41	B.T...	Severe symptoms.....	Atebrin, gr. 1½, t.i.d.	5	4	Recovery after fifth day.
42	B.T...	Mild case.....	Atebrin, gr. 1½, t.i.d.	2	2	Normal recovery.
43	B.T...	Mild case.....	Atebrin, gr. 1½, t.i.d.	1	1	Normal recovery.
44	B.T...	Usual symptoms.....	Atebrin, gr. 1½, t.i.d.	2	3	Normal recovery.
45	E.A...	Typical case.....	Atebrin and plasmochin.	3	3	Normal recovery.
46	B.T...	Mild case.....	Atebrin, gr. 1½, t.i.d.	2	2	Normal recovery.
47	E.A...	Mild case; B.T. malaria 1 month ago.	Atebrin and plasmochin.	2	1	Normal recovery.
48	E.A...	Typical case.....	Atebrin and plasmochin.	2	2	Normal recovery.
49	E.A...	Mild case.....	Atebrin and plasmochin.	2	1	Normal recovery.
50	B.T...	Mild case.....	Atebrin, gr. 1½, t.i.d.	2	2	Slight paroxysm after 5 tablets.
51	E.A...	Mild case.....	Atebrin and plasmochin.	3	3	Normal recovery.
52	B.T...	Mild case.....	Atebrin, gr. 1½, t.i.d.	3	2	Normal recovery.
53	B.T...	Mild case.....	Atebrin, gr. 1½, t.i.d.	4	2	Normal recovery.

LEPROSY IN THE PHILIPPINES

By M. E. Higgins, Captain, Medical Corps, United States Navy

Leprosy is one of the outstanding health problems of the Philippines. The Culion colony with its 5,700 patients is probably the largest leprosarium in the world. There are approximately 2,000 lepers segregated at other places, so that the total number under observation is about 8,000. Excellent facilities are afforded for study and research and Philippine workers have made and are making valuable contributions to our knowledge of the disease. A

recent bibliography compiled by the Director of the Philippine Health Service cites 177 references in the literature from Philippine sources.

The writer has recently visited the leprosaria at Culion, Iloilo, Cebu, and Zamboanga. At all of these stations every opportunity was afforded him to observe the medical and administrative procedures employed in the management of the disease.

Culion is an island with an area of nearly 200 square miles lying 250 miles south of Manila. The Culion Leper Colony was established in 1906 as the principal center for the segregation and treatment of leprosy. It is also the chief center for bacteriological and pathological research and for the manufacture of antileprosy drugs.

The colony has a 500-bed hospital, dormitories for 2,000 patients, clinic buildings, and laboratories. These buildings are of concrete construction. In other respects the colony resembles the ordinary Philippine town, most of the houses being bamboo and nipa construction of the type commonly seen throughout the islands. As far as practicable the patients are encouraged to carry on their usual vocations; some manage retail shops; others are engaged in fishing; a number cultivate small farms on the outskirts of the town and sell their products in the colony.

There are about 1,000 nonlepers on the island—physicians, nurses and administrative assistants who live in a special reservation separated from the main colony.

A portion of the island has been set aside for the development of an agricultural community to be operated by paroled lepers who can no longer be cared for in the colony proper, but who do not, for various reasons, wish to return to their former homes. This "negative varrio" is 16 kilometers from the main colony and is reached by an excellent road constructed almost entirely by the patients themselves.

The Eversley Childs Treatment Station near Cebu is the second most important station in the Philippines. It was designed to accommodate 500 patients and built under the auspices of the Leonard Wood Memorial. It consists of a hospital, dormitories, clubhouse, administrative buildings, and a chapel—all of concrete construction. The ample grounds have been attractively laid out and planted with a variety of palms and flowers so that the general effect is extremely pleasing.

The Western Viscayas Treatment Station is located on the island of Panay near Iloilo. It accommodates 275 patients. All of the buildings at this station are of temporary or light construction.

The central station for the island of Mindanao is at Zamboanga. A new 50-bed frame hospital has just been completed; it replaces a number of small structures of temporary construction.

In addition to the above stations the Bihol Treatment Station in southern Luzon and the San Lazaro Hospital at Manila accommo-

date about 800 cases. There are also a number of substations throughout the Philippines where cases can be held temporarily pending transfer to one of the central stations. In the larger cities skin clinics have been established which are proving to be a valuable factor in the detection of incipient cases.

The Leonard Wood Memorial is a $2,000,000 foundation established in memory of General Wood who was much interested in the control of leprosy. It works in close cooperation with the Government Health Service and is especially interested in research work bearing on the etiology, pathology, and bacteriology of leprosy. Its chief activities are at Culion and Cebu. At the former place a thoroughly equipped laboratory and experimental wards are nearing completion. At the Eversley Childs Station at Cebu which was built by the foundation at a cost of $200,000, it is proposed to devote particular attention to the question of etiology.

Dr. H. W. Wade, who was for many years the director of the pathological section at Culion, is the field director of the memorial with headquarters at Culion.

Observations in the Philippines bear out the generally accepted belief that leprosy is a familial disease and that infection occurs almost exclusively during infancy and early childhood. Adult infection seems to be extremely rare. During the 25 years that Culion has been in operation there has not been a single case of the disease developing among the physicians, nurses, and attendants. One Sister of Charity who has been in the colony since its beginning and in the most direct contact with active cases has remained free from infection.

It is estimated that 95 percent of all cases occur among the poorer classes. Bad hygienic surroundings and defective diet undoubtedly play a prominent role in determining susceptibility to the disease.

The ethyl esters of the oil of the *Hydnocarpus wightiana* are now generally used throughout the Philippines in the treatment of leprosy. The crude oil is imported from India and refined in the chemical laboratories of the Culion colony. One half of 1 percent metallic iodine is added to the esters in order to lessen irritation. The drug is injected either intradermally or intramuscularly. Usually a combination of these methods is employed. The total dose is 5 cubic centimeters. When given intradermally not more than 0.1 cubic centimeter is injected at any one point. Injections are made two or three times weekly. The site of the intradermal injections is marked by a characteristic bluish discoloration which may last for several months. The general belief in the Philippines is that this treatment does not cure the disease. "Once a leper always a leper" is an opinion frequently expressed by many experienced workers. There is no doubt however that acid-fast organisms disappear from nasal and dermal lesions and that the progress of tubercular lesions is arrested. These

are the "negative" or "arrested" cases that are paroled. It has been found that a considerable number of these paroled cases again become active.

Apparently the question of diet as a therapeutic measure has not been given any special consideration, chief reliance having been placed upon segregation and the administration of chaulmoogra oil. It is generally recognized that the diet of the poorer classes is not well balanced, being deficient especially in proteins and fats. Vitamin deficiency also occurs. Beri-beri is not infrequently a concomitant disorder. Since vitamin B is often lacking it is not unreasonable to suppose that other important factors are inadequate. Certain observers believe that a scientifically controlled diet and hygienic management along the lines now employed in the treatment of tuberculosis may have a profound effect upon the progress of early leprosy. Apart from the similarity of the causative agents the parallelism existing between tuberculosis and leprosy is striking.

Segregation and chaulmoogra oil have apparently failed to make any marked reduction in the number of cases of leprosy occurring annually in the Philippines. Under present conditions the eradication of the disease will have to wait upon such slow factors as rising cultural and economic levels. Additional measures that have been recommended include the removal of the children of leprous parents at birth, the sterilization of male lepers, and birth control. The application of all these procedures is attended by manifest difficulties. The education of the masses regarding early diagnosis and treatment has yielded valuable results. At all of the stations more and more people are voluntarily applying for examination and treatment. One hopeful feature in the control of the malady is that it is no longer regarded with the exaggerated horror which has so long characterized the public conception of the disease.

The expense incident to maintaining 8,000 lepers in segregation places a great strain on the budget of the Philippine Health Service. It is estimated the Government has expended $11,000,000 on the control of leprosy since the establishment of the Culion Colony in 1906. At the present time nearly one third of the health appropriations is devoted to leprosy. Many physicians feel that this amount is inordinate and that some of the funds now devoted to leprosy should be expended in the fight against the tuberculosis, a disease in which the mortality and morbidity are vastly greater. The number of lepers in segregation is constantly increasing and practically all the stations are at their capacity level. Unless some means can be found to lessen this growing financial burden, the situation will soon become a serious problem.

SUMMARY

1. There are at present about 8,000 lepers in segregation in the Philippines.

2. The iodized esters of the oil of the *Hydnocarpus wightiana* are used in the treatment of the disease.

3. The consensus of opinion is that permanent cure is not obtained.

4. In early cases marked improvement occurs in tubercular lesions; acid-fast organisms disappear from nasal and dermal lesions.

5. Adult infection is extremely rare, the belief being that infection takes place during infancy and childhood.

6. The control of leprosy absorbs one third of the funds available for health work in the Philippines.

CLINICAL NOTES

SPONTANEOUS BILATERAL PNEUMOTHORAX

Case Report

By Paul Richmond, Lieutenant Commander, Medical Corps, United States Navy

In the May 1932 number of the American Journal of the Medical Sciences (vol. CLXXXIII, no. 5), there is a case report of a spontaneous bilaterial pneumothorax with a review of the literature. The author states that this is a very rare condition, less than 50 cases having been reported.

The following case was observed at the United States Naval Hospital, Canacao, P.I.

The patient, J. L., seaman first class, United States Navy, age 31, was admitted on December 26, 1932, with diagnosis undetermined (pneumonia, lobar). X-ray on admission showed the right lung to be completely collapsed with a right pneumothorax. There was no fluid in the right pleural cavity. There was no displacement of the mediastinum. The left lung appeared normal.

The patient complained of intense headache, pain in the right side of the chest and persistent cough. Onset had been sudden the previous day with the above symptoms and dyspnœa. There was no history of contusion to the chest, violent exertion or any severe previous paroxysm of coughing. Patient had a nonproductive cough for several months prior to his present illness. He was on the sick list in February 1932 with a diagnosis of bronchitis. Previous illnesses had been gonococcus infection in 1927 with epididymitis and another gonorrhœa in 1932. During 11 years of naval service, there had been no other illness.

Pulse was 85 on admission and later increased to 120, blood pressure 138/98. Temperature was 99.4° F. on admission, reaching 100° F. the same evening. Respiration was 24 to 32 per minute and labored. Moderate cyanosis was present. There was a persistent productive cough. No blood was present in the sputum. Breath sounds were absent on the right side but exaggerated throughout the left side. There were a few moist râles in the upper half of the lung. Heart sounds normal. Apex beat not displaced. Physical examination showed otherwise normal findings. White blood count 10,200 (polymorphonuclears, 68; lymphocytes, 29; large mononuclears, 1 percent; eosinophiles, 2 percent). Urine showed 1 plus albumin and finely granular casts. Sputum negative for acidfast bacilli.

Patient was given codein ¼ grain at approximately 3-hour intervals for cough, a cathartic, and kept in bed with a pneumonia jacket on his chest. On December 27, he seemed to show no change. At 8:30 p.m., December 27, he was given barbital, grains X, for restlessness but appeared to be no worse.

At 1 a.m., December 28, cough was more severe and he was given codein ¼ grain. Patient was observed at about 5 a.m. by the corpsman on watch and seemed to be asleep but breathing with difficulty. At about 5:30 a.m., Decem-

ber 28, patient was cyanosed and was not breathing. He was dead when the officer of the day reached him a few minutes later.

Autopsy.—Right lung completely collapsed except for two areas about 5 centimeters in diameter which were markedly distended with air. No blood or fluid in right pleural cavity. No gross rent in right lung found. Numerous small tubercles at right apex mostly calcified but a few contained caseous material. No tuberculous cavity present. Lower lobe of left lung completely collapsed. No fluid in left pleural cavity. Left upper lobe partly distended, emphysematous and adherent to chest wall at apex and posteriorly but no consolidated areas present. Some calcified and some caseous nodules in left apex but no cavity found. Gross rent of left lower lobe near hilus posteriorly which, from the amount of blood infiltrating the tissues, seemed to have been antemortem but, as it was associated with the separated pleural adhesions, this point was uncertain. Liver markedly enlarged and showing passive congestion. Spleen not enlarged, soft and on section showed what appeared to be numerous tubercles about 2 millimeters in diameter. Heart and aorta normal. Other abdominal viscera normal. Brain not examined.

Cause of death considered to be due to double spontaneous pneumothorax secondary to tuberculosis of moderately advanced extent.

Microscopic examination of autopsy material by Maj. J. E. Ash, Medical Corps, United States Army.

Lung blocks not identified as to lobes. One block shows an organized croupous pneumonia with active miliary tuberculosis. Another shows atelectasis, moderate anthracosis and congestion. Another, apparently from same lobe, shows less collapse. Another block includes an encapsulated, inactive, caseous tuberculous nodule with active miliary tubercles in surrounding parenchyma. No atelectasis in this block. Blocks from spleen do not include any tubercles.

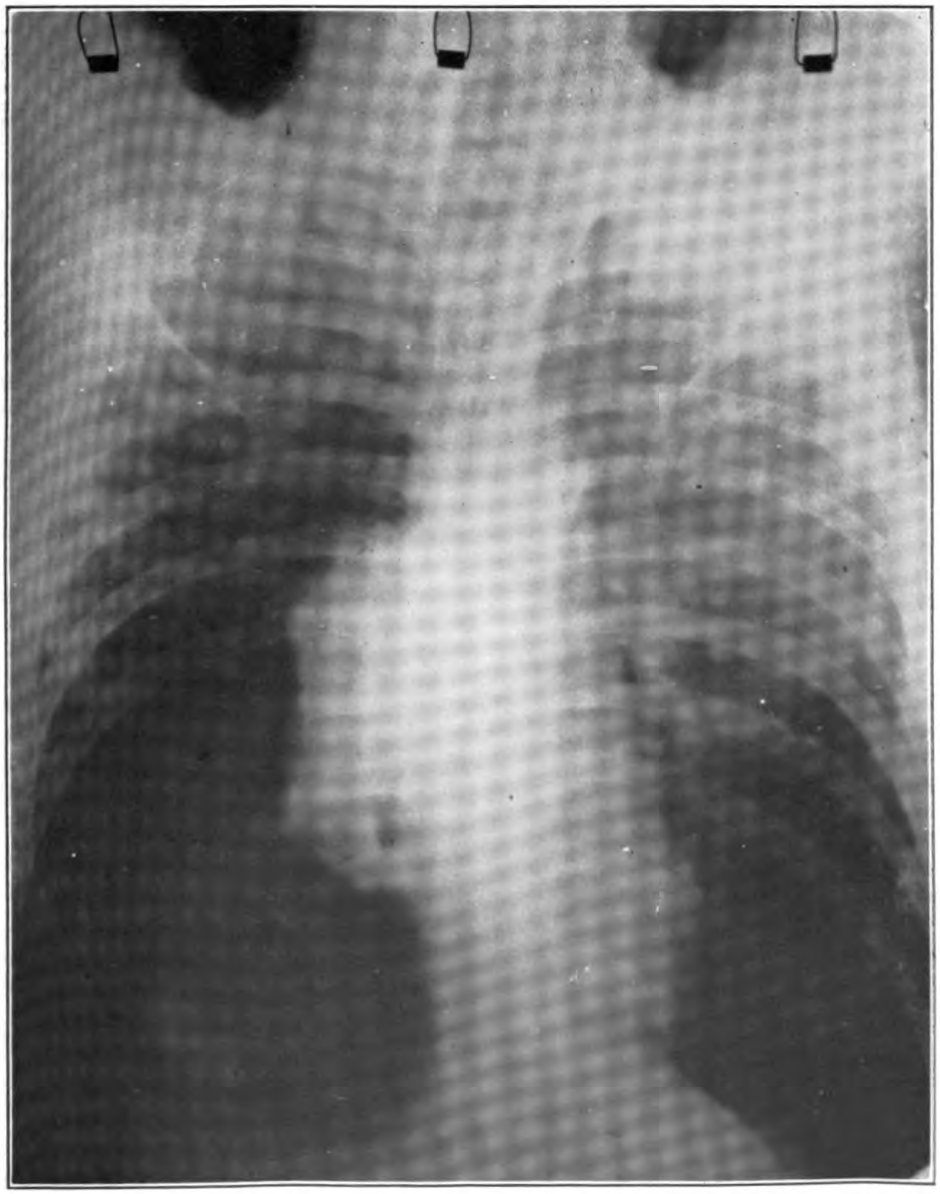
CUTANEOUS MYIASIS, A CASE REPORT

By W. G. Wieand, Lieutenant (junior grade), Medical Corps, United States Navy

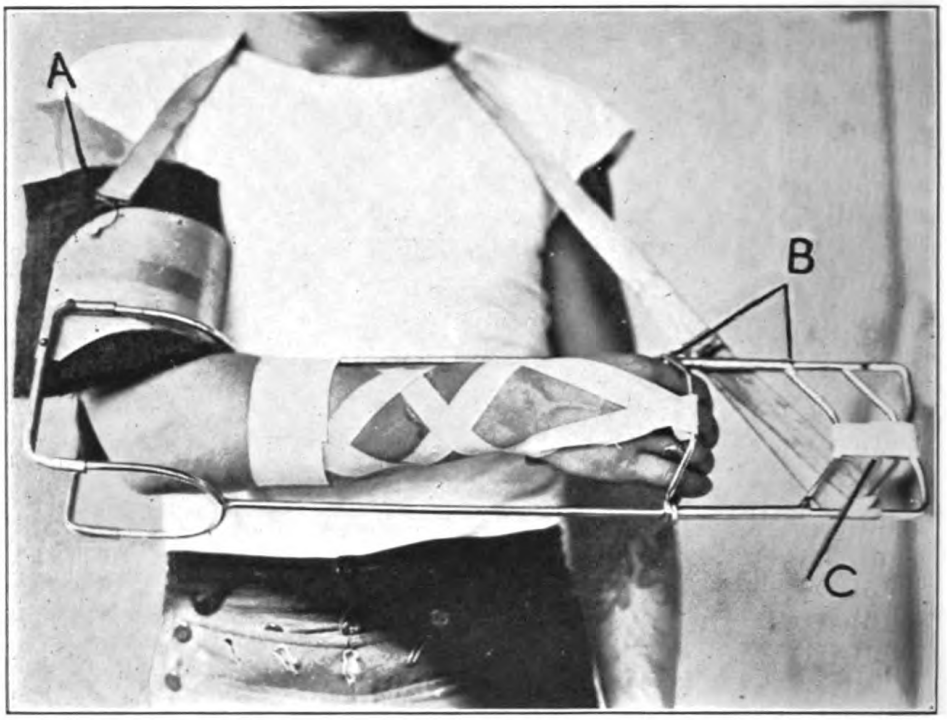
A civilian patient, an American, entered the field hospital at Managua, Nicaragua, complaining of pain and swelling in the left forearm. He gave the history of having just walked from Honduras and had suffered severe sunburn and many mosquito bites on the exposed parts of his body. He had been surveyed from the United States Army in 1913 with pulmonary tuberculosis.

The patient was not acutely ill and, upon examination, was found to have a temperature of 99° with a typical picture of advanced cellulitis of the left forearm. It was a dusky red in color and markedly edematous. Scattered over the forearm were several large boils which were very hard and showed no evidence of suppuration except a few which had a slight serous discharge. The epitrochlear glands were enlarged and tender. There was another lesion on the right wrist but with very little inflammation about it.

Hot wet dressings were applied to the arm, and in a few hours, because the pain was so great, the largest of the lesions was incised. A sero-sanguinous drainage resulted with some relief of pain. The following day much of the inflammation had subsided, and some of the boils showed a slight serous discharge through a small opening in the center. Close examination of this opening showed a slight motion there, and a wormlike object protruded itself and rapidly withdrew. The patient at this time stated that he felt a sensation of something crawling under his skin. The object protruded itself about once a minute and on one occasion was seized and withdrawn. It was found to be the larva of the bot fly,

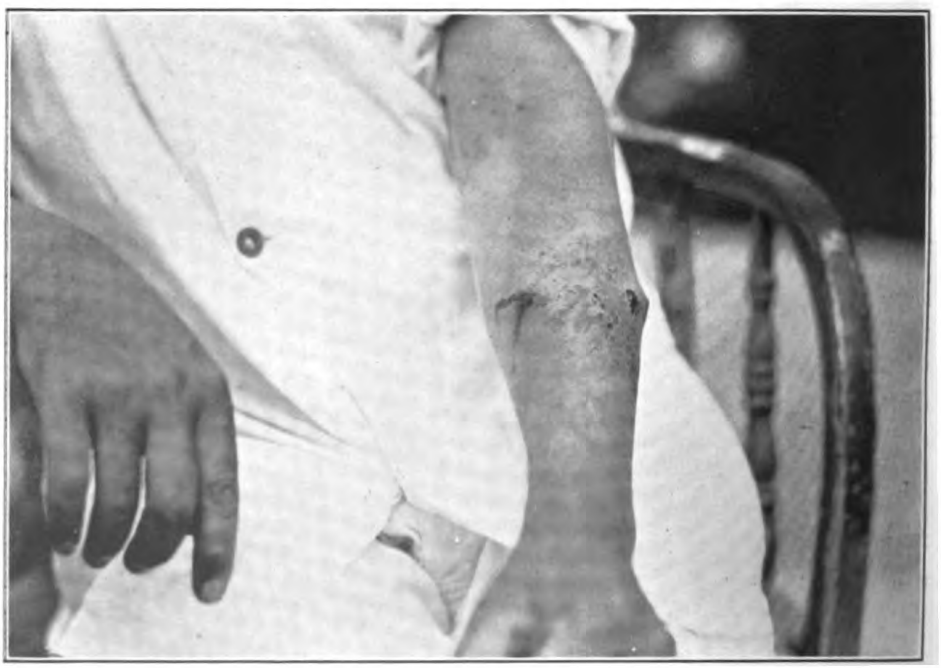


X-RAY TAKEN ON DAY OF ADMISSION SHOWING RIGHT PNEUMOTHORAX WITH COMPLETE COLLAPSE OF RIGHT LUNG.



THE SPLINT WITH THE FOREARM IN THE MIDPRONE POSITION. THE WIDE BAND OF ADHESIVE WAS ATTACHED TO ONE SIDE OF THE SPLINT TO CORRECT THE LATERAL BOWING.

A, Shows the heavy felt padding used to protect the arm; B, the sliding part of the splint that allows traction to be adjusted to the case; C, shows the manner in which the traction is secured.



CUTANEOUS MYIASIS (WIEAND).

Dermatobia hominis. It was wormlike in shape, segmented, and had several double rows of short, stiff, hairlike projections which caught the skin when the larva was extracted and caused the patient slight pain. This procedure was repeated on several other lesions with the same results. The larvae of some of the lesions were club shaped and varied in size. The tops of all the lesions were removed with the scalpel, and movements could be seen in all of them, but the larvae could not all be withdrawn. Into these a small amount of chloroform was injected and into all the others 70 percent alcohol was instilled. A blood count taken at this time was normal except for the eosinophiles which were 36 percent.

The following day all the lesions which were injected with alcohol, after the extraction of the larvae, showed signs of rapid healing. Those injected with chloroform were incised, and the larvae removed with a small amount of purulent discharge. These larvae showed no motion and some were macerated in a small degree. Alcohol dressings were applied and there was rapid healing of the wounds.

According to Stitt, cutaneous myiasis occurs most frequently in Central Africa and Central America. Several of the local Nicaraguan physicians stated that they had never seen a case there. The type here described is caused by the larva of the bot fly, *Dermatobia hominis*. The skin lesion resulting from the infestation resembles a large boil with a central opening through which the larva protrudes its spiracle and breathes. There is usually no pain except occasionally that caused by the movements of the larva with its stiff hairlike projections. The larva usually matures in 6 to 8 weeks. It is first club shaped and is then termed "ver macaque", later developing into a wormlike body called "torcel", in Venezuela and "berne" in Brazil. The several rows of stiff whisker projections are probably responsible for the pain which sometimes occurs in the movements of the larva and these also offer resistance to the attempts at extraction.

The adult fly has no puncturing parts to deposit the larvae in the skin and accomplishes this by depositing the eggs in a gluelike substance on the under surface of mosquitoes, biting flies, and ticks. The larva usually develops in about a week and enters the skin of the host through the wound made by the insect's bite. After the larva matures, it escapes after the disintegration of the tumorlike mass caused by its presence.

Stitt recommends the treatment described here. The natives are said to use tobacco juice poultices which cause the spiracle to protrude, after which the larva is squeezed out.

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STITT: Diagnostics and Treatment of Tropical Diseases.

THE CLAYTON FOREARM SPLINT IN FRACTURES OF THE RADIUS AND ULNA IN THE UPPER THIRD

By J. W. SHUMATE, Lieutenant (junior grade), Medical Corps, United States Navy

The forearm is the site of more fractures than any other extremity, chiefly because of fractures in the lower third of the radius. More or less uncommon, however, are fractures of both bones in the upper third. This is a fortunate fact, as such fractures present great difficulty in reduction and are often followed by unsatisfactory results.

The chief difficulties in the reduction and treatment of these fractures are:

1. They almost always result from direct violence and usually are accompanied by massive tissue injury or compounding.
2. Great muscular development and exaggerated muscle tension make deformity easy and reduction difficult.
3. The deformity is in three planes—antero-posterior, lateral, and rotary.
4. There are 4 fragments to oppose, instead of the usual 2, and the bones are small.
5. There is a constant danger of cross-union and synostosis.

The difficulty experienced in the treatment of these fractures by the usual closed methods is so general that open reduction is often resorted to before the closed methods have been given a fair chance to accomplish the desired result. Open reduction in the hands of an experienced operator will give excellent anatomical and functional results in a large percentage of cases, but the chances for failure, even in his hands, are not remote.

In fractures of both bones of the forearm skeletal traction is almost, if not entirely, out of the question and any other form of continuous traction does not hold out the promise for anatomical reduction and restoration of function that it does in the lower extremity. Even with its limitations, however, traction seems to offer the most favorable chance for satisfactory results and should be attempted before considering more radical procedures.

My experience with fractures of both bones of the forearm in the upper third has been limited, but two such cases that I have treated with the Clayton forearm splint (Zimmer) have responded so satisfactorily to the traction treatment that I have been convinced of its usefulness in the treatment of these fractures. These two cases, one of which is reported in brief detail below, were compounded, had marked displacement, and neither responded with any degree of satisfaction to other closed methods of treatment.

The Clayton splint, see figure, has been entirely satisfactory in the treatment of all simple fractures of the shaft of the radius or ulna, but has been particularly useful in difficult cases such as the one described. It possesses, in my opinion, distinct advantages over the plaster cast, the make-shift metal splint or the usual methods of continuous traction for the following reasons:

1. It is a light, ambulatory splint, affords a comfortable means of immobility and provides sufficient traction when required.
2. It allows dressing, massage, and minor adjustments without removing the splint or loosening the traction.
3. It allows mobility of the fingers and restrained motion of the wrist.
4. It reduces the danger of paralysis and gangrene occasionally encountered in the use of the plaster cast.
5. It allows the extremity to be put up in the optimum position; that is, prone, mid-prone, or supine.

(Attention should be called to the fact that the supine position is optimum, other things being equal, as the forearm has its greatest strength toward pronation and most purposeful motions are in that direction.)

Case Report

W. M., age 21, reported to the officer of the day, United States Naval Hospital, Newport, R.I., and presented all the cardinal symptoms of a fracture of the radius and ulna in the upper third of the left forearm. There was a punctured wound on the dorso-mesial surface of the forearm, the result of compounding. The deformity, confirmed by X-ray, included marked overlapping of the fragments and displacement in three planes. After attempted reduction by other means the extremity was placed in the Clayton splint and reduction performed under the fluroscope. The splint was then secured and the patient allowed to go home with instructions to report to the hospital every second day. On the fourth day there was a marked lateral bowing, probably the result of a fall. This bowing was corrected as shown in the accompanying figure. Daily massage was instituted after the first week and active and passive motion started on the tenth day. The patient was discharged in good condition at the end of the seventh week and when seen 2 years later he presented a perfect extremity, anatomically and functionally.

1927—33——3

NAVAL RESERVE

MEDICAL CORPS

APPOINTMENTS, SECOND QUARTER, 1933

Name	Rank	Appointed
Cannon, Frank M	Lieutenant, M.C.-V (S)	Mar. 30, 1933
Cromwell, Henry A	Lieutenant (junior grade) M.C.-V (G)	Apr. 1, 1933
Gerty, Francis J	Lieutenant M.C.-V (S)	Mar. 15, 1933
Green, Dove W	Lieutenant M.C.-V (G)	Apr. 5, 1933
Hatchette, Charles V	Lieutenant (junior grade) M.C.-V (G)	Apr. 12, 1933
Keim, Silas A	Lieutenant (junior grade) M.C.-V (S)	Apr. 20, 1933
Meck, Floyd S	Lieutenant (junior grade) M.C.-V (G)	Feb. 21, 1933
Millard, Robert D	Lieutenant M.C.-V (S)	Apr. 10, 1933
Nuckolls, Chester R	Lieutenant (junior grade) M.C.-V (G)	Mar. 16, 1933
Parker, Francis P	Lieutenant (junior grade) M.C.-V (S)	Apr. 17, 1933
Shea, Frank R	Lieutenant (junior grade) M.C.-V (G)	Apr. 12, 1933

PROMOTIONS

Name	From—	To—
Behneman, Harold M. F	Lieutenant (junior grade) M.C.-V (G)	Lieutenant M.C.-V (G)
Chapman, Sims A	Lieutenant (junior grade) M.C.-F	Lieutenant M.C.-F.
Creswell, Samuel M	Lieutenant (junior grade) M.C.-F	Lieutenant M.C.-F.
Davis, George B	Lieutenant (junior grade) M.C.-F	Lieutenant M.C.-F.
Lawson, Edwin H	Lieutenant (junior grade) M.C.-V (S)	Lieutenant M.C.-V (S).

TRANSFERS

Name	From—	To—
d'Alessio, Joseph A	Lieutenant (junior grade) M.C.-V (G)	Lieutenant (junior grade) M.C.-F.
Cardwell, John L	Lieutenant (junior grade) M.C.-V (G)	Lieutenant (junior grade) M.C.-F.
Fulcher, Oscar H	Lieutenant (junior grade) M.C.-V-G	Lieutenant (junior grade) M.C.-V (S).
Hammond, Thomas V	Lieutenant commander M.C.-F	Lieutenant commander M.C.-V (G).
Thomas, Charles C	Lieutenant M.C.-V (G)	Lieutenant M.C.-F.
Stephens, Doran J	Lieutenant (junior grade) M.C.-F	Lieutenant (junior grade) M.C.-V (G)

DENTAL CORPS

APPOINTMENTS, SECOND QUARTER, 1933

Name	Rank	Appointed
Bovik, Ellis G	Lieutenant (junior grade), D.C.-V (G)	May 15, 1933
Della Croce, Francis R	Lieutenant (junior grade), D.C.-V (G)	Mar. 31, 1933
Farmer, Willard T	Lieutenant (junior grade), D.C.-V (G)	Mar. 17, 1933
Hayes, Louis V	Lieutenant Commander, D.C.-V (S)	May 3, 1933
Turner, Kenneth O	Lieutenant (junior grade), D.C.-V (G)	June 28, 1933
Willhelmy, Glenn E	Lieutenant (junior grade), D.C.-V (G)	Mar. 28, 1933

PROMOTIONS

Name	From—	To—
Waas, Clifford J	Lieutenant (junior grade), D.C.-V. (G)	Lieutenant D.C.-V. (G).

NOTES AND COMMENTS

DECORATIONS

The Nicaraguan Government has recently conferred decorations upon the officers of the United States Navy in recognition of services they have rendered to Nicaragua.

The Nicaraguan Medal of Merit, for exceptional service, was awarded the following officers:

Roy Aikman, chief pharmacist,
Victor C. Barringer, Jr., lieutenant commander,
Robert L. Douthat, lieutenant (junior grade) (M.C.),
Leon H. French, chief pharmacist,
Harry S. Harding, lieutenant commander (M.C.),
Freeman C. Harris, lieutenant (junior grade) (M.C.),
Vincent Hernandez, lieutenant commander (M.C.),
Hardy V. Hughens, lieutenant commander (M.C.),
Walter G. Kilbury, lieutenant (M.C.),
Colonel H. Mansfield, lieutenant (Ch.C.),
Hugh E. Mauldin, lieutenant (D.C.),
George H. Mills, lieutenant (junior grade) (D.C.),
Louis E. Mueller, lieutenant commander (M.C.),
William W. Smith, commander,
Warren G. Wieand, lieutenant (junior grade) (M.C.),
Maurice M. Witherspoon, commander (Ch.C.).

Medals of Distinction, for distinguished service, were awarded the following officers:

Walter L. Bach, lieutenant (M.C.),
Thomas L. Morrow, lieutenant commander (M.C.),
Louis E. Mueller, lieutenant commander (M.C.),
Victor B. Riden, lieutenant commander (M.C.),
William H. H. Turville, lieutenant commander (M.C.).

The Presidential Medals of Merit were awarded the following officers for services rendered on the occasion of an earthquake at Managua, Nicaragua, March 31, 1931:

Horace R. Boone, lieutenant commander (M.C.),
Warwick T. Brown, lieutenant commander (M.C.),
Gordon D. Hale, commander (M.C.).

The Haitian Government, on December 29, 1932, awarded Commander Walter C. Espach (M.C.), United States Navy, the Order of Honor and Merit in the rank of officer for distinguished service as officer of the Garde d'Haiti.

MERCUROCHROME AND IODINE SOLUTIONS AS LOCAL TISSUE DISINFECTANTS

Maj. J. S. Simmons, Medical Corps, United States Army, writing in Surgery, Gynecology, and Obstetrics of January 1933, presents experimental data concerning the relative bactericidal value of such mercurochrome and iodine solutions as are commonly used locally for the purpose of sterilizing the unbroken skin, oral mucous membranes, abrasions, and both superficial and deep wounds.

The report, according to the author, deals with the continuation of a study of local tissue antiseptics which was begun several years ago at the request of the Medical Supply Division of the Office of the Surgeon General, United States Army.

The summary and conclusion of the article follow:

Summary

1. Three types of wounds—skin abrasions, superficial incisions, and deep incisions—contaminated with undiluted broth cultures of either *Staphylococcus aureau* or *Streptococcus pyogenes* were treated for various periods of time with solutions of iodine and mercurochrome, respectively.

2. Application of tincture of iodine to 151 wounds contaminated with staphylococci resulted in sterile cultures as follows: Abrasions 83.4 percent; superficial incisions 83.1 percent; and deep incisions 31.2 percent; while its use on 59 wounds contaminated with streptococci resulted in sterilization as follows: Abrasions 75 percent; superficial incisions 80.9 percent; and deep incisions 82 percent. In brief, of 210 contaminated wounds treated with tincture of iodine, the cultures from 156, or 74.2 percent, were sterile.

3. Mercurochrome used under similar conditions caused relatively little reduction in the numbers of viable test organisms and failed to sterilize any of the 210 wounds.

Conclusions

The 2 percent aqueous solution of mercurochrome advocated for the first-aid treatment of wounds is a relatively weak antiseptic. When used experimentally for the destruction of *Staphylococcus aureau* or *Streptococcus pyogenes* in abrasions or incised wounds, it was decidedly less bactericidal than tincture of iodine. Mercurochrome is comparatively so ineffective in the sterilization of contaminated living tissues that it should not be considered as a substitute for iodine.

TREATMENT OF BURNS WITH GENTIAN VIOLET

J. H. Connell, T. J. Fatherree, C. B. Kennedy, and G. H. McSwain, New Orleans (Journal A.M.A., Apr. 22, 1933), report a series of 15 cases of burns treated with gentian violet, each patient having a first-, second-, or third-degree burn covering from 5 to 35 percent of the surface of the body. They treated 10 patients by the method of Alrich.[1] The remaining 5 patients were treated by their method, using a gentian violet jelly. They made complete cytologic and

[1] Alrich, R. H.: The Role of Infection in Burns: The Theory and Treatment with Special Reference to Gentian Violet, New England J. Med. 208: 299–309, Feb. 9, 1933.

chemical studies of the blood in all cases. Low-chloride and high-corpuscular concentrations were found, corroborating the observations of other workers. In every case in which the burned area occupied 10 percent or more of the surface of the body the chloride content, expressed as sodium chloride, was well under 400 milligrams per 100 cubic centimeters of blood, and the corpuscular concentration was over 56 percent. The regulation of these factors constituted an additional routine in the treatment of their patients. Dextrose and nonprotein nitrogen determinations and white and red blood-cell counts were made, which did not, however, give any information that was especially beneficial. The authors found no reports in the literature of blood-sedimentation rates in cases of burns, but they observed that this procedure gave them information of inestimable importance in carrying out the treatment and in establishing the prognosis. Of their 15 patients, 1 died. In all the other cases the end results were satisfactory. No contraction scars remained, the course of the patient was relatively fever free, and an early ambulatory condition resulted. In some cases infection appeared beneath the antiseptic dressing, manifested by softening, so that further applications of gentian violet were required. After several such experiences, in some of which many applications of the dye were necessary, the authors decided to incorporate the dye into a jellylike base. They added 30 grams of tragacanth to 1,000 cubic centimeters of a 1 percent aqueous solution of gentian violet. They placed a thick layer of this jelly on 4 or 5 sheets of gauze and applied the dressing to the burned area. Repeated applications were not necessary except in severe cases. A thin, moist, sterile protective layer was formed over the burned area, and rapid healing resulted. They conclude that the results they obtained from the latter treatment were far superior to those obtained from the use of the aqueous solution. In no case treated with gentian violet jelly did the fever rise to over 99.8° F., nor was any evidence of toxemia present.

STAINING METHOD FOR THE DIRECT TYPING OF PNEUMOCOCCI

F. C. O. Valentine, in the Lancet of January 7, 1933, gives the following simple and reasonably quick technic for the staining of the "neutralized" capsule in the direct typing of pneumococci:

> Three or four loopfuls of type I serum are mixed on a slide with a fair-sized loopful of sputum and mounted with a cover glass. The edges of the cover glass are sealed with vaseline, so that it will not become stuck to the slide. Similar preparations are made with types II and III sera. It is essential that complete neutralization of the capsule should be obtained, and to ensure this the preparations should be left for 20 to 30 minutes. After this the cover slips are slid off and discarded. The films left on the slides are allowed to dry and the bulk of the vaseline is scraped off with a knife. The vaseline remaining will not interfere

with the staining and blotting of the slides, and it may finally be removed by warming until it is just melted and washing over with xylol. Before staining wash the film under the tap to remove the serum, stain for 2 to 3 minutes with dilute carbol fuchsin, wash and counterstain for 10 seconds with carbol thionin; wash and blot dry.

It will be found that the bodies of all bacteria stain practically black, and that everything else is red. The capsules of pneumococci treated with the homologous serum appear quite large and stain a strong red, whereas the "unneutralized" capsule does not stain.

The dilute carbol fuchsin used has been filtered Ziehl-Neelsen stain freshly diluted in a test tube with 5 or 6 parts of water. The carbol thionin contains 9 parts 5 percent phenol in water and 1 part 50 percent alcohol saturated with thionin. In a new bottle this stain usually precipitates, but once the bottle is lined with deposit it lasts well. Saffranin may be used in place of carbol fuchsin, but the capsules are then less intensely stained. Tap water may be used for washing slides throughout.

Similar methods are very successful in typing the pneumococci in pus, but in this case a few cocci may be seen with lightly stained capsules in mixtures with the heterologous sera. This is probably due to the fact that the patient has had time to produce some antibodies, but it does not appear likely to cause confusion.

A STUDY OF THE INCIDENCE AND TIME DISTRIBUTION OF COMMON COLDS

W. H. Frost and M. Gover, in the United States Public Health Service weekly report of September 2, 1932, report the results of a study of the prevalence, crude symptomatology, and certain broad epidemiological features of the so-called "common colds."

The investigation was conducted by the United States Public Health Service in cooperation with the Department of Epidemiology of the Johns Hopkins University School of Hygiene and Public Health.

The summary of the study follows:

Data are presented on the incidence and certain epidemiological features of the minor respiratory diseases, as indicated by regular semimonthly reports rendered by rather large groups of students at several American universities in widely separated localities and by similar reports from some 1,500 families. The students' reports cover 18 months, and the family reports extend through 2½ years.

For the year ended May 30, 1925, the mean attack rate in the 10 groups of student reporters was 2,947 per 1,000, an average of approximately 3 attacks per person. For the entire period, and for each of its major seasonal subdivisions, the attack rates in the several student groups are remarkably uniform, showing no consistent relation to latitude, longitude, or climate.

In the family group, the attack rates in corresponding periods were consistently lower than in the student groups, but it is possible that this may have been due wholly or in part to more complete reporting by the students.

Both in the student and the family groups, the attack rates in corresponding seasons of successive years (1923–26) showed a declining trend. This may have been due, however, to progressive slackening of interest in reporting.

Taking the mean weekly attack rate throughout the year as an axis, the weekly attack rates in each group and in each year were quite consistently below this

level from about the first of April to the first of September, and generally above this level from September to March, inclusive. The minimum attack rates were observed usually in the latter half of July or the first half of August.

During the season of high prevalence, from September to March, inclusive, the incidence curve in each locality exhibited a series of oscillations, constituting a succession of epidemics, each of several weeks' duration, rather irregular in sequence and magnitude, but clearly not attributable to mere chance fluctuation.

These epidemics in six student groups in widely separated localities showed a striking time correspondence of about the same order as was observed in the influenza epidemics of 1918, 1919, and 1920.

Cases reported as influenza constituted about 5.6 percent of the total reported from the student groups from December 1923 to May 1925 and about 11.7 percent of those recorded in the family group for the same period of 18 months.

While the gross attack rates from all the minor respiratory disorders tended generally to decrease throughout the period of observation, the reported incidence of so-called "influenza" tended to increase, being highest in the winter of 1925–26.

The seasonal distribution of cases reported as influenza differed from that of cases classed (clinically) as coryza in that the latter reached their highest prevalence in the autumn, while the highest incidence of influenza occurred each year in the winter or spring months. Hence, the autumn epidemics observed each year differed from those observed in the late winter and spring in that the latter comprised larger proportions of cases classed as influenza.

The increased prevalence of so-called "influenza" observed in most of the student groups in the winter and spring of 1924–25 coincided generally with an increase in mortality from influenza-pneumonia in the cities represented. However, in individual cities the extent of the increase in mortality bore no obvious relation to that of the increase in prevalence of influenza.

SIXTY-SECOND ANNUAL MEETING AMERICAN PUBLIC HEALTH ASSOCIATION

The American Public Health Association, foremost sanitary organization in the United States, announces its sixty-second annual meeting, to be held in Indianapolis, Ind., October 9–12, 1933.

It was in Indianapolis in 1900 at the twenty-ninth convention of the American Public Health Association that Dr. Walter Reed read a paper entitled "The Etiology of Yellow Fever—A Preliminary Note" indicating that the mosquito serves as the intermediate host for the parasite of yellow fever. History was being made in the Old German House that day, yet it is reported by some of those present that the epochal report was received with only mild interest.

At the sixty-second annual meeting it is planned to honor the only living participant in the famous yellow fever experiment, Dr. John R. Kissinger, at a special memorial session.

The scientific program will discuss every aspect of modern public health practice, from the viewpoint of the health officer, the laboratory worker, the epidemiologist, the child hygienist, the industrial hygienist, the nurse, the vital statistician, the health educator, the

food and nutrition expert, the sanitary engineer. Distinguished scientific pronouncements may be expected from the outstanding personalities in the public-health profession who will contribute to the program.

The American Public Health Association, 450 Seventh Avenue, New York City, will be glad to send more complete information about its Indianapolis annual meeting to anyone interested.

THE DIVISION OF PREVENTIVE MEDICINE

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

TOXIC EFFECTS OF ARSENICAL COMPOUNDS EMPLOYED IN THE TREATMENT OF DISEASE IN THE UNITED STATES NAVY—1932 [1]

By O. J. Mink, Captain, 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 arsphenamine, neoarsphenamine, etc., administered. A separate account of every case in which ill effects are noted is also required.

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, and April 1933 numbers of the United States Naval Medical Bulletin and covered the subject up to and including the calendar year 1931. The present article supplements these and is in the interest of continuous published information relative to the ill effects experienced from the administration of arsenicals in the United States Navy. This installment takes into consideration the doses of arsenical compounds administered in the treatment of syphilis and diseases other than syphilis during the calendar year 1932 and includes some comparative figures from the experience of previous years.

It is the desired intention to present the Navy's arsenical experience by years, and we have endeavored to continue the classification of reactions in the manner adopted by Comdr. J. R. Phelps, Medical Corps, United States Navy, in his fourth contribution, which appears in the Division of Preventive Medicine section of the United States Naval Medical Bulletin of July 1930.

The "reactions" or ill effects encountered during the calendar year 1932 are as follows:

Encephalitis hemorrhagica	2
Vasomotor phenomena (shocklike, nitritoid, anaphylactoid, etc.)	38
Arsenical dermatitis and its complications	25
Exfoliative	18
Macular	3
Urticarial	2
Erythematous	1
Maculo-papular	1
Acute yellow atrophy of the liver	0
Jaundice	2

[1] Case histories of cases herein described are given as reported by the medical officers having cognizance of the case.

Acute renal damage	1
Ulcerative enteritis	0
Polyneuritis	0
Aplastic anemia	2
Herxheimer reactions	0
Jarisch-Herxheimer	2
Reactions of minor importance, including those in which there were insufficient data for classification	0
Total	72

The following table shows the number of doses of various arsenical compounds which were administered by naval medical officers in the treatment of syphilis and diseases other than syphilis during the 7-year period, 1925–31, the calendar year 1932, and the totals for the 8 years 1925–32, with the number and ratio of fatal and nonfatal toxic effects and of deaths to the number of doses administered:

	Number of doses administered	Mild reactions	Severe reactions	Fatal reactions	Total reactions	Ratio of reactions to doses. 1 reaction to—	Ratio of deaths to doses. 1 death to—
7-year period, 1925–31:							
Arsphenamine	30,389	25	13	1	39	779	30,389
Neoarsphenamine	495,312	269	110	22	401	1,235	22,514
Silver arsphenamine [1]	32	0	0	0	0	0	0
Sulpharsphenamine	9,120	3	3	0	6	1,520	0
Tryparsamide	19,725	1	0	0	1	19,725	0
Bismuth-arsphenamine-sulphonate [2]	58	0	0	0	0	0	0
Total	554,636	298	126	23	447	1,241	24,115
Year 1932:							
Arsphenamine	174	0	0	0	0	0	0
Neoarsphenamine	128,540	39	29	4	72	1,785	32,135
Silver arsphenamine	158	0	0	0	0	0	0
Sulpharsphenamine	2,614	0	0	0	0	0	0
Tryparsamide	6,783	0	0	0	0	0	0
Total	138,269	39	29	4	72	1,920	34,567
8-year period, 1925–32:							
Arsphenamine	30,563	25	13	1	39	784	30,563
Neoarsphenamine	623,852	308	139	26	473	1,319	23,994
Silver arsphenamine [1]	190	0	0	0	0	0	0
Sulpharsphenamine	11,734	3	3	0	6	1,956	0
Tryparsamide	26,508	1	0	0	1	26,508	0
Bismuth-arsphenamine-sulphonate [2]	58	0	0	0	0	0	0
Total	692,905	337	155	27	519	1,335	25,663

[1] First administrations were during the year 1931.
[2] First administrations were during the year 1929. The heading bismuth-arsphenamine-sulphonate is herewith included in lieu of the heading "Miscellaneous", which heading has been used in previous articles.

Referring to the above table, the ratios of reactions and deaths to doses administered for 1932 are: Reactions 1 to 1,920 and deaths 1 to 34,567. The averages for the 7-year period, 1925–31, are: Reactions 1 to 1,241 and deaths 1 to 24,115. The averages for the 8-year period, 1925–32, are: Reactions 1 to 1,335 and deaths 1 to 25,663.

The following table shows a recapitulation of the number of doses of arsenical compounds which were administered during the year 1932, to active-service personnel (U.S. Navy and U.S. Marine Corps) and to persons other than active-service personnel (all others) for syphilis and diseases other than syphilis:

Dosage	Arsphenamine		Neoarsphenamine		Silver arsphenamine		Sulpharsphenamine		Trypars-amide		Total
	U.S. Navy and Marine Corps	All others	U.S. Navy and Marine Corps	All others	U.S. Navy and Marine Corps	All others	U.S. Navy and Marine Corps	All others	U.S. Navy and Marine Corps	All others	
0.9 gram to 3 grams	0	0	0	0	0	0	0	0	1,928	4,840	6,768
0.9 gram	0	0	2,473	283	0	0	10	0	0	0	2,766
0.6 gram to 0.9 gram	0	0	50,358	9,594	0	0	14	4	0	0	59,970
Less than 0.6 gram	143	31	55,633	10,199	43	115	786	1,800	10	5	68,765
Total doses	143	31	108,464	20,076	43	115	810	1,804	1,938	4,845	138,269
Grand totals	174		128,540		158		2,614		6,783		138,269

The following table shows the number of fatal and severe reactions which followed the administration of 623,852 doses of neoarsphenamine during the 8 years, 1925–32, and the ratio of deaths and severe reactions to the total number of doses administered:

Complication	Number of fatal reactions	Ratio of deaths to total number of doses administered	Number of severe reactions with recovery	Ratio of severe reactions to total number of doses administered	Total fatal and severe reactions	Ratio of combined fatal and severe reactions to total number of doses administered
Hemorrhagic encephalitis	12	1 : 51,988	0	0 : 623,852	12	1 : 51,988
Vasomotor phenomena	5	1 : 124,770	35	1 : 17,824	40	1 : 15,596
Arsenical dermatitis	5	1 : 124,770	95	1 : 6,567	100	1 : 6,239
Acute yellow atrophy of the liver	1	1 : 623,852	0	0 : 623,852	1	1 : 623,852
Jaundice	0	0 : 623,852	6	1 : 103,975	6	1 : 103,975
Acute renal damage	0	0 : 623,852	1	1 : 623,852	1	1 : 623,852
Ulcerative enteritis	0	0 : 623,852	0	0 : 623,852	0	0 : 623,852
Polyneuritis	0	0 : 623,852	1	1 : 623,852	1	1 : 623,852
Aplastic anemia	3	1 : 207,951	4	1 : 155,963	7	1 : 89,122
Herxheimer	0	0 : 623,852	0	0 : 623,852	0	0 : 623,852
Total	26	1 : 23,994	[1] 142	1 : 4,393	168	1 : 3,713

[1] Explanatory note is made under similar table which is published in the NAVAL MEDICAL BULLETIN of July 1933, p. 179.

The following table shows the percent of arsenical reactions, by age groups, which occurred in the treatment of syphilis during the 3-year period 1930–32, in relation to the percent of syphilis admissions by age groups and the percent of enlisted men of the United States Navy and Marine Corps by age groups for the same 3-year period.

Age group	Percent of men in each age group	Percent of syphilis admissions in each age group	Percent of total reactions			Total reactions. Percent of reactions in relation to each age group
			Mild reactions	Severe reactions	Fatal reactions	
16–19	10.56	6.16	4.47	1.68		6.15
20–24	42.14	54.05	29.05	14.53	1.12	44.69
25–29	21.90	24.55	18.99	7.82	.46	27.37
30–34	15.39	10.65	7.82	7.82	1.12	16.76
35–39	6.67	3.14	2.79	1.12		3.91
40–44	2.40	1.15		1.12		1.12
45–49	.65	.21				
50–54	.17	.03				
55–59	.04	.01				
60–74	.01					
Total	99.93	99.95	63.13	34.08	2.79	100.00
	Total number of men in all age groups	Total number of admissions in all age groups	Total number of mild reactions in all age groups	Total number of severe reactions in all age groups	Total number of fatal reactions in all age groups	Total number of reactions in all age groups
	298,662	8,796	113	61	5	179

Deaths charged to the administration of arsenical compounds in the treatment of syphilis during the past 14 years were recorded as follows:

Year	Arsphenamine	Neoarsphenamine	Kind not specified	Total	Year	Arsphenamine	Neoarsphenamine	Kind not specified	Total
1919	2	0	1	3	1927	1	4	0	5
1920	1	1	0	2	1928	0	6	0	6
1921	3	1	0	4	1929	0	3	0	3
1922	0	4	0	4	1930	0	3	0	3
1923	0	1	0	1	1931	0	0	0	0
1924	1	2	0	3	1932	0	4	0	4
1925	0	2	0	2					
1926	0	4	0	4	Total	8	35	1	44

ANNUAL CENSUS OF PERSONS TREATED FOR SYPHILIS AND DISEASES OTHER THAN SYPHILIS

For years prior to 1931 the actual number of persons treated for syphilis is unknown for many reasons, the most important of which is the fact that each year there have been many persons treated who began their treatment in previous years. These persons are not necessarily readmitted to the sick list because of their current treatment.

In order to obtain information as to the number of persons in the naval service who had syphilitic histories, the Bureau of Medicine and Surgery revised N.M.S. Form A by Bureau of Medicine and Surgery Circular Letter No. 538, 1931, dated October 31, 1931, and included an annual section which directed that a census be taken of all

health records of service personnel on December 31, 1931, to determine how many persons had a history of syphilis; how many persons were treated for syphilis during the year with an arsenical compound, heavy metal, or other mixed treatment; and how many persons were treated during the year with an arsenical compound for diseases other than syphilis. Included in the instructions were directions to make a similar census regarding treatment accorded during the year to "all others", or persons other than active-service personnel (Veterans' Administration patients, beneficiaries, etc.).

As a result of this census the Bureau of Medicine and Surgery was able to tabulate information of interest to all naval medical officers. These tables were published in the UNITED STATES NAVAL MEDICAL BULLETIN, April 1933, page 181.

Individual reports of the annual census for the year 1932, taken on December 31, 1932, from all ships and shore stations, indicate that the reports were more carefully prepared than in the previous year, but many of the reports had to be returned for various corrections, etc., which leads to the conclusion that there is a doubt in the minds of some medical officers and hospital corpsmen as to the proper method of preparing this report.

The Bureau of Medicine and Surgery desires that all medical officers and all hospital corpsmen on independent duty bear in mind, when preparing N.M.S. Form A for the month of December, that the purpose of the annual census (reverse side of form A) is threefold:

1. To find the actual number of persons in the naval service who have a history of syphilis.
2. To find the number of all syphilitics in the naval service *who actually received treatment* during the year.
3. To determine what compounds were given to the persons actually treated during the year.

Hence, from the total number of persons who received, for example, neoarsphenamine, and already knowing the number of doses used in the service the average number of doses per person can be determined.

It follows that in order to avoid duplication of data *each person having custody of health records* must take the data *only from the health records actually in his custody on December 31st of the year covered by the report* and not from the treatment records of the activity.

In order to assure uniformly complete, accurate, and progressive health records the Bureau of Medicine and Surgery desires that all medical officers and all hospital corpsmen on independent duty make reference to the Manual of the Medical Department, United States Navy, 1927, paragraph 2331, subparagraphs (d) and (e).

The data for "All others", those persons who are other than active-service personnel, *must be taken* from the treatment records of the activity where the individual received treatment.

As a result of the census taken on December 31, 1932, the following tables show a recapitulation of data obtained. The first table shows a recapitulation of the number of persons treated for syphilis and the number of persons treated for diseases other than syphilis; the second table separates the diseases other than syphilis and shows the number of persons treated for each disease:

	U.S. Navy and Marine Corps (persons)	All others (persons)	Totals (persons)
Average strength, calendar year 1932	110,717	----------	110,717
Average strength, December 1932	105,558	----------	105,558
Syphilis census (persons)	13,791	----------	13,791
Number of persons treated for syphilis:			
Silver arsphenamine	0	18	18
Arsphenamine	8	26	34
Neoarsphenamine	8,761	2,423	11,184
Sulpharsphenamine	67	245	312
Tryparsamide	99	598	697
Mixed courses (bismuth, mercury, etc.)	4,907	1,617	6,524
Total persons treated for syphilis	13,842	4,927	18,769
Number of persons treated for diseases other than syphilis:			
Neoarsphenamine	255	1,388	1,643
Sulpharsphenamine	17	215	232
Acetarsone (stovarsol)	0	25	25
Acetarsone (spirocid)	0	123	123
Total persons treated for diseases other than syphilis	272	1,751	2,023
Grand total	14,114	6,678	20,792

Disease	Number of persons treated for diseases other than syphilis		
	U.S. Navy and Marine Corps (persons)	All others (persons)	Totals (persons)
NEOARSPHENAMINE			
Abscess, chronic, multiple	0	3	3
Acne	16	0	16
Adenitis, inguinal	4	0	4
Alopecia	1	0	1
Arthritis, gonorrheal	2	0	2
Cellulitis (location not stated)	1	0	1
Chancroid (location not stated)	3	0	3
Chancroid, abdomen	1	0	1
Dysentery, amoebic, chronic	3	0	3
Eczema	0	1	1
Furunculosis	13	0	13
Gangosa	0	5	5
Gonococcus infection, urethra	2	0	2
Impetigo contagiosa	1	0	1
Malaria	2	0	2
Prostatitis (nonvenereal), chronic	0	1	1
Provocative Kahn	13	82	95
Psoriasis	1	5	6
Ulcer, abdomen, chronic	0	1	1
Ulcer, leg	1	0	1
Vincent's infection	101	33	134
Vincent's infection (intravenous)	4	0	4
Vincent's infection (oral)	10	1	11
Vincent's infection (neoarsphenamine with glycerin)	27	20	47
Vincent's infection (neoarsphenamine with mercurochrome)	42	34	76
Yaws	7	1,202	1,209
Total	255	1,388	1,643

Disease	Number of persons treated for diseases other than syphilis		
	U.S. Navy and Marine Corps (persons)	All others (persons)	Totals (persons)
SULPHARSPHENAMINE			
Acne	3	0	3
Chancroid, penis	2	0	2
Epididymitis (nonvenereal)	12	0	12
Yaws	0	215	215
Total	17	215	232
ACETARSONE (STOVARSOL)			
Yaws	0	25	25
ACETARSONE (SPIROCID)			
Yaws	0	123	123
Grand total	272	1,751	2,023

The following table shows a recapitulation, for the year 1932, of the total number of persons (U.S. Navy, U.S. Marine Corps, and all others) treated with various arsenical compounds, the total number of doses of each compound administered, and the average number of doses per person.

	Number of persons treated (U.S. Navy, U.S. Marine Corps, and all others	Number of doses administered	Average number of doses per person
Silver arsphenamine	18	158	8.77
Arsphenamine	34	174	5.11
Neoarsphenamine	12,827	128,540	10.02
Sulpharsphenamine	544	2,614	4.80
Tryparsamide	697	6,783	9.73
Mixed courses (bismuth, mercury, etc.)	6,524	[1]	(?)
Acetarsone (spirocid and stovarsol)	[2] 148	[2] 447	3.02
Total for all diseases	20,644	138,269	6.69
Total for syphilis only	18,769	[3]	(?)
Total for diseases other than syphilis	1,875	[3]	(?)

[1] Data of number of doses of mixed treatment (bismuth, mercury, etc.) administered is not available as N.M.S. Form A does not require the reporting thereof.
[2] These figures are not included in the totals.
[3] Data of the number of doses administered in the treatment of syphilis and the number of doses administered in the treatment of diseases other than syphilis is not available as N.M.S. Form A does not require that these separations be made.

Hemorrhagic Encephalitis

Two cases of hemorrhagic encephalitis chargeable to the effects of arsenicals occurred during the year 1932. Both cases resulted in death. Case histories are as follows:

Neoarsphenamine.—(1-1932.) A patient was given a diagnosis on March 17, 1921, as the result of general glandular enlargement and three successive Wassermann blood tests which were reported as

four plus each. Treatment was instituted and was administered as follows:

March 17, 1921, to May 16, 1921, 12 injections of arsenicals, amounts not stated; and 22 one half grain injections of mercury succinimide.

September 2, 1926, to November 26, 1926, 7 injections of arsenicals, amounts not stated.

December 14, 1926, to January 4, 1927, 4 injections of arsenicals, amounts not stated.

March 9, 1927, to June 7, 1927, 8 injections of arsenicals, amounts not stated.

August 30, 1927, to October 18, 1927, 8 injections of arsenicals, amounts not stated; and daily inunctions of mercury.

November 1, 1927, to ———, 3 injections of arsenicals, amounts not stated.

February 25, 1928, to April 30, 1928, 9 injections of arsenicals, amounts not stated.

September 12, 1928, to February 6, 1929, 7 injections of arsenicals, amounts not stated; and 7 injections of mercury.

August 1, 1929, to September 15, 1929, 6 inunctions of mercury.

February 3, 1931, to March 24, 1931, 8 injections of bismuth.

May 5, 1932, to August 25, 1932, 17 intramuscular injections, 0.1 gram each, of bismosol.

The patient began his ninth course of arsenical treatment on August 31, 1932, and he received a 0.45 gram intravenous injection of neoarsphenamine on that date. On September 7, 14, 21, and 28 he received 0.45 gram intravenous injections. Following the injection on September 28 he had a slight reaction which consisted of nausea and abdominal discomfort. He did not report again for treatment until October 19, 1932, when he was given a 0.2 gram intravenous injection of neoarsphenamine. The dilution of this injection was stated as 0.45 gram of neoarsphenamine dissolved in 10 cubic centimeters of water with a 0.2 gram dose administered. The rate of injection was stated as 30 seconds for each 0.1 gram of the drug administered.

Immediately after receiving the last injection of neoarsphenamine, given on October 19, the patient felt all right. Upon investigation it was learned that he had gone home about noon and to bed with a remark to one of his neighbors that he felt sick and nauseated and had a pain in his back. At 4:05 p.m. a telephone call was received at the sick bay stating that the patient was at his home and sick. An ambulance was immediately sent and on the return trip the patient talked to the driver of the ambulance and smoked a cigarette. Just before the ambulance reached the sick bay he suddenly collapsed and his skin became pale and covered with perspiration. When the ambulance arrived at the sick bay at 4:55 p.m. the patient was unconscious, respiration had ceased, lips were cyanotic, skin was clammy, and the pulse was almost imperceptible. He was given artificial respiration and 1 cubic centimeter injection of adrenalin into the neck vein, but the heart ceased beating at 5:05 p.m., about

8 hours and 35 minutes after the injection of neoarsphenamine. Two cubic centimeters of adrenalin were injected directly into the heart and artificial respiration was continued for some time but without results.[2]

The autopsy findings in brief were: Intense hyperemia of all abdominal and thoracic viscera and brain, with normal amounts of blood tinged pleural, spinal, and abdominal fluids; marked capillary engorgement; large vessels depleted and heart empty. Blood showed no tendency to coagulate.

Pathological and chemical examinations were as follows:

Gross examination—Specimen consists of tissues from autopsy received in frozen state by use of "dry ice."

Microscopical examination—Kidney.—Section shows a markedly and acutely congested kidney. The small vessels and capillaries are distended with blood and there is some extravasation into the interstitial tissue. Capillary loops in many of the glomeruli are distended with blood. There is marked granular degeneration of epithelium of the convoluted tubules. Cellular outline is obscured. The lumen of many of the tubules is filled with cellular debris.

Lungs.—Sections show marked congestion in the alveolar walls with areas of extravasation in the alveoli. Some areas show alveoli containing pigmented macrophages. Marked edema is present, both in the alveoli and in the alveolar wall, being more pronounced in the latter, causing the walls to appear greatly thickened and distended.

Spleen.—The spleen is markedly congested. The sinusoids are distended with blood which for the most part is fairly well hemolyzed leaving only the outline of the cells. The malpighian corpuscles are more pronounced than normal. Reticulo-endothelial cells contain considerable brown pigment. Eosinophiles are fairly numerous.

Liver.—The liver cords appear on section as abnormally thin strands, leaving what appears to be widely dilated sinusoids. The sinusoids contain red cells, some well preserved and others completely hemolyzed, leaving only remnants of the cell. The most markedly dilated sinusoids are free of cellular elements, and it is probable, in view of the generalized acute congestion, that they have dropped out in the process of freezing, thawing, and fixing. The cells of the liver cords show marked granular degeneration with abolition of all cellular outline.

Pancreas.—Shows such marked post-mortem change that any ante-mortem change is obscured.

Heart.—Heart muscle is pale and shows some post-mortem change. Cross striations are fairly distinct.

[2] The medical officer reported that 0.45 gram of the drug from the same ampule was given to another man and it caused no reaction.

Bone marrow.—There is congestion of the sinusoids and capillaries. There appears to be a decrease in the cellular elements and many cells are necrotic or show marked degeneration.

Blood from the heart.—Blood fails to coagulate and smears show complete hemolysis of the erythrocytes and degeneration of the leukocytes, making it impossible to examine the cellular elements.

Brain.—Sections from the cortex, pons, and medulla and basal ganglion show considerable post-mortem change. An occasional field shows congested cerebral vessels with extravasation of erythrocytes in the adjacent brain tissue and small areas of necrosis.

Chemical examination.—"The stomach and contents, and liver were examined in accordance with generally accepted principles of toxicology and found negative for common poisons other than arsenic."

Arsenic was found to the extent of 2.4 milligrams (as arsenic trioxide) in 320 grams of liver substance.

Pleural cavity fluid for van den Bergh.—Indirect reaction 3.0 milligrams per 100 cubic centimeters.

Abdominal cavity fluid for van den Bergh.—Indirect reaction 2.66 milligrams of bilirubin per 100 cubic centimeters.

Pathological diagnosis.—

Hemorrhagic encephalitis.
Edema—lungs.
Acute parenchymatous degeneration—liver and kidneys.
Acute passive congestion—liver, kidneys, spleen, lungs.
Post-mortem degeneration—pancreas.
Acute hypoplastic bone marrow.

These findings are compatible with neoarsphenamine poisoning.

Chemical findings.—Arsenic was found to the extent of 2.4 milligrams (as arsenic trioxide) in 320 grams of liver substance.

(2-1932.) A patient was exposed in December 1922 at New York City. A penile lesion appeared and a Kahn blood test was reported 4 plus in January 1923. A Kahn blood test taken August 4, 1923, was reported as "very weakly positive." From March 2, 1925 to August 19, 1931, five Kahn blood tests were reported negative. No further entries about syphilis were noted in the health record but apparently the patient had a reinfection in February 1932, as a notation was made on the medical history sheet to the effect that a Kahn blood test, taken on February 29, 1932, was reported as 4 plus. Another notation was made on the medical history sheet, under date of March 4, 1932, to the effect that a sore was noted on the penis. No further comments or histories were made.

The following arsenical treatment was administered from the time of the original admission for syphilis until the date of the last injection, i.e., the date of the injection administered prior to arsenical reaction:

First course, December 18, 1922, to January 26, 1923, 6 injections, salvarsan, amount not stated.

Second course, February 6, 1923, to February 13, 1923, 2 injections, salvarsan, amount not stated.

Third course, August 23, 1923, to ———, 12 injections, salvarsan, amount not stated.

Fourth course, December 15, 1924, to May 16, 1925, 9 injections, salvarsan, amount not stated.

Fifth course, January 13, 1927, to April 20, 1927, 9 injections, salvarsan, amount not stated.

Sixth course, December 1, 1927, to January 12, 1928, 6 injections, salvarsan, amount not stated.

Seventh course, September 15, 1928, to November 3, 1928, 8 injections, neoarsphenamine, total 6.25 grams.

Eighth course, August 3, 1929, to October 29, 1929, 7 injections, salvarsan, amount not stated.

Ninth course, July 30, 1930, to ———, 1 injection, salvarsan, amount not stated.

REINFECTION

Tenth course (first)[a], March 14, 1932, to April 26, 1932, 8 injections, neoarsphenamine, total 4.50 grams.

Eleventh course (second)[a], July 2, 1932, to August 20, 1932, 8 injections, neoarsphenamine, total 3.75 grams.

Twelfth course (third)[a], October 18, 1932, to November 1, 1932, 3 injections, neoarsphenamine, total 1.12 grams.

The twelfth course (third course after reinfection) consisted of a 0.3 gram intravenous injection of neoarsphenamine administered on October 18, 1932, a 0.45 gram injection administered on October 25 and a 0.5 gram injection administered on November 1, 1932. The dilution of the last injection, administered on November 1, was 0.5 gram of neoarsphenamine to 10 cubic centimeters of sterile triple-distilled water and the rate of injection was 3 minutes for the 10 cubic centimeters of solution administered.

The following intercurrent and concurrent treatment was administered:

December 18, 1922, to January 26, 1923, 6 injections, mercury.
February 6, 1923, to February 13, 1923, 2 injections, mercury.
December 15, 1924, to May 16, 1925, mercury inunctions.
January 13, 1927, to April 20, 1927, 13 injections, mercury.
December 1, 1927, to January 12, 1928, 12 injections, mercury.
September 15, 1928, to November 3, 1928, mercury inunctions.
August 3, 1929, to October 29, 1929, mercury inunctions.
May 4, 1932, to June 21, 1932, 8 injections, bismuth salicylate, 0.13 gram each.
August 16, 1932, to October 19, 1932, 10 injections, mercury succinimide, 0.026 gram each.

The patient had a history of a cough and head cold on October 31, 1932. He did not complain or seek treatment. The following day he received a 0.5 gram intravenous injection of neoarsphenamine

[a] For statistical purposes it has been considered that the patient had a reinfection and that the third injection of the third course of neoarsphenamine, after reinfection, was the contributory cause of the reaction.

and that evening he had a fever, ached over his body and back, had a cough, etc. There was no diarrhea, nausea, vomiting, or rash, and no history of a previous arsenical reaction. The next day, November 2, he was given an ounce of magnesium sulphate, 10 grains of aspirin every 4 hours, and a nasal spray every 4 hours. He was transferred to a naval hospital on November 3 with a diagnosis undetermined (influenza), because he complained of "aches over his body, fever, cold in his head, and a cough." His temperature was 100.2° F., pulse 106, and respiration 18. The blood pressure was 112/82. His pharynx was injected and scattered moist rales were heard in both lungs. The diagnosis was changed this date to catarrhal fever. At noon the next day, November 4, he talked rationally and stated that he felt well. He slept most of the afternoon and seemed drowsy and difficult to arouse but he ate his supper and answered questions readily. At the evening sick call his condition was not considered unusual by the officer of the day, who recalled that he awakened the man and asked him how he felt. At 8 a.m. the next morning, November 5 (approximately 94½ hours after the injection of neoarsphenamine), the patient was found to be stuporous and comatose. His temperature was 99.6° F., pulse 74, and respiration 18–26 (varying in rate and depth). His pupils were small and reacted sluggishly to light. His neck was rigid, the head was drawn back, and he had a vertical nystagmus. Brudzinski's sign was positive. He had tremors of the tongue and tonic spasm of the right hand and wrist. Abdominal reflexes were absent. Bilateral Kernig. Hyperactive K-J. Positive Babinski and Oppenheim on both sides. The spinal fluid was clear, pressure decreased, and no increase on jugular compression. The urine showed arsenic by Autenrieth's test. Diagnosis was changed to "Poisoning, neoarsphenamine, acute (antisyphilitic)", and then to "Encephalitis, acute (hemorrhagic)." Symptoms of increased intracranial pressure became more marked, coma deepened, convulsive movements of the tongue, right hand and wrist (at first tonic, then clonic) occurred, and the temperature gradually rose to 109.5° F. (by axilla) before he died. The blood Kahn and spinal Kahn were negative. The spinal fluid globulin was increased. At 8:30 a.m. a spinal puncture revealed partial block of both sides, and there was evidence of increased intracranial pressure. Blood sugar test showed 214 milligrams of sugar per 100 cubic centimeters of blood. The urine revealed a trace of sugar. The patient was given 1½ ounces of magnesium sulphate by mouth and 300 cubic centimeters of a 35 percent solution of magnesium sulphate as a retention enema for 1 hour. At 4 p.m. he was given 500 cubic centimeters of a 5 percent solution of glucose intravenously and 100 units of insulin. At 7 p.m. he was given an intravenous injection of sodium thiosulphate, 1 gram in 10 cubic centimeters of sterile water. At 9

p.m. he was given a hypodermic injection of 1 grain of caffeine sodium benzoate. The patient's condition gradually became worse, and death occurred at 10:13 p.m., 108 hours and 43 minutes after the injection of neoarsphenamine.

A post-mortem examination made the following day, November 6, revealed the following: Hyperemia of the liver, spleen, and kidneys. The brain was edematous with increased cerebrospinal fluid, congested meninges and dura, small hemorrhagic areas over the hemispheres. The cerebellar lobes were jammed into the foramen magnum, especially on the left side. A well-defined pressure cone on the cerebellum was seen where this herniation into the foramen magnum had occurred.

Cases suggesting border-line relationship between acute hemorrhagic encephalitis and the other forms of acute poisoning by arsenical compounds used in the treatment of syphilis.—During the year 1932 one fatal reaction [3] occurred which seems to fall under this heading or classification.

(3-1932.) A patient was exposed at San Francisco, Calif. He was given a diagnosis of syphilis on January 5, 1932, because darkfield examinations of the serum from the lesion were positive for *Treponema pallidum*.

Arsenical treatment was instituted and the patient was administered 0.3 gram intravenous injections of neoarsphenamine on February 16 and 19, 1932. On February 23 and March 1, 1932, he was given 0.6 gram intravenous injections of neoarsphenamine. The dilution of the injection given on March 1 was 0.9 gram of neoarsphenamine dissolved in 20 cubic centimeters of sterile distilled water and the rate of injection was stated as 2 minutes for the 13 cubic centimeters (0.6 gram dose) of the solution administered. The total dosage to date of this first course of arsenicals was 1.8 grams of neoarsphenamine which was administered over a period of 15 days, or an average of 120 milligrams per day. Five hours after the administration of the last injection of neoarsphenamine the patient complained of a dull headache, general aching, and a feeling of torpor. He was ordered to bed; temperature was recorded as 99.4° F. and pulse 90. Examination revealed no erythema or other skin manifestation and no acute distress. He was given forced fluids, liquid diet, and placed under observation. The urinalysis made previous to the injection of neoarsphenamine was reported negative. During that evening and night the patient was quiet.

The following morning, March 2, he was found to be acutely ill with a temperature of 104° F., and pulse of 110. He was restless, somewhat confused, and appeared to be toxic and dull. When questioned he answered that he "felt rotten and all in". Examina-

[3] This case is included in the figures for vasomotor phenomena for statistical purposes.

tion revealed no edema, normal conjunctivae and sclerae, no tenderness of the liver or spleen, a pulse that was full, firm, regular, and in rhythm but rapid, and normal respiration. Blood counts were taken and were reported as follows: White blood count, 13,500; Differential: polymorphonuclear leukocytes, 78 percent; small lymphocytes, 10 percent; large lymphocytes, 8 percent; transitionals, 2 percent; and monoculears, 2 percent. The patient stated that he had urinated about one half hour previously. At 11 a.m. the patient was given 1 gram of sodium thiosulphate which was dissolved in 25 cubic centimeters of sterile distilled water. About 1 hour later no change in condition was noted. At 1 p.m. the patient was transferred to a naval hospital for observation and treatment. Upon admission to the hospital an examination revealed the patient to be drowsy and apathetic. There was some edema of the eyelids with purplish-blue discoloration; a circumoral pallor with some pigmentation of the face and neck in spots which resembled large freckles; a blood pressure of 110/20 (diastolic impulse audible to the bottom of the scale); a pulse rate of 110, regular in rate, rhythm, and intensity but of weak and easily compressible quality; sclerae of a muddy appearance; injection of the conjunctivae; posterior cervical adenitis; and exaggerated deep and diminished superficial reflexes but no Kernig, Babinski, ankle clonus, or Brudzinski. Laboratory examinations were reported as follows: Red blood count, 4,570,000; white blood count, 18,800; hemoglobin, 80 percent; polymorphonuclears, 81 percent; and lymphocytes 19 percent. Malarial parasites were not found. At 4 p.m. the patient was given a subcutaneous injection of 1 cubic centimeter of adrenalin; at 5 p.m. an intravenous injection, 20 cubic centimeters of a 50 percent solution of glucose; at 6 p.m. a 1-gram intravenous injection of sodium thiosulphate; at 7 p.m. a 1 cubic centimeter subcutaneous injection of adrenalin and a double seidlitz powder; and at 10:05 p.m. a 1-gram intravenous injection of sodium thiosulphate.

The next day, March 3, the patient showed some apparent improvement in the morning. The blood pressure was recorded as 110/60. His temperature was 103° F., pulse 134, and respiration 36. Laboratory examinations were recorded as follows: Red blood count 4,010,000, white blood count 13,700, hemoglobin 75 percent, polymorphonuclears 85 percent, and lymphocytes 15 percent. The patient had now developed a croupous nonproductive cough and an examination revealed definite impaired resonance over the right lower lobes, sharp chest pains which were aggravated by inspiration, and diminished breath sounds and fremitus. Radiographic examination revealed some collapse of the lower and middle lobe with fluid in the pleural sac. At 7 p.m. the symptoms and signs were those of a developing pneumonic process in spite of the white blood count of 7,600 and a differential count showing polymorphonuclears 63 percent, lympho-

cytes 26 percent, large mononuclears 1 percent, and transitionals 1 percent. During the day the following treatment had been administered: At 9 a.m. the patient was given an intravenous injection of 1 gram of sodium thiosulphate; at 11:30 a.m., a hypodermic injection of 1 cubic centimeter of adrenalin hydrochloride; at 8 p.m. he was started on a proctoclysis, the rate of flow being 20 drops to the minute.

The following day, March 4, the diagnosis was changed to pneumonia, broncho, as the result of complications. The patient was given forced fluids and sedatives. Throughout the day little change was noted and at times the patient seemed irrational. His temperature was recorded as 103.8° F., pulse 120, and respiration 26. Laboratory examination of the spinal fluid, which was made the previous day, was Kahn negative, globulin no increase, and cell count 4. Blood counts were reported as follows: White blood count 8,550, polymorphonuclears 74 percent, lymphocytes 25 percent, and large mononuclears 1 percent. Treatment administered throughout the day was as follows: At 2:05 a.m. the patient was given ½ grain of codeine for his cough; at 6:30 a.m. he was given a second dose of ½ grain of codeine; at 8 a.m. he was given a hypodermic injection of morphine sulphate, ¼ grain, and atropine, $\frac{1}{150}$ grain; at 3:15 p.m. the patient was catheterized and 1,150 cubic centimeters of urine was obtained; at 3:50 p.m. he was given $\frac{1}{100}$ grain of atropine; and at 4 p.m. he was given an intravenous injection of 1 gram of sodium thiosulphate.

The patient was distinctly worse the next day. He became cyanotic and had labored breathing, chest pains, and cough. His temperature was 102.2° F., pulse 108, and respiration 22. At times he became quite stuporous, talking and moaning incoherently. Throughout the day he took fluids well and accepted small amounts of nourishment. Laboratory examination of urine reported the specimen to contain a few coarse granular casts and to be 4 plus for albumin. The blood counts taken during the day were reported as follows: Red blood count 3,880,000; white blood count, 4,300; hemoglobin 70 percent; polymorphonuclears 63 percent, lymphocytes 36 percent, and large mononuclears 1 percent. The patient was administered ½ grain of codeine at 2:45 a.m. for his cough. At 8:15 a.m. he was given 1 ounce of magnesium sulphate; at 8:35 a.m. an intravenous injection of 1 gram of sodium thiosulphate; at 10:15 a.m. a hypodermic injection of morphine sulphate, ¼ grain, and atropine $\frac{1}{150}$ grain; at 11:25 a.m. a blood transfusion of 450 cubic centimeters of whole blood with beneficial results; at 3 p.m. another dose of ½ grain of codeine for his cough; at 8 p.m. a third dose of ½ grain of codeine; and at 9:15 p.m. he was given a second hypodermic injection of morphine sulphate, ¼ grain, and atropine $\frac{1}{150}$ grain.

The next day, March 6, little change was noted in his condition. He did not void voluntarily and at 3:40 p.m. he was catheterized and

1,220 cubic centimeters of urine were obtained. At 11:30 p.m. he was again catheterized and 320 cubic centimeters of urine were obtained. The patient took liquids and was given an enema for distention with good results. His temperature was recorded as 102.6° F., pulse 120, and respiration 32. Blood counts taken this date were reported as follows: Red blood count 4,200,000; white blood count 5,750; hemoglobin 75 percent; polymorphonuclears 73 percent; and lymphocytes 27 per cent. Urinalysis was reported as cloudy amber color, reaction acid, specific gravity 1.017, albumin 4 plus, sugar negative, and very few leukocytes. Throughout the day the following treatment was administered: At 4 a.m. the patient was given ½ grain of codeine sulphate; at 8:40 a.m., a hypodermic injection of atropine, $\frac{1}{150}$ grain, and morphine sulphate, ¼ grain; at 8:50 a.m., an intravenous injection of 1 gram of sodium thiosulphate; at 11:20 a.m., a blood transfusion of 500 cubic centimeters of whole blood, which was followed by some reaction of short duration; at 11:30 a.m., a second hypodermic injection of morphine sulphate, ¼ grain, and atropine, $\frac{1}{150}$ grain; at 1:05 p.m., atropine sulphate, $\frac{1}{150}$ grain; and at 7:20 p.m., a third injection of morphine sulphate, ¼ grain, and atropine sulphate, $\frac{1}{150}$ grain.

The patient became delirious and restless toward the end of the next day (March 7). His respirations were 36 and labored. Cyanosis was increasing and radiograph of the left lower lobe revealed an extensive broncho-pneumonic process. Blood counts were reported as follows: Red blood count 4,680,000; white blood count 8,400; hemoglobin 75 percent; polymorphonuclears 82 percent; lymphocytes 17 percent; and large mononuclears 1 percent. Urinalysis reports were as follows: Color, cloudy dark amber; reaction—first specimen neutral, second specimen slight alkaline; specific gravity, first specimen 1.018, second specimen 1.017; albumin 4 plus; sugar negative; a few triple phosphate crystals in the second specimen. The following treatment was administered throughout the day: At 3:30 a.m. the patient was given ½ grain of codeine; at 9 a.m., an intravenous injection of 1 gram of sodium thiosulphate; at 10 a.m., a hypodermic injection of morphine sulphate, ¼ grain, and atropine sulphate, $\frac{1}{150}$ grain; at 5 p.m., a hypodermic injection of morphine sulphate, ¼ grain, and atropine sulphate, $\frac{1}{150}$ grain; at 9:40 p.m., a hypodermic injection of digitaline, $\frac{1}{100}$ grain; at 9:45 p.m., an ounce of whisky; and at 10:35 p.m. he was given a hypodermic injection of morphine sulphate, ¼ grain, and atropine sulphate, $\frac{1}{150}$ grain.

The following day, March 8, the patient was rapidly declining and he had no periods of lucidity. His temperature was 105° F., pulse 140, and respirations 40. Abdominal distention was relieved by turpentine stoops and enema. At 1:40 a.m. he was given a hypodermic injection of digitaline, $\frac{1}{100}$ grain; at 2:30 a.m., a hypodermic injection of morphine sulphate, ¼ grain, and atropine sulphate,

1/150 grain; at 5:25 a.m., a hypodermic injection of digitaline, 1/100 grain; at 6 a.m., an intravenous injection of 1,000 cubic centimeters of normal saline solution with glucose; and at 6:50 a.m. he was given atropine sulphate, 1/100 grain. The patient's respirations were becoming more labored, irregular, and shallow. The pulse was very rapid, full, and bounding. Some pulmonary edema was apparently developing. He gradually became worse and death occurred at 7:25 a.m., about 7 days after onset of symptoms of reaction.

Proportion of deaths from hemorrhagic encephalitis to the number of persons treated for syphilis, and to the number of doses of arsenical compounds administered for all diseases.—For years previous to 1931 the actual number of persons treated for syphilis is unknown for many reasons. These reasons are stated under the subject, "Annual Census of Persons treated for Syphilis and Diseases other than Syphilis", which is printed on previous pages of this article.

An annual census for the year 1931 disclosed that 16,235 persons (U.S. Navy, U.S. Marine Corps, and all others) were treated for syphilis. As there were no deaths from hemorrhagic encephalitis in 1931 which could be chargeable to arsenicals, the proportion of deaths from hemorrhagic encephalitis to the number of persons treated for syphilis would be 0 to 16,235.

The annual census for the year 1932 disclosed that 18,769 persons (U.S. Navy, U.S. Marine Corps, and all others) were treated for syphilis. In 1932 there were 2 deaths from hemorrhagic encephalitis chargeable to arsenicals. The proportion of deaths from hemorrhagic encephalitis to the number of persons treated for syphilis is therefore 1 to 9,385.

For the 2 years, 1931–32, there were 35,004 persons (U.S. Navy, U.S. Marine Corps, and all others) treated for syphilis. During this period of time there were 2 deaths from hemorrhagic encephalitis chargeable to arsenicals. The proportion of deaths from hemorrhagic encephalitis to the number of persons treated for syphilis is 1 to 17,502.

During the year 1932 there were 174 doses of arsphenamine and 128,540 doses of neoarsphenamine administered with 2 deaths due to hemorrhagic encephalitis.

During the 8-year period, 1925–32, there were 30,563 doses of arsphenamine administered with 1 death due to hemorrhagic encephalitis; during the same period of time there were 623,852 doses of neoarsphenamine administered with 12 deaths due to hemorrhagic encephalitis, giving a ratio of 1 death to 51,988 doses of neoarsphenamine administered.

During the 8-year period, 1925–32, there were 11,734 doses of sulpharsphenamine and 26,508 doses of tryparsamide administered with no deaths from any cause referable to the arsenical compound.

Vasomotor Phenomena

In previous articles on this subject covering the years up to and including the year 1932, 198 cases reported by medical officers have been grouped as examples of vasomotor phenomena following the intravenous injection of arsenical compounds.

During the year 1932 there were 38 cases reported, 1 of which (fatal case no. 3-1932) has been listed above under reactions of borderline relationship between acute hemorrhagic encephalitis and other forms of acute poisoning by arsenical compounds. The remaining 37 reactions were classified as 30 mild reactions and 7 severe reactions. No reaction classed under vasomotor phenomena resulted in death.

Deaths.—As previously mentioned, there was no death during the year 1932 that can be attributed to vasomotor phenomena following the intravenous injection of an arsenical compound.

For statistical purposes, however, we have included the death (case no. 3-1932), which is listed on previous pages under borderline reactions, under the heading of vasomotor phenomena. With the inclusion of this death the ratio of deaths (vasomotor phenomena) to the number of doses of neoarsphenamine administered during the 8-year period, 1925–32, is 1 death to 124,770 doses administered. The ratio of deaths (vasomotor phenomena) to the number of doses of arsphenamine administered during the 8-year period, 1925–32, is 1 death to 30,563 doses administered.

Nonfatal cases of arsphenamine and neoarsphenamine poisoning classed as vasomotor phenomena (1932)

Arsphenamine.—No reactions were reported as having resulted from the intravenous injection of 174 doses of arsphenamine.

Neoarsphenamine.—Of the 37 reactions reported during the year 1932, 27 occurred at different times and on different ships and stations; the remaining 10 include one "flare up" which was believed to be due to a cause not directly chargeable to the drug (possibly water contamination). These 37 reactions are presented below in two groups, consisting of 27 and 10 reactions, respectively, with comparative data given in tabular form:

FIRST GROUP—27 CASES

Key[1]	Course of treatment	Injection	Dose in grams, neoarsphenamine	Onset after injection	Duration or reported time of recovery	Apparent first symptoms
				Hours		
5	First [2]	First	0.3	[3] 7	4 days	Extreme emotional instability, chills, fever, dizziness.
24	do	do	.45	8	8 hours	Severe chills, rapid pulse, skin flushed, warm and dry, fever.
4	do	Second	.3	6	13 days	Headache, chill, fever, nausea, vomiting, macular rash.
8	do	do	.6	([4])	14 days	Headache, vomiting, fever.
18	do	do	.45	72	5 days	Hyperemic rash, sore throat, conjunctivitis, pharyngitis, fever, rapid pulse.
30	do	do	.3	12	6 days	Urticarial rash, intense itching of skin, eyelids and lips swollen.
7	do	Third	.6	2	2 days	Nauseated, vomited, chills, headache, fever, rapid pulse, face flushed, pupils dilated, erythema on face and chest.
15	do	do	.6	1½	1 day	Severe headache, general malaise, fever, nausea, abdominal pain.
20	do	do	.6	½	2 days	Nervousness, weakness, fever.
23	do	do	.45	[3] 4	3 days	Severe chill, fine scarlet rash, fever, rapid pulse.
29	do	Fourth	.6	1	10 days	Fever, nausea, slight delirium, slight erythema, and swelling of the extremities.
22	do	Ninth	.45	1	3½ hours	Headache, chills, backache, rapid pulse.
12	Second	Second	.6	1	2 hours	Headache, nausea, severe chill, pallor.
17	do	Fourth	.2	1/20	½ hour	Dizziness, flushing of face, cough.
26	do	do	.3	½	About 13 hours	Skin cold and clammy, subnormal temperature, rapid pulse (feeble).
11	Third	First	.45	1	2 hours	Headache, nausea, severe chills, pallor.
28	do	Third	.45	1½	12 hours	Nervous, skin flushed, dazed, chilly, cyanosis, rapid pulse, abdominal pain, fever.
14	do	Sixth	.6	10	2 days	Intense occipital headache, vomiting, weakness, chills, slight jaundice of sclerae, rapid pulse.
10	do	Ninth	.6	[3] ½	23 days	Chills, weakness, nausea, headache, fever, rapid pulse.
16	Fourth	First	.45	2	17 days	Urticarial areas on the flexor surface of the forearms and abdomen.
6	do	Second	.5	1	3 days	Pain in legs, nausea, vomiting, spitting of blood, chills, severe supraorbital headache.
27	do	do	.3	2	8⅙ hours	Slight headache, general malaise, aches, chills, fever, rapid pulse, sweating.
21	do	Fourth	.45	2	24 hours	Frontal headache, chills, dizziness, pallor, rapid pulse, sweating, shallow respirations, lowered blood pressure.
9	do	Fifth	.6	[3] 20	17 days	Severe pains in abdomen, nausea, vomiting, fever, rapid pulse.
25	do	do	.225	1/12	3 hours	Prostration and temporary complete disability.
19	Sixth	Second	.6	1/30	4 hours	Weak, gagged, skin cold and clammy, extremities flushed with slight cyanosis, rapid pulse (feeble and irregular).
13	Seventh	Fourth	.6	1	2 hours	Headache, nausea, severe chill, pallor.

[1] "Key" refers to individual case number.
[2] This patient had completed 3 courses after original infection. He was reinfected and had just received his first injection of the first course after reinfection when the reaction occurred.
[3] Approximately.
[4] Immediate reaction.

SECOND GROUP—10 CASES

Key[1]	Course of treatment	Injection	Dose in grams, neoarsphenamine	Onset after injection	Duration or reported time of recovery	Apparent first symptoms
				Hours		
31	Third	First	0.3	[2]	Within 24 hours	Chill, headache, general malaise, nausea, vomiting.
32	do	do	.3	[2]	do	Chill, headache, general malaise, nausea.
33	do	do	.3	[2]	Within 12 hours	Headache, general malaise.
34	Fourth [3]	do	.3	[2]	Within 24 hours	Chill, headache, general malaise, vomiting.
35	Fourth	do	.3	[2]	Within 18 hours	Headache, general malaise, nausea.
36	do	do	.3	[2]	do	Headache, chill, malaise, nausea.
37	Fifth	Fourth	.6	[2]	Within 24 hours	Chill, headache, general malaise, vomiting.
38	Sixth [4]	First	.3	[2]	do	Headache, general malaise.
39	Seventh	do	.3	[2]	do	Headache, chill, general malaise, vomiting.
40	Eighth	do	.3	[2]	do	Chill, headache, general malaise, vomiting.

[1] "Key" refers to individual case number.
[2] The time elapsing between the injection and the first symptoms of reaction was approximately 15 minutes in all 10 cases.
[3] This patient had completed 3 courses after his original infection. He was reinfected and had completed 3 courses after reinfection.
[4] This patient had completed 3 courses after his original infection. He was reinfected and had completed 5 courses after reinfection.

Temperature.—In the 27 reactions included above in the first group, an elevation of temperature of over 102° was reported in 8 cases, an elevation of temperature of 100° to 102° was reported in 4 cases, a temperature of less than 98.6° was reported in 2 cases, fever was reported in 2 cases but the degree was not mentioned, and in the remaining 11 cases the temperature was not reported.

In the 10 reactions of the second group, the "flare up" group, the temperature was not reported.

Symptoms.—The various manifestations encountered in the 27 reactions of the first group follow in order of frequency:

Chills	15	Intense itching of the skin	1
Fever	14	Extremities flushed with slight cyanosis	1
Headache	13	Extreme emotional instability	1
Rapid pulse	13	Slight delirium	1
Nausea	10	Prostration	1
Vomiting	6	Eyelids and lips swollen	1
Weakness	4	Slight jaundice of the sclerae	1
Pallor	4	Conjunctivitis	1
Dizziness	3	Pupils dilated	1
Abdominal pain	3	Sore throat	1
Urticaria	2	Pharyngitis	1
General malaise	2	Backache	1
Nervousness	2	Pain in legs	1
Skin cold and clammy	2	Aches	1
Face flushed	2	Spitting of blood	1
Sweating	2	Lowered blood pressure	1
Macular rash	1	Cyanosis	1
Hyperemic rash	1	Shallow respirations	1
Fine scarlet rash	1	Subnormal temperature	1
Erythema of the face and chest	1	Cough	1
Slight erythema and swelling of the extremities	1	Gagged	1
Skin flushed	1	Dazed	1
Skin flushed, warm and dry	1		

The manifestations occurring in the 10-case "flare up" group in order of frequency were:

Headache	10	Vomiting	5
General malaise	10	Nausea	4
Chills	7		

Nonfatal cases—(Case histories).—(4-1932.) A patient who was exposed at Long Beach, Calif., was given a diagnosis of syphilis on August 4, 1932, as the result of darkfield examinations which were positive for *Treponema pallidum.* Treatment was instituted and the patient was administered 0.3 gram of neoarsphenamine intravenously on August 12, 1932. Four days later the patient received a second 0.3 gram intravenous injection of neoarsphenamine, the dilution of which was 0.6 gram of neoarsphenamine to 20 cubic centimeters of water and the rate of injection was 3 minutes for the 0.3 gram administered. As concurrent treatment he had received two intramuscular injections, 0.13 gram each, of bismuth salicylate. Six hours after the injection given on August 16 the patient developed a headache, chill, fever, nausea, vomiting, and a macular rash.

On August 26 he was given an intravenous injection of 1 gram of sodium thiosulphate and three days later was considered recovered after 13 days on the sick list from the time of first symptoms.

(5-1932.) A patient was exposed at Cavite, P.I. He was given a diagnosis of syphilis on July 19, 1932, as the result of darkfield examinations which were positive for *Treponema pallidum,* and a 4-plus Kahn blood test. It was the opinion of the reporting medical officer that this infection was a new infection or a second syphilitic infection after the original infection acquired in 1930. Prior to the new infection and immediately after the original infection the following arsenical treatment was administered:

First course, May 1, 1930, to June 23, 1930; and second course, August 1, 1930, to October 7, 1930, 23 injections of neoarsphenamine comprised these two courses (amounts not stated).

Third course, May 26, 1931, to September 26, 1931, 10 injections of neoarsphenamine—5.7 grams.

The patient also received the following concurrent or intercurrent treatment:

May 1, 1930, to June 23, 1930, 40 inunctions of mercury.

August 1, 1930, to August 21, 1930, 3 injections of bismuth salicylate, 2 grains each.

July 22, 1931, to September 16, 1931, 9 injections of bismosol, 1 cubic centimeter each.

February 2, 1932, to March 20, 1932, 7 injections of bismuth salicylate, 1 grain each.

After exposure and reinfection the patient was started on a course of arsenical treatment on August 2, 1932. (Considered his first course

of treatment after reinfection). This injection of neoarsphenamine had a dilution of 1:40, or 0.3 gram of neoarsphenamine dissolved in 12 cubic centimeters of sterile water. The rate of injection was 3 minutes for the 0.3-gram dose administered.

About 7 hours after this injection, the patient developed extreme emotional instability and was somewhat irrational. He had chills, fever, vertigo, and a temperature of 102.6° F. He was immediately transferred to a naval hospital for treatment. Upon admission to the hospital his temperature was 105° F., and he was thrashing about wildly. He was talking in a disconnected manner and was obsessed with the idea of putting on his clothes and leaving. Physical examination was essentially negative except for an indurated ulcer on the foreskin of the penis. Patient was administered 0.5 cubic centimeter of a 1:1000 solution of adrenalin intravenously. He was also given an intravenous injection of 1 gram of sodium thiosulphate and placed on a liquid diet with forced fluids.

The following day, August 3, the patient seemed much better; he was rather weak but entirely rational. Chart recordings were: Temperature 98.8° F., pulse 76, and respiration 18. Kahn blood test was reported as 4 plus.

The patient felt entirely well the next day and had no complaints. The neurological examination was negative. The ulcer on the penis was nearly healed.

On August 6, the patient was considered fully recovered and was returned to duty after 4 sick days.

(6-1932.) A patient, who was infected at Norfolk, Va., was given a diagnosis of syphilis on February 23, 1923, because of a primary lesion on the penis and a 4-plus Wassermann test. Arsenical treatment was instituted and administered as follows:

First course, February 15, 1923, to [?] 7 injections; second course, November 15, 1923, to January 27, 1924, 2 injections; January 31, 1924, to February 7, 1924, 2 injections, third course, January 14, 1930, to January 18, 1930, 1 injection; February 18, 1930, to March 4, 1930, 3 injections.

No concurrent or intercurrent treatment was reported. A Kahn blood test taken on July 28, 1932, was reported as negative.

On August 30, 1932, the patient was started on his fourth course of arsenical treatment with a 0.3-gram intravenous injection of neoarsphenamine. One week later, September 6, the patient was given his second injection, which consisted of 0.5 gram of neoarsphenamine injected intravenously. The dilution of this injection was stated as 1 gram of neoarsphenamine dissolved in 20 cubic centimeters of distilled water and the rate of injection was 2 minutes for the five-tenths dose administered. As concurrent treatment he was given an intramuscular injection of 1 cubic centimeter of bismosol on September 2.

At 11 a.m., 1 hour after the injection was given on September 6, the patient complained of pain in legs, nausea, vomiting, spitting of blood, chilly sensations, and a severe supraorbital headache. It was noted that there was some intradermal hemorrhage over the abdomen. The patient was placed in bed and, at 11:15 a.m., he was given 0.5 gram of sodium thiosulphate injected intravenously. Hot-water bottles were applied to the body and extremities, the chills quickly subsided, and the patient felt much better. At 1:30 p.m., the patient was given a second 0.5-gram intravenous injection of sodium thiosulphate after which he stated that he felt much better. Upon questioning, the patient stated that he had always had some reaction following the administration of neoarsphenamine but never one as severe as this particular reaction. The patient was transferred to a naval hospital for observation and treatment, at which time he complained of weakness and showed a slight rash on the arms and abdomen. He had been vomiting (greenish-yellow liquid) but there was no trace of blood in the vomitus. Pulse was of a good quality. The patient had voided no urine prior to his transfer to the hospital but a 6-hour specimen voided at the hospital was reported as negative for arsenic. He was given forced fluids and heat was applied to the body. On September 8 the patient was very much improved. The temperature, pulse, and respirations were normal. There was no nausea or vomiting and the rash had disappeared. This date the patient was allowed to be up and about and was discharged to duty the following day (September 9) after 3 sick days.[4]

(7-1932.) A patient was infected at Annapolis, Md., on September 3, 1932. He was given a diagnosis of syphilis as the result of positive darkfields which were taken on September 17.

On September 19 the patient received an intravenous injection of 0.3 gram of neoarsphenamine, and on September 22 an intravenous injection of 0.45 gram of neoarsphenamine. Four days later he received a third injection which consisted of 0.6 gram of neoarsphenamine injected intravenously. The dilution of this injection was 0.6 gram of neoarsphenamine dissolved in 20 cubic centimeters of sterile distilled water, the solution filtered through sterile gauze and injected intravenously with a sterile Luer syringe. The rate of injection was stated as 1 minute for the 0.6-gram dose administered.

At noon, 2 hours after this last injection, the patient became nauseated and vomited. He felt drowsy and was unable to eat any lunch. At 4:30 p.m., he developed a chill which continued for 10 minutes. At 7 p.m., the patient had a severe headache; temperature 103° F.; pulse 110; face was flushed; pupils dilated; an erythema on his face and chest; and he continued to be drowsy and nauseated.

[4] It was the opinion of the medical officer that the reaction was due to the patient's intolerance for arsenic rather than to faulty materials.

At 7:30 p.m., he was given an intravenous injection of 1 gram of sodium thiosulphate. At 9 p.m., the temperature had dropped to 101° F., and pulse to 90.

The next day, September 27, the patient complained of general malaise and nausea but no vomiting. The temperature was 100.6° F., the erythema on the face and chest had disappeared, but he still had a slight headache. He was administered an intravenous injection of 1 gram of sodium thiosulphate and placed on a liquid diet with complete rest in bed.

The patient was discharged to duty the following day after 2 sick days.

(8-1932.) A patient, who was infected at Cavite, P.I., was given a diagnosis of syphilis because of the appearance of a lesion on his scrotum on April 28, 1932, and as the result of a Kahn blood test which was reported as 4 plus on May 11, 1932. Arsenical treatment was immediately instituted and the patient was given his first injection on May 11, consisting of 0.3 gram of neoarsphenamine injected intravenously. One week later the patient was given a second injection which consisted of 0.6 gram of neoarsphenamine injected intravenously. The dilution of this injection was stated as 1:33, or 0.6 gram of neoarsphenamine dissolved in 20 cubic centimeters of sterile water. The rate of injection was 3 minutes for the 20 cubic centimeters of solution injected.

Immediately following this injection the patient had a reaction but failed to report to the medical officer until 48 hours later when he complained of headache and vomiting which increased in intensity. His temperature was 101.2° F.; Dickens' test was negative for arsenic; van den Bergh test was negative; urine was alkaline in reaction; white blood count was 23,000 which increased to 42,750; differential count was 89 percent polymorphonuclears and 11 percent lymphocytes. There was no redness of the skin and no skin eruption. The patient was administered hypodermic injections of adrenalin, minims X, every 4 hours; sodium bicarbonate, grains XXX, every 4 hours by mouth; and 2 grams of sodium thiosulphate (method of administration not stated). On May 21 the patient was transferred to a naval hospital for observation and treatment, at which time he stated that the nausea and vomiting had increased in intensity and that he had been unable to retain any food. Physical examination revealed his blood pressure as 130/60; the conjunctivae were injected; face red and perspiring freely; pulse full, bounding, and regular; some abdominal distention; no enlargement or tenderness of the liver or spleen and no eruption or itching of the skin. The patient appeared mentally normal. Laboratory reports of blood examination were red blood count, 4,200,000; white blood count, 42,750; hemoglobin, 75 percent; polymorphonuclears, 82 percent; lymphocytes, 17 percent; large mononuclears, 1

percent. The patient was given 1 gram of sodium thiosulphate intravenously, 1 cubic centimeter of adrenalin intravenously, and an intravenous injection of 500 cubic centimeters of saline solution and 8 percent of glucose. The next day, May 22, the blood picture was as follows: Red blood count, 5,150,000; white blood count, 14,550; hemoglobin, 85 percent; polymorphonuclears, 81 percent, lymphocytes, 16 percent; large mononuclears, 2 percent; eosinophiles, 1 percent. The Dickens' test was negative for arsenic. The patient was given an intravenous injection of 1 gram of sodium thiosulphate.

On May 23 the white blood count was 7,750; polymorphonuclears, 64 percent; lymphocytes, 32 percent; large mononuclears, 2 percent; eosinophiles, 2 percent. The Dickens' test was negative for arsenic. Report of urinalysis was: Appearance, clear; color, amber; reaction, alkaline; specific gravity, 1.023; albumin, negative; sugar, negative. The patient was administered 1 gram of sodium thiosulphate intravenously.

The following day the white blood count was 10,500; polymorphonuclears, 63 percent; lymphocytes, 27 percent; eosinophiles, 6 percent; large mononuclears, 3 percent; transitionals, 1 percent. The van den Bergh test was reported as negative. The patient was given 1 gram of sodium thiosulphate intravenously.

On May 25 the patient was administered another 1-gram intravenous injection of sodium thiosulphate and, on June 1, 1932, he was considered fully recovered and was discharged to duty, 14 days after appearance of first symptoms.

(9-1932.) A patient was infected at Manila, P.I. On February 10, 1926, he was given a diagnosis of syphilis as the result of a Kahn blood test which was reported as 2 plus. Arsenical treatment was instituted and administered as follows:

First course

Arsphenamine:	Gram	Neoarsphenamine—Continued	Gram
February 22, 1926	0.2	March 20, 1926	0.9
March 1, 1926	0.3	March 27, 1926	0.9
March 8, 1926	0.4	April 12, 1926	0.9
Neoarsphenamine:		April 22, 1926	0.9
March 13, 1926	0.9		

Mercury inunctions daily from March 13, 1926, to April 30, 1926.

Second course

Neoarsphenamine:	Gram	Neoarsphenamine—Continued	Gram
June 5, 1926	0.3	July 3, 1926	0.9
June 12, 1926	0.45	July 10, 1926	0.9
June 19, 1926	0.6	July 17, 1926	0.9
June 26, 1926	0.75	July 24, 1926	0.9

Mercury inunctions 4 times each week from June 5, 1926, to July 30, 1926.

Third course

Neoarsphenamine:	*Gram*	Neoarsphenamine—Continued	*Gram*
August 28, 1926	0.3	November 2, 1926	0.9
October 12, 1926	0.45	November 10, 1926	0.9
October 19, 1926	0.6	November 16, 1926	0.9
October 26, 1926	0.75	December 7, 1926	0.9

Eight mercury salicylate injections, 1 grain each, between October 15, 1926, and December 17, 1926.

The fourth course of treatment was instituted and the patient was given 0.45 gram of neoarsphenamine intravenously on March 1, 1932. On March 8, 15, 29, and April 5, he received 0.6-gram injections of neoarsphenamine intravenously. The total amount administered during this fourth course was 2.85 grams of neoarsphenamine administered over a period of 36 days, or an average of 79 milligrams per day. No concurrent treatment was reported as having been administered.

The dilution of the injection given on April 5 was reported as 0.9 gram of neoarsphenamine to 22.5 cubic centimeters of water with a 0.6-gram dose administered. The rate of injection was 2 minutes for the 0.6-gram dose administered.

About 20 hours after this injection the patient reported to the sick bay with severe cramping pains throughout the abdomen which were accompanied by nausea and vomiting. Examination revealed temperature of 100° F., pulse 90 and of good quality, and the abdomen soft and flaccid. Examination otherwise negative. The patient stated that, after injections of previous courses, he had slight intestinal upsets but that he had experienced no ill effects following treatment on this course until the present time. He was given 0.5 cubic centimeter of adrenalin intramuscularly and 1 gram of sodium thiosulphate intraveneously. After the administration of the sodium thiosulphate he seemed somewhat relieved of the nausea, but he had passed two watery stools since his admission. The patient was then transferred to a naval hospital for further treatment. Examination in the hospital revealed the blood pressure as 120/85, bilateral epitrochlear adenopathy, absent superficial and diminished deep reflexes, some slight tenderness over the liver but no alteration in size, skin and mucous membranes normal, no edema, and no abdominal tenderness or pain. The Dickens' test was reported as weakly positive for arsenic. Complete blood count was reported as white blood count, 13,300; polymorphonuclears, 90 percent; lymphocytes, 6 percent; large mononuclears, 2 percent; transitionals, 1 percent; and basophiles, 1 percent. Treatment administered was rest in bed, light diet, liquids, and sodium thiosulphate intravenously, 1 gram daily for 4 doses. The following day, April 7, urinalysis was reported as follows: Appearance, clear, amber; reaction, acid; specific gravity, 1.026; albumin, negative; sugar, negative.

The patient was considered recovered on April 23, after 17 days on the sick list.

(10-1932.) A patient was given a diagnosis of syphilis as the result of a Wassermann blood test which was reported 4 plus on February 19, 1924. The time and place of infection was not stated.

The first course of arsenical treatment (type of treatment not stated) consisted of 6 injections which were administered between the dates of February 4 and May 14, 1924. The second course of arsenical treatment was started December 23, 1931, and consisted of 6 injections. (Type of treatment and date of completion of this course not stated.)

The patient began his third course of arsenical treatment on March 24, 1932, and received 9 intravenous injections of neoarsphenamine between that date and July 9, 1932. The dose of the intravenous injection given on July 9, 1932, was 0.6 gram of neoarsphenamine but the dilution and the rate of injection were not stated. As concurrent treatment with the third course of arsenical treatment the patient was given 10 injections of bismosol.

Approximately one half hour after the patient had received the injection of neoarsphenamine on July 9 he complained of chills which were followed by extreme weakness, nausea, headache, fever of 102° F., and pulse of 108. He was transferred to a naval hospital and it was noted that his skin was warm and dry but no rash was present. Examination of urine revealed a slight trace of arsenic. Sodium thiosulphate, 1 gram dose, was given intravenously when admitted to hospital and 2 days later this treatment was repeated.

The patient's condition gradually improved and on August 2, 23 days after the first symptoms of reaction, he was considered fully recovered.

(11-1932.) A patient was infected at Los Angeles, Calif. On March 7, 1932, he was given a diagnosis of syphilis as the result of a primary lesion on the penis and repeated Kahn blood tests, which were 4 plus. Arsenical treatment was administered as follows:

First course, March 9, 1932, to May 4, 1932, 8 injections, neoarsphenamine (total 4.65 grams).

Second course, June 15, 1932, to August 10, 1932, 8 injections, neoarsphenamine (total 4.65 grams).

As concurrent treatment the patient was administered the following:

March 9, 1932, to May 4, 1932, bismuth salicylate, intramuscular, 0.1 gram each.

June 15, 1932, to August 10, 1932, bismuth salicylate, intramuscular, 0.1 gram each.

On September 9 the patient was started on his third course of arsenical treatment and was given an intravenous injection of 0.45 gram of neoarsphenamine on this date. The dilution of the injection

was 0.9 gram of neoarsphenamine dissolved in 15 cubic centimeters of sterile water, and the rate of injection was 90 seconds for the administration of 7.5 cubic centimeters of the solution.

One hour after the administration of this injection the patient complained of headache, nausea, severe chills, and pallor. The Dickens' test showed normal elimination of arsenic. The patient was given an intravenous injection of 1 gram of sodium thiosulphate and 2 hours later he was considered recovered and was discharged from the sick bay.

(12-1932.) A patient was infected at Los Angeles, Calif. On May 10, 1932, he was given a diagnosis of syphilis as the result of the appearance of a small, hard ulcer on the mucous surface of the prepuce. Repeated Kahn blood tests, which were 4 plus, verified the diagnosis. The first course of arsenical treatment was administered between the dates of May 10 and July 6. This course consisted of 8 intravenous injections of neoarsphenamine for a total dosage of 4.65 grams. As concurrent treatment he received 8 intramuscular injections of bismuth salicylate for a total dosage of 0.86 gram.

On September 14 the patient was started on his second course of arsenical treatment and was given 0.45 gram of neoarsphenamine that date. One week later, September 21, he was given 0.6 gram of neoarsphenamine intravenously as his second injection, making a total of 1.05 grams of neoarsphenamine administered to date on the second course. The dilution of this injection was stated as 0.9 gram of neoarsphenamine dissolved in 15 cubic centimeters of sterile water and the rate of injection was stated as 90 seconds for the administration of 10 cubic centimeters of the solution.

One hour later the patient complained of headache, nausea, severe chill, and pallor. The Dickens test showed normal elimination of arsenic. He was given 1 gram of sodium thiosulphate intravenously and 2 hours later he was considered recovered and was discharged from the sick bay.

(13-1932.) A patient was infected at Seattle, Wash. On September 12, 1928, he was given a diagnosis of syphilis as the result of the appearance of a typical hard chancre on the prepuce. Darkfield examinations of the serum from the lesion were positive for *Treponema pallidum*. Arsenical treatment was instituted and administered as follows: First course, September 12, 1928, to November 8, 1928; second course, November 28, 1928, to January 31, 1929; third course, February 14, 1929, to April 11, 1929; fourth course, December 5, 1929, to February 28, 1930; fifth course January 9, 1932, to March 12, 1932; sixth course, April 3, 1932, to May 6, 1932. In addition, 4 injections were administered, the dates of which were not given. The amounts of the particular courses as listed above were not stated, but the information was recorded that the patient had received 3 courses of

7.2 grams each, 1 course of 4.8 grams, 2 courses (amounts not stated), and 4 injections (amounts not stated). The report did not state whether the patient had received concurrent or intercurrent treatment.

On August 31, 1932, the patient was started on his seventh course of arsenical treatment and was given an intravenous injection of 0.45 gram of neoarsphenamine on that date. On September 7, 14, and 21 he received his second, third, and fourth injection of neoarsphenamine, each injection 0.6 gram. The total for the seventh course was 2.25 grams of neoarsphenamine. The dilution of the fourth injection, that given on September 21, was 0.9 gram of neoarsphenamine dissolved in 15 cubic centimeters of sterile water, and the rate of injection was 90 seconds for 10 cubic centimeters of the solution.

One hour after this fourth injection the patient complained of headache, nausea, severe chill, and pallor. The Dickens' test showed normal elimination of arsenic. The patient was given an intravenous injection of 1 gram of sodium thiosulphate, and 2 hours later he was discharged from the sick bay completely recovered.

(14-1932.) A patient, whose health record states that there is no evidence of a venereal ulcer, was given a diagnosis of syphilis as the result of Kahn blood tests taken on May 18 and 21, 1931, and reported as 4 plus. The spinal fluid test was reported as slight globulin, 16 cells. Arsenical treatment was instituted and administered as follows:

First course---May 28, 1931, to August 11, 1931, ? injections, neoarsphenamine,
Second course---January 6, 1932, to March 1, 1932, 8 injections, neoarsphenamine, total 4.8 grams.

On July 14, 1932, the patient was started on his third course of arsenical treatment and 0.3 gram of neoarsphenamine was administered. The course was continued on August 11 when he received 0.45 gram of neoarsphenamine. On August 18, 25, September 1, and 8 he received his third, fourth, fifth, and sixth injections, 0.6 gram each, of neoarsphenamine. The total dosage for this third course was 3.15 grams. As concurrent treatment the patient received nine 2-grain intramuscular injections of bismuth salicylate between the dates of July 15 and September 7, 1932. It was reported that the dilution of the sixth injection of neoarsphenamine, that given on September 8, was 0.9 gram of neoarsphenamine dissolved in 18 cubic centimeters of sterile distilled water, and the rate of injection was stated as 3 minutes for the administration of 12 cubic centimeters of the solution.

About 10 hours after the administration of the sixth and last injection of neoarsphenamine the patient complained of intense occipital headache, vomiting, weakness, and chills. Examination revealed a slight jaundice of sclera, temperature 97.6° F., and pulse 92. The patient was then given an intramuscular injection of 10 cubic centimeters of a 10 percent solution of sodium thiosulphate, and later during the day he was administered 4 doses of 5 grains each of aspirin

and phenacetin and 500 cubic centimeters of a 1.5 percent saline solution by proctoclysis.

On September 10, two days after appearance of initial symptoms, the patient was considered fully recovered and was returned to duty.

(15-1932.) A patient who was exposed at Norfolk, Va., about June 23, 1932, was given a diagnosis of syphilis on August 17 as the result of the appearance of genital lesions, general adenopathy, congested pharynx, and a 4 plus Kahn blood test.

Arsenical treatment was instituted on August 9 and the patient given a 0.3 gram intravenous injection of neoarsphenamine. On August 16 he was given a second 0.3 gram intravenous injection and on August 23 a third intravenous injection which consisted of 0.6 gram of neoarsphenamine. The dilution of this third injection was 0.6 gram of neoarsphenamine dissolved in 20 cubic centimeters of double distilled sterile water and the rate of injection was 3 minutes. As concurrent treatment the patient was administered 2 cubic centimeters of bismosol, injected intramuscularly, on August 16 and 1 cubic centimeter intramuscular injection on August 19.

About 1½ hours after the last injection of neoarsphenamine, that given on August 23, the patient complained of severe headache and general malaise. About 7 hours later the patient's temperature rose and he complained of severe abdominal pain and nausea. He did not report his condition until about 9 p.m., about 10½ hours after the injection. At this time he was given 1 gram of sodium thiosulphate intravenously. The condition improved somewhat, but resembled appendicitis, with pain, nausea, temperature 102.6° F., rapid pulse, localized abdominal tenderness, and white blood count of 13,600. At about 10:15 p.m., about 12 hours after the injection, the patient was transferred to a hospital ship. Examination on admission to the hospital ship revealed the abdominal pain and other symptoms to be subsiding but the temperature had risen to 103° F. The next morning the temperature reduced to 99° F. and gradually returned to normal. The patient's symptoms subsided and he remained symptomless and was considered recovered after 1 day on the sick list.[5]

(16-1932.) A patient was infected at Honolulu, T.H. On June 15, 1928, he was given a diagnosis of syphilis as the result of a typical chancre on the penis and a 4 plus blood test.

The patient received a course of arsenical treatment which was completed in July 1928; a second course which was completed in June, 1929; and a third course which was completed in March 1932. A Kahn blood test taken on May 2 was reported as 4 plus.

On July 19 the patient was started on his fourth course of arsenical treatment and was administered an intravenous injection of 0.45

[5] The medical officer reported that 20 other patients had received injections the same day and no reactions were manifested; therefore, it was his opinion that the patient was sensitive to arsenic and that the dosage was not excessive in ordinary treatment, nor the technique faulty.

gram of neoarsphenamine on that date. The dilution of this injection was 0.9 gram of neoarsphenamine dissolved in 10 cubic centimeters of double distilled water and the rate of injection was 3 minutes for the 0.45 gram dose administered. No intercurrent or concurrent treatment was administered.

Two hours after this injection of neoarsphenamine the patient was admitted to the sick list with large circular urticarial areas on the flexor surface of the forearms and abdomen. General physical examination was essentially negative except for the urticarial areas. The patient was given an intravenous injection of 1 gram of sodium thiosulphate about 8 hours after he had received the intravenous injection of neoarsphenamine.

On July 22 the urticaria was still present but subsiding. The patient was transferred this date to a hospital ship for further antiluetic treatment.

During hospitalization on the hospital ship the patient's urine was negative for casts and albumin; blood chemistry was normal; phenolsulphonphthalein elimination normal; and Kahn blood test negative. The patient had received two injections, 0.13 gram each, of bismuth salicylate; one intravenous injection, 0.3 gram, of neoarsphenamine; and one intravenous injection, 0.5 gram, of neorsphenamine. The arsenic elimination tests following each of the injections showed normal rate of elimination. At the time of his admission to the hospital ship and during the period on the sick list he showed no signs or symptoms of arsenical poisoning.

On August 5, after 17 sick days, the patient was discharged to duty and it was recommended that the course of arsenical treatment be completed in the usual manner.

(17-1932.) A patient, in whom no definite history of infection was obtainable, was transferred to a naval hospital with "Diagnosis Undetermined (syphilis)" because of repeated positive Kahn blood tests. He was later returned to duty after a diagnosis of "No Disease" had been made. Upon discharge to duty it was recommended that antiluetic treatment be given as Kahn blood tests were reported as 4 plus. Because of the recommendations made by the hospital the patient was given six intravenous injections of neoarsphenamine between the dates of November 3 and December 8, 1931. The total dosage of this first course of treatment was 3.45 grams of neoarsphenamine. As intercurrent treatment the patient was given 9 injections, 1 cubic centimeter each, of bismosol between the dates of December 24, 1931, and February 24, 1932, when the course was discontinued because of bismuth stomatitis.

On March 24, 1932, a second course of arsenical treatment was started and the patient was given a 0.45 gram intravenous injection of neoarsphenamine. An injection was not given one week later as

the patient had developed scabies and it was feared that this condition might conceal a skin reaction. On April 7 the patient was given a 0.2 gram intravenous injection of neoarsphenamine and suffered no reaction except some dizziness. No further arsenical treatment was given until May 26, 1932, because the patient had a persistent cold and tonsillitis. From April 21 to May 5, 1932, the patient was given daily mercurial inunctions and ascending doses of potassium iodide. On May 26 he was given a 0.2 gram intravenous injection of neoarsphenamine without a reaction other than a slight cough and a slight sensation of dizziness. On June 2 the patient was prepared by the administration of a 1/50 gr. injection of atropine sulphate. Following this injection he was given a 0.2 gram intravenous injection of neoarsphenamine, the dilution of the injection being 0.06 gram of neoarsphenamine per cubic centimeter and the rate of injection 2 minutes for for the 0.2 gram dose administered. Three minutes after the injection the patient complained of dizziness, flushing of face, and a cough. Five minims of adrenalin were administered hypodermically and in half an hour all symptoms had cleared up. Dickens' test showed normal elimination of arsenic.

(18-1932.) A patient was given a diagnosis of syphilis on May 8, 1932, because of the appearance of two small ulcerations on the prepuce, mucous patches, generalized lymphadenopathy. A Kahn blood test was reported 4 plus on May 16.

Arsenical treatment was instituted and the patient was given a 0.3 gram intravenous injection of neoarsphenamine on May 17, 1932. One week later he was given a 0.45 gram intravenous injection of neoarsphenamine, the dilution of which was 0.45 gram of neoarsphenamine dissolved in 10 cubic centimeters of distilled water. The rate of injection was 2 minutes.

Three days later, May 27, the patient developed a diffuse hyperemic rash mainly on the trunk, sore throat, conjunctivitis, temperature 103° F., and pulse 110. There was also present a generalized glandular adenopathy and an acute pharyngitis. The patient was treated with sodium thiosulphate, 0.1 gram, every 24 hours for a period of time not stated. The Dickens test was reported negative on May 20, 3 plus on May 24; 1 plus on June 3; and 1 plus and 2 plus on June 7. Routine and microscopical urinalysis was essentially negative on May 14, 25, and June 3.

The rash began to fade and by June 1, five days after onset of reaction, all signs and symptoms had disppeared.

(19-1932.) A patient who was infected at Manila, P.I., on December 29, 1928, was given a diagnosis of syphilis as the result of positive darkfield examinations for *Treponema pallidum* and a four plus blood test. Arsenical treatment was administered as follows:

First course, May 1929 to August 1929, 8 injections neoarsphenamine and 11 injections bismuth.

Second course, August 1929 to October 1929, 8 injections neoarsphenamine and 7 injections bismuth.

Third course, November 1929 to February 1930, 8 injections neoarsphenamine and 8 injections bismuth.

Fourth course, January 1931 to March 1931, 6 injections neoarsphenamine and mercury inunctions.

———, May 1931 to June 1931, 5 injections bismuth.

Fifth course, August 1931 to October 1931, 7 injections neoarsphenamine and mercury inunctions.

———, December 1931 to March 1932, 12 injections bismuth.

The sixth course of arsenical treatment was instituted and the patient was given a 0.45 gram intravenous injection of neoarsphenamine on May 6, 1932. One week later the patient was given his second injection, 0.6 gram of neoarsphenamine injected intravenously. The dilution of the injection was neoarsphenamine 1:3 and the rate of injection was 4 minutes for the 0.6 gram dose.

Two minutes after this second injection the patient felt weak; gagged a few times; skin was cold and clammy; the extremities, face, and neck were flushed with a slight cyanosis; the pulse was feeble, rapid, and irregular; blood pressure 164/80, dropped beat every third systole. The urinalysis and Dickens' tests were reported as negative. The patient was immediately given 1 cubic centimeter of a 1:1000 solution of adrenalin hydrochloride hypodermically and placed in bed with an ice cap to his head. Four hours later he was considered fully recovered.

(20-1932.) A patient was infected November 6, 1932, at Philadelphia, Pa. The diagnosis of syphilis was established by a primary lesion on his penis, rash on his body, and a 4 plus Kahn blood test.

Arsenical treatment was instituted and he was given a 0.3 gram intravenous injection of neoarsphenamine on November 23. He was given a 0.45 gram intravenous injection on December 1 and a 0.6 gram intravenous injection on December 8. As concurrent treatment he was given intramuscular injections, 2 grains each, of bismuth salicylate on November 28, December 2, and December 5, 1932. The dilution of the last injection of neoarsphenamine, given on December 8, was 0.9 gram of neoarsphenamine dissolved in 18 cubic centimeters of sterile distilled water, the rate of injection being about 2 minutes for the 0.6 gram (12 cubic centimeters) dose. Thirty minutes after the administration of the last injection of neoarsphenamine, that given on December 8, the patient complained of nervousness, weakness, and fever. He was placed in bed and was given an intravenous injection of 10 cubic centimeters of a 10 percent solution of sodium thiosulphate. Alkalies and fluids were given orally.

On December 10, 2 days after the onset of reaction, the patient was considered fully recovered.

(21-1932.) A patient who was infected at Norfolk, Va., on September 18, 1917, was given a diagnosis of syphilis because of the presence

of mucous patches, general glandular enlargement, macular rash, and a 4 plus Wassermann blood test. The patient was given his first course of arsenical treatment in 1917–18 and a second and third course in 1918. According to the report, no further antiluetic treatment was given until the patient began his fourth course of treatment in March 1932. On March 22, 29, April 5, and April 12, 1932, the patient received 0.45 gram intravenous injections of neoarsphenamine as the first, second, third, and fourth injections of the fourth course of treatment. The dilution of the injection given on April 12 was 0.45 gram of neoarsphenamine to 10 cubic centimeters of water and the rate of injection was 45 seconds for the 0.45 gram dose.

Two hours after this injection the patient complained of frontal headache, chills, and a sense of pressure in his head which was accompanied by dizziness and pallor. Examination disclosed signs of recent mild circulatory collapse with pallor, tachycardia, sweating, rapid shallow respiration, and lowered blood pressure of 118/84. The heart sounds were poor with a slight blowing systolic murmur at the apex. There was no apparent cardiac enlargement. The Dickens' test showed poor elimination of arsenic. The patient was placed in bed with the foot of the bed elevated and an ice cap applied to his head. He was then administered 7 drops of adrenalin hypodermically and a liquid diet ordered.

On April 13, or approximately 24 hours after onset, the patient was considered fully recovered.

(22-1932.) A patient who was exposed at Norfolk, Va., was given a diagnosis of syphilis on January 28, 1932, because of the appearance of a primary lesion on his penis. This diagnosis was confirmed on February 24 as the result of darkfield examinations of the serum from the lesion which were positive for *Treponema pallidum*. The patient was given a 0.3 gram intravenous injection of neoarsphenamine on February 18 and 0.45 gram injections on February 21, 25, March 3, 10, 17, 24, 31, and April 7, making a total of 3.9 grams for the nine injections. The dilution of the ninth injection, given on April 7, was 0.45 gram of neoarsphenamine in 10 cubic centimeters of sterile distilled water, the rate of injection being 5 minutes for the 0.45 gram dose. As concurrent treatment the patient was administered mercury inunctions.

One hour after the last injection of neoarsphenamine the patient complained of headache, chills, and backache. Examination revealed a rapid pulse, weak and slight response of reflexes. The Dickens' test showed elimination of arsenic. He was given 1 ounce of magnesium sulphate by mouth, 1 gram of sodium thiosulphate intravenously, and 15 grains of aspirin by mouth.

Three and one half hours later the patient was considered recovered.

(23-1932.) A patient who stated he had sexual intercourse on October 15, 1932, at San Francisco, Calif., appeared at sick call on November 17 with a small ulcer on his penis and a slight painless adenopathy of the inguinal glands. Darkfield examination of the serum from the lesion was reported positive for *Treponema pallidum*.

Arsenical treatment was instituted. He was given a 0.3 gram intravenous injection of neoarsphenamine on November 17 and 0.45 gram intravenous injections on November 24 and on December 1. The dilution of the last injection was 0.45 gram of neoarsphenamine in 20 cubic centimeters of freshly triple distilled water, the rate of injection being approximately 5 minutes for the 20 cubic centimeters of solution administered. As concurrent treatment the patient was given ⅛ grain intramuscular injections of mercury succinimide on November 18 and 25.

Approximately 4 hours after the last injection, given on December 1, the patient experienced a severe chill and reported to the sick bay. Examination revealed a fine scarlet rash over the entire body. His temperature was 103° F., pulse 90, and respiration 28. He was placed in bed immediately with hot water bottles and blankets applied for the chills which lasted for 30 minutes. His temperature rose quickly to 105° F., pulse 140, and respiration 30. Marked prostration was present and the skin was warm and dry. The patient showed no signs of cyanosis and complained of no pain at any time. A leukocyte count was reported as 13,000. He was given an intravenous injection of sodium thiosulphate, 1 gram in 10 cubic centimeters of triple distilled water, about 15 minutes after onset of reaction. About 6 hours later a similar injection was administered but of a 0.5 gram dose. Nine hours after inception of the reaction the Dickens' test was reported as faintly positive, but after this time arsenic could not be detected in any test. The symptoms gradually increased in intensity for about 6 hours after which they gradually subsided except the rash and a temperature which ranged from 99° F. to 101° F. The temperature persisted until December 3 when it returned to normal. At this time the rash had largely disappeared. On this date the patient was given a 0.5 gram intravenous injection of sodium thiosulphate which had been dissolved in 10 cubic centimeters of triple distilled water. He showed no ill effects from this date and the rash completely disappeared, thus it was considered that the reaction completely subsided in 3 days after the onset.

(24-1932.) A patient was exposed at Los Angeles, Calif., on January 30, 1932. He reported to the sick bay on March 18, stating that a sore had appeared on his penis the previous day. Examination revealed a typical Hunterian chancre on the mucous surface of the prepuce, dorsally at the corona. A moderate inguinal adenopathy was present. On March 21 a Kahn blood test was reported as 3 plus.

The patient was given an intramuscular injection of ⅛ grain of mercury succinimide on March 18 and an intravenous injection, 0.45-gram dose, of neoarsphenamine on March 21. The dilution of this injection was stated as 0.45 gram of neoarsphenamine dissolved in 20 cubic centimeters of freshly triple-distilled and freshly sterilized water. The rate of injection was 8 minutes for the 20 cubic centimeters of solution administered.

Eight hours after the injection of neoarsphenamine, or 5 p.m., the patient took a drink of water and was suddenly seized with severe chills which lasted about 30 minutes. During and following this period his pulse was rapid and varied from 120 to 140; the respirations were only slightly accelerated; the skin was flushed, warm, and dry; he showed no cyanosis and complained of no pains at any time; and the temperature made a rapid rise and reached a maximum of 104° F. at 8:30 p.m. The patient was placed in bed, covered with blankets, and surrounded with hot-water bottles which were removed upon completion of the chilly stage. Adrenalin hydrochloride, 5 minims of a 1:1,000 solution, was administered hypodermically. The Dickens' test was positive, and 11 hours after the inception of the reaction it was reported as faintly positive. After this time arsenic could not be detected in the urine. The white blood count was reported as 13,000, and the differential blood count as polymorphonuclears 85 percent, lymphocytes 11 percent, and mononuclears 4 percent.

At 1 a.m., or 8 hours after onset, temperature and pulse were normal and the patient showed no ill effects at this time.

(25-1932.) A patient was infected at Shanghai, China. On May 16, 1929, he was given a diagnosis of syphilis because of the appearance of a generalized maculo-papular rash of a brownish color, generalized adenopathy, slight fever, and a 4-plus Kahn blood test. The patient was administered a course of arsenical treatment in 1929, one in 1930, and a third course in 1931. From August 19 to December 30, 1931, he was given 20 intramuscular injections of bismosol, total dosage 2 grams.

On January 6, 1932, the patient was started on his fourth course of arsenical treatment and was administered an intravenous injection of 0.3 gram of neoarsphenamine on that date. On January 13, 20, and 27 he was given 0.45 gram intravenous injections of neoarsphenamine. Following the injections on January 27 the patient had a slight reaction and further treatment was omitted until February 10, when he received his fifth intravenous injection which consisted of 0.225 gram of neoarsphenamine. The total dosage of this course to date was 1.875 grams. The dilution of the injection given on February 10 was 0.9 gram of neoarsphenamine dissolved in 20 cubic centimeters of freshly distilled water sterilized in an autoclave, and the rate of

injection was 4 minutes for the 5 cubic centimeters of solution (0.225-gram dose) administered.

About 5 minutes after this injection the patient had a moderate vasomotor reaction of the vagotonia type accompanied by marked prostration and complete disability. The patient was placed in bed with external warmth applied. No drugs were administered.

The Dickens' test prior to January 27 had shown a prompt (within 1 to 2 hours) reaction of normal intensity. The response on January 27 showed only a moderate trace in 1½ hours and good elimination in 3½ hours. On February 10 the Dickens' test showed only the barest trace in 1½ hours and a normal response in 3½ hours. Urinalysis was normal at all times.

Three hours after onset of reaction the patient recovered and was returned to duty.

(26-1932.) A patient who was exposed at Shanghai, China, was given a diagnosis of syphilis on May 3, 1932, as the result of a dark-field examination of the serum from the lesion being positive for *Treponema pallidum*. Arsenical treatment was instituted and the patient was given a 0.45-gram intravenous injection, and on May 18, 27, June 3, 10, and 17, he was given 0.5-gram intravenous injections of neoarsphenamine. This course was discontinued due to cholangitis, acute (no history of case). The total amount of this first course was 3.25 grams. As concurrent and intercurrent treatment the patient was given 12 intramuscular injections, 2 grains each, of bismuth salicylate between the dates of June 2 and August 25.

On August 27 the patient was admitted to the sick list with a second attack of cholangitis, acute. The chief complaints were vomiting and loss of appetite. Examination revealed considerable jaundice pigmentation of the eyes and skin. He was given routine treatment and a fat-free diet. On September 5 he was discharged to duty under treatment after 9 sick days.

A second course of neoarsphenamine treatment was started on September 16, and he was given a 0.3-gram intravenous injection on that date. On September 23 he received a 0.45-gram intravenous injection. Treatment was temporarily discontinued due to transfer to another ship for duty. He then received a 0.5-gram intravenous injection on October 4 and a 0.3-gram intravenous injection on October 12. The total for this second course was 1.55 grams which was given over a period of 27 days, or an average of 57 milligrams per day. The dilution of the last injection was 18 cubic centimeters of sterile distilled water to one 0.9-gram ampule of neoarsphenamine, the patient receiving 6 cubic centimeters of this solution. The rate of injection was about 1 minute for the 6 cubic centimeters (0.3-gram dose) of solution administered.

At 9:45 a.m., about 30 minutes after the patient had received the injection of neoarsphenamine, he was brought to the sick bay from the head where he had collapsed. An examination revealed cold and clammy skin, subnormal temperature, and feeble and rapid pulse. He was immediately given a hypodermic injection, VII minim dose, of adrenalin hydrochloride. Blankets and hot water bottles were applied for external heat. At 10:30 a.m. he was given a hypodermic injection of caffeine and sodium benzoate. The patient then became very restless and at 1:30 p.m. he was given a hypodermic injection of morphine sulphate (one eighth grain) and atropine sulphate (one-two hundredth grain). The patient rapidly improved, but he was retained in the sick bay for observation. The next day, October, 13, laboratory findings were reported as follows:

First blood count	
R.b.c	5,920,000
W.b.c	16,400
Polys—percent	84
S. lymphs—do	10
L. lymphs—do	2
L. monos—do	2
Trans—do	2

Later blood count	
R.b.c	4,520,000
W.b.c	8,600
Polys—percent	72
S. lymphs—do	22
L. lymphs—do	2
Monos—do	2
Trans—do	1
Eosins—do	1

Urinalysis	
Specific gravity	1.025
Color	Amber
Reaction	Acid
Sugar	Faint trace
Albumin	Negative
Bile	Negative
Microscopic	Numerous squamous epithelium

According to the report the patient was transferred to a naval hospital but no date or reason for transfer was stated. The naval hospital recorded no manifestations of reaction at time of admission or subsequent to admission. No treatment was administered while the patient was in the hospital and it was the opinion of the hospital authorities that he had completely recovered from the reaction prior to admission.

From the reports it was considered that the patient recovered from the reaction about 13 hours after the first symptoms were noted.[1]

(27-1932.) A patient was exposed at San Diego, Calif., on June 15, 1931. On October 6 a primary lesion was noted on his penis and a darkfield examination of the serum from the lesion was reported positive for *Treponema pallidum*. Arsenical treatment was instituted and he received his first course of arsenical treatment between the dates of October 10, 1931, and January 9, 1932; his second course was administered between the dates of February 6 and April 9; and the third course between May 14 and August 26.

[1] The reporting medical officer stated that 6 other men had received neoarsphenamine injections of the same drug without reaction.

On October 5 the patient began his fourth course of arsenical treatment and received a 0.3 gram intravenous injection of neoarsphenamine on that date. Treatment was discontinued (reason not stated) until November 9, when he was given a 0.3 gram intravenous injection of neoarsphenamine as his second injection of the fourth course. The dilution of this injection was 1–20 and the rate of injection 70 seconds for the 0.3 gram dose administered. As concurrent treatment the patient was given 0.13 gram intramuscular injections of bismuth on October 11, 18, 25, and November 1, 1932.

The patient received his last injection of neoarsphenamine at 9:50 a.m. on November 9. Two hours later he developed a slight headache, general malaise, aches, and chilly sensations, followed by a fever which rose rapidly to 103.6° F. at 2:45 p.m. At this time the pulse was recorded as 120, respirations 22, and the patient was sweating profusely. Laboratory findings were reported as follows: white blood count 8,900; segmented 59 percent; lymphocytes 32 percent; basophiles 1 percent; eosinophiles 8 percent. The Dickens tests were reported negative for arsenic at 1:30 p.m. and 6 p.m. The patient was given 0.025 gram of ephedrin at noon and 2 p.m. He was also given an intravenous injection of 0.5 gram of sodium thiosulphate in 10 cubic centimeters of water and 480 cubic centimeters of Fischer's solution per rectum.

At 6 p.m., 8 hours and 10 minutes after the injection, the patient's temperature returned to normal and he went to sleep. It was then considered that he had fully recovered from the reaction.

(28-1932.) A Veterans' Administration patient acquired syphilis in 1916 while in the Hawaiian Islands. He was given the diagnosis of syphilis because of a lesion on his penis and by reason of positive darkfield and blood tests. He received a course of arsenical treatment in 1919 and another course in 1920.

He was started on another course (possible third course) of arsenical treatment on April 19, 1932, at which time he was given a 0.3 gram intravenous injection of neoarsphenamine. One week later, April 26, he was given a 0.6 gram intravenous injection and on May 3, a 0.45 gram intravenous injection of neoarsphenamine. The dilution of this third injection was stated as 0.9 gram of neoarsphenamine dissolved in 30 cubic centimeters of water and the rate of injection was about 1½ minutes for the 0.45 gram dose administered. As concurrent treatment the patient received 11 intramuscular injections of bismosol between the dates of April 20 and May 28.

In 1½ hours after the third injection of neoarsphenamine the patient appeared to be nervous, looked flushed, and seemed dazed. Upon questioning he complained of chilly sensations. He was placed in bed and hot-water bottles were placed about him. He was immediately given an intravenous injection of 1 gram of sodium thiosul-

phate dissolved in 10 cubic centimeters of distilled water. The patient reacted favorably for a short time but about 45 minutes after the injection his lips and hands began to show some cyanosis, the pulse became rapid, and respirations were increased. He then complained of crampy pains in the abdomen and dull aching pains in both loins. Adrenalin, X minims of a 1 : 1000 solution, was administered hypodermically and in about 15 minutes the cyanosis began to subside, respirations became more regular, and the patient expressed great relief. Soon thereafter the temperature began to rise, reaching 102° F., but no further chills or pains were complained of. In about 12 hours after the onset of reaction the symptoms had entirely subsided.

The next day the Dickens test was reported as positive, and urinalysis was as follows: Amount, 225 cubic centimeters; color, light clear amber; reaction, acid; specific gravity, 1.015; albumin, negative; sugar, negative; casts and cylindroids, 0; mucus, little; leukocytes, 1–2 per field (HD); erythrocytes, none noted; epithelium, occasional; crystals, none.[6]

(29-1932.) A patient (female-supernumerary) who was infected about March 1932 was given a diagnosis of syphilis because of a large persisting gland in the right side of the neck, which apparently followed tonsillitis; the appearance of maculo-papular secondaries on the arms and legs; and repeated Kahn blood tests which were reported as 4 plus. Prior to this time the patient had been given potassium-iodide treatment for about 2 weeks by a civilian doctor. Arsenical treatment was instituted and a 0.3 gram intravenous injection of neoarsphenamine was administered on June 4,1932. On June 7 and 10, 0.5 gram injections were administered, and on June 14 a 0.6 gram injection was administered. The dilution of this injection was stated as 65 milligrams to each 2 cubic centimeters of distilled water, and the rate of injection was 2 minutes for the 0.6 gram dose administered. The total dosage was 1.9 grams administered over a period of 11 days, or an average of 173 milligrams per day.

One hour after the injection given on June 14 the patient had a fever of 105° F., nausea, slight delirium, and later slight erythema and swelling of the extremities. About 4 hours after the reaction was manifested 10 cubic centimeters of a 10 percent solution of sodium thiosulphate was administered intravenously.

The following day the patient felt well except for itching of the extremities. This persisted for several days.

On June 24, 10 days after onset of reaction, the patient was considered fully recovered.

[6] The medical officer reported that 29 other patients had received injections of the same arsenical preparation on the same date and using the same technique without any untoward symptoms developing.

(30-1932.) A patient who denied exposure and infection (married) had been received on board a ship while under treatment for scabies. This condition had not responded to treatment. A darkfield examination showed numerous atypical spirocheta and a Kahn blood test was reported as plus-minus. The patient was given a 0.3-gram intravenous injection of neoarsphenamine as provocative. On November 6 and 9, 1932, he received 0.3-gram intravenous injections of neoarsphenamine as his first and second injections of the first course. The dilution of the last injection was stated as 0.3 gram of neoarsphenamine in 7 cubic centimeters of distilled water and the rate of injection was 2 minutes for the 0.3 gram dose administered.

Twelve hours after the last injection of neoarsphenamine the patient complained of generalized giant urticarial wheals accompanied by intense itching. The lips and eyelids were swollen. He was given a 1-gram intravenous injection of sodium thiosulphate and immediately transferred to a naval hospital. Upon admission to the hospital an examination revealed symptoms as noted while the chief complaint of the patient was an itching of the skin. He was given a 1-gram intravenous injection of sodium thiosulphate one fourth cubic centimeter of adrenalin, 1 ounce of magnesium sulphate, and 20 grains of sodium bicarbonate.

On November 15, 6 days after onset of symptoms, it was considered that the patient had fully recovered from the reaction. He was retained at the hospital for observation and treatment for the scabies. While in the hospital the following Kahn blood tests were taken and reported:

	Kahn blood test
Nov. 11	2 plus.
Nov. 22	Plus-minus.
Nov. 29	Negative.

On December 12 the patient was considered fully recovered and was discharged to duty.[7]

(31, 32, 33, 34, 35, 36, 37, 38, 39, 40-1932.) In September 1932 10 men on 1 ship experienced reactions. The cause was not definitely determined. The reporting medical officer suspected the drug due to the fact that all patients treated on that date had similar reactions.

Nine of the men received 0.3 gram intravenous injections of neoarsphenamine. The tenth received a 0.6 gram intravenous injection

[7] It was the opinion of the reporting medical officer that the peculiar Kahn reactions may have been false serum reactions. The patient had scabies, but he was of the opinion that the urticaria was due to neoarsphenamine, although patch test performed with neoarsphenamine was negative.

because it was the fourth injection of his fifth course. Listed below are the injections, courses, and dosage:

	Injection	Course	Dosage in grams
No. 1	First	Third	0.3
No. 2	do	do	.3
No. 3	do	do	.3
No. 4	do	Fourth	[1].3
No. 5	do	do	.3
No. 6	do	do	.3
No. 7	do	Sixth	[1].3
No. 8	do	Seventh	.3
No. 9	do	Eighth	.3
No. 10	Fourth	Fifth	.6

[1] Reinfection.

In case no. 4 it was considered a case of reinfection. After the first infection the individual had received 3 courses of arsenicals; after reinfection he received 3 more courses of arsenicals and had just received the first injection of his fourth course when the reaction occurred.

In case no. 7 it was considered a case of reinfection. After the first infection the individual had received 3 courses of arsenicals; after reinfection he received 5 more courses of arsenicals and had just received the first injection of his sixth course when the reaction occurred.

The dilution of 9 of the injections was stated as 0.3 gram of neoarsphenamine to 5 cubic centimeters of distilled water of unknown age but freshly sterilized. The dilution of 1 injection was 0.6 gram of neoarsphenamine to 10 cubic centimeters of distilled water of unknown age but freshly sterilized. The rate of injection in all cases was stated as 5 seconds for each cubic centimeter of solution administered.

In all cases the onset of symptoms of reaction was approximately 15 minutes after the individual had received the injection of neoarsphenamine. Symptoms of all cases are as follows:

	Cases
Headaches	10
General malaise	10
Chills	7
Vomiting	5
Nausea	4

Treatment prescribed was as follows:

	Cases
Magnesium sulphate, 1 ounce, either 2 or 3 hours after the first symptoms	10
Potassium acetate, grains XX for 3 doses, either 2 or 3 hours after the first symptoms	10
Codeine ½ grain and ephedrin ¼ grain, capsules, 2 hours after the first symptoms	5
Codeine, ½ grain by mouth, 2 hours after the first symptoms	1

	Cases
Codeine, ½ grain by mouth, 4 hours after the first symptoms	1
Sodium thiosulphate, 0.6-gram dose intravenously, the following morning after the reaction	6

Urinalysis was reported negative in 6 cases and no mention was made of report of urinalysis in the remaining 4 cases. No facilities were available for making other laboratory examinations.

Complete recovery occurred in all cases within 24 hours. Individual recovery was reported as follows: 1 case within 12 hours; 2 cases within 18 hours; and 7 cases within 24 hours.

Samples of the neoarsphenamine of the same lot number were forwarded to the Bureau for analysis. These samples passed satisfactory toxicity tests at the National Institute of Health, United States Public Health Service. The following is quoted from the report of examination of the samples submitted:

> The toxicity test was satisfactory, passing well over the official requirements for neoarsphenamine; it is indicated that the product is apparently not unusually toxic and is satisfactory for clinical use. The history of the water being used does not offer definite explanation for the reaction, the only suggestion being that it was of unknown age.

Arsenical Dermatitis

Deaths.—One *fatal* case (41-1932) of arsenical dermatitis following the use of neoarsphenamine occurred during the year 1932. The case history is listed below.

During the 8-year period (1925–32) there were five deaths due to arsenical dermatitis following the administration of neoarsphenamine. During this period there were 623,852 doses of neoarsphenamine administered, giving a ratio of 1 death to 124,770 doses administered.

Using the figures of a previous article, which were based on the 5-year period 1927–31, the case fatality rate for neoarsphenamine dermatitis cases for the 6-year period 1927–32 is 4.72 percent. This calculation is based on 106 cases with 5 deaths following the administration of neoarsphenamine. The calculations thus derived reduce the case fatality rate from 4.94 percent for the 5-year period 1927–31 to 4.72 percent for the 6-year period 1927–32, or a reduction of 0.22 percent.

Three cases with no deaths followed the administration of sulpharsphenamine.

Fatal case—(case history).—(41-1932.) A patient (female supernumerary) was given a diagnosis of syphilis on January 16, 1932, because a secondary rash appeared on her body and a Kahn blood test was reported as 4 plus. Arsenical treatment was instituted and the patient was given a 0.4-gram intravenous injection of neoarsphenamine on January 19, a 0.5-gram injection on January 22, and 0.6-gram injections on January 26, 29, February 2, 9, 16, 23, and March 1. The total dosage administered was 5.1 grams, given over a period of

43 days, or an average of 119 milligrams per day. The dilution of the last injection, given on March 1, was 0.6 gram of neoarsphenamine in 12 cubic centimeters of water. The rate of injection was stated as "slowly" for the 12 cubic centimeters of solution administered. As concurrent treatment the patient was given intramuscular injections of bismuth salicylate, 1.5 grams each, on February 19 and 26. On March 1 she was given 2 grams of bismo-cymol intramuscularly.

Three days after the last injection of neoarsphenamine, that given on March 1, the patient had a rash on her body, rapid pulse, temperature 103° F., and later a peeling of the skin which was diagnosed as dermatitis, exfoliative. Laboratory reports of blood counts were: Red blood count 3,600,000, white blood count 17,500, hemoglobin 75 percent, neutrophiles 84 percent, eosinophiles 6 percent, large lymphocytes 2 percent, small lymphocytes 6 percent, and transitionals 2 percent. Laboratory examination of urine was reported as positive, 2 plus, for albumin.

On March 4 the patient was administered 0.8 gram of sodium thiosulphate intravenously and sodium thiosulphate by mouth.

On March 22, 18 days after the first symptoms of reaction, the patient died with broncho-pneumonia, which was determined as the immediate cause of death.

Nonfatal cases of arsenical dermatitis.—During the year 1932 there were 24 cases which followed the use of neoarsphenamine. These cases were classified into 20 cases with *severe* reactions and 4 cases with *mild* reactions. Some of the available data for these cases are presented below in tabular form:

NEOARSPHENAMINE

Key[1]	Previous course of treatment	Current course of treatment							
		Course	Dose followed by arsenical dermatitis	Total grams	Number of days in course	Average milligrams per day	Concurrent treatment	Onset of arsenical dermatitis after last dose	Duration of arsenical dermatitis (days)
59	None	First	Second	1.0	5	200	None	3 days	11
63	do	do	do	.75	7	107	do	2 days	[2]
65	do	do	do	.75	7	107	3 injections bismuth	3 days	8
60	do [3]	do	Third	1.4	29	48	None	1 day	5
64	do	do	do	1.35	7	193	do	1 day	15
55	do [4]	do	Fourth	1.75	29	60	do	13 days	63
44	do	do	Fifth	2.8	29	97	4 injections bismuth	3 days	134
47	do	do	Sixth	3.15	18	175	12 injections bismuth	5 days	53
51	do	do	do	2.55	36	71	None	7 days	56
56	do [5]	do	do	2.4	36	67	do	3 days	76
57	do [5]	do	do	3.3	36	92	do	do	45
45	do	do	Seventh	3.6	51	71	8 injections bismuth and potassium iodide drops.	33 days	190
53	do	do	do	3.9	52	75	Mercurial inunctions 3 times a week.	8 days	56
58	do	do	do	3.15	43	73	1 injection bismuth	3 hours	58
62	do [6]	do	do	3.8	43	88	10 injections bismuth	1 day	62
42	do	do	Eighth	4.35	50	87	8 injections bismuth	About 10 days	77
54	do	do	Eleventh	5.85	[7] 79	74	4 injections bismuth	1 day	8

[1] "Key" refers to individual case number.

[2] 15 plus.

[3] This patient had a severe reaction following his second injection of his first course (key 59). Treatment was discontinued for a period of 23 days. He was then administered a third injection of his first course. A severe reaction followed this injection.

[4] Native (N-seaman second class) of Guam treated for "yaws."

[5] Natives (civilians) of Guam treated for "yaws."

[6] Supernumerary pateint (V.A.P.).

[7] This patient received 4 injections of neoarsphenamine, total 1.65 grams administered over a period of 22 days or an average of 75 milligrams per day. He was then returned to his ship for duty and his treatment was resumed after an interval of 14 days. He then received 7 injections of neoarsphenamine, total 4.2 grams administered over a period of 43 days or an average of 98 milligrams per day. The entire course consisted of 11 injections of neoarsphenamine, total 5.85 grams administered over a period of 79 days or an average of 74 milligrams per day.

NEOARSPHENAMINE—Continued

Key	Previous course of treatment	Current course of treatment							
		Course	Dose followed by arsenical dermatitis	Total grams	Number of days in course	Average milligrams per day	Concurrent treatment	Onset of arsenical dermatitis after last dose	Duration of arsenical dermatitis (days)
43	Oct. 19, 1928, to Nov. 20, 1928, 2.7 grams neoarsphenamine; Dec. 10, 1928, to Feb. 16, 1929. 40 inunctions of mercury and 7 injections of bismuth; Aug. 12, 1932, to Oct. 9, 1932, 12 days' treatment with mercury and iodide by mouth; Aug. 26, 1932, to Oct. 3, 1932, 7 injections mercury.[8]	Second	First	.225	1		None	do	37
48	Aug. 2, 1929, to Oct. 2, 1929, 6 injections of salvarsan, total (?), and 6 injections of mercury; Apr. 19, 1930, 1 injection neoarsphenamine, total (?).	do	do	.1	1		do	8 hours	56
61	Nov. 18, 1929, to Jan. 7, 1930, 5.85 grams neoarsphenamine; Dec. 2, 1931, to Dec. 23, 1931, 17 injections bismuth; daily inunctions of mercury, dates (?).	do	do	.45	1		do	1 hour	25
46	Jan. 15, 1925, to Feb. 17, 1925, 3 injections arsenicals, type and amount not stated; Jan. 15, 1925, to Feb. 17, 1925, 7 injections mercury and potassium iodide; Nov. 18, 1931, to Jan. 14, 1932, 8 injections bismuth.	do	Seventh	4.05	43	94	do	7 days	42
52	Apr. 29, 1931, to July 14, 1931, 10 injections neoarsphenamine, total 4.5 grams, and 19 biweekly injections of mercury bichloride; Sept. 8, 1931, to Nov. 17, 1931, 9 injections neoarsphenamine, total 4.05 grams, and 20 biweekly injections of mercury bichloride.	Third	Third	1.35	15	90	5 injections mercury bichloride.	2½ days	15
50	Sept. 21, 1928, to Dec. 28, 1928, 11 injections sulpharsphenamine, total (?); 1929, 6 injections neoarsphenamine, total (?); Oct. 28, 1931, to Dec. 22, 1931, 40 mercurial inunctions.	do	Eighth	4.80	50	96	None	1 day	74
49	Feb. 12, 1931, to Apr. 14, 1931, arsenicals, type and amount (?); Aug. 13, 1931, to Oct. 22, 1931, arsenicals, type and amount (?); Feb. 11, 1932, to Apr. 15, 1932, arsenicals, type and amount (?).	Fourth	do	3.60	51	71	8 injections bismuth	4 days	32

[8] This patient had experienced previous attacks of arsenical dermatitis which were recorded as follows:
ACD—12-3-28. Dermatitis, exfoliative. D—2-26-29. To duty. 85 sick days.
Interval of 22 days.
RA—3-20-29. Dermatitis, exfoliative. D—5-8-29. To duty. 49 sick days.
Interval of 11 months and 1 day.
A—4-9-30. Dermatitis, exfoliative. D—4-18-30. To duty. 9 sick days.
Interval of 5 days.
RA—4-23-30. Dermatitis, exfoliative. D—5-8-30. To duty. 15 sick days.

In all but 3 cases there is a history of from 2 to 11 injections of neoarsphenamine in periods ranging from 5 to 79 days, with varying amounts of the drug averaging from about 48 to 200 milligrams per day over the period comprising the current course of treatment. Of the 24 cases, 8 cases received bismuth injections, 2 cases received mercury treatment, and 1 case received bismuth injections and potassium iodide drops as concurrent treatment. No concurrent treatment was recorded as having been administered to the remaining 13 cases.

Primary clinical symptoms in cases of arsenical dermatitis and primary and later clinical symptoms noted throughout the duration of dermatitis—Neoarsphenamine.—The following clinical symptoms ("primary" and "primary and later") were noted in reactions resulting from the use of neoarsphenamine:

Primary symptoms

Dermatitis, exfoliative, location not stated	3	Urticarial-like eruption, mildly erythematous, most marked on hands, forearms, and legs	1
Dermatitis, exfoliative, of shoulders, arms, and chest	1	Urticarial rash	1
Dermatitis, exfoliative, extensive, of hands, arms, shoulders, and neck	1	Rash, copper colored, over arms, trunk, and legs	1
Dermatitis, dry, scaly, of face, arms, legs, and body	1	Redness of the entire body	1
Dermatitis of face, chest, and legs	1	Weeping of skin	1
Macular rash, fine, profuse, generalized	1	Fever	13
Macular rash, fine, generalized	1	Pruritus	7
Macular rash, fine, confluent, of face, neck, and chest	1	Headache	5
Maculo-papular rash on dorsal aspect of hands and ankles	1	General malaise	3
Maculo-papular rash on hands, face, and trunk	1	Chilliness	2
Maculo-papular eruption over the trunk, axillae, and extensor surface of the forearms	1	Chills	2
Maculo-papular eruption	1	Nausea	2
Maculo - erythenatous rash of trunk, shoulders, and arms	1	Edema of face	1
Erythematous rash, generalized	1	Edema of lips and eyes	1
Pruritic rash, dry, scaly, over face and body	1	Edema of skin around the eyes	1
Pruritic eruption of axillae, groins, waist line, and flexor surfaces	1	Edema of face and eyelids	1
		Conjunctivitis, moderate	1
		Conjunctiva, injection of	1
		Sclerae of eyes injected	1
		Bilateral lesions of the eyes	1
		Mouth and pharynx, mucuous membrane, inflamed and swollen	1
		Jaundice, slight	1
		Rapid pulse	1
		Stupor	1
		Pharynx congested	1
		Tongue fissured	1
		Pustules	1

Primary and later symptoms

Exfoliation present	17	Skin thickened, opposed surfaces fissured with considerable moisture present	1
Weeping of skin	5	Skin thickening, generalized	1
Dermatitis, exfoliative, location not stated	3	Fever	20
Dermatitis, exfoliative, of shoulders, arms, and chest	1	Pruritus	12
Dermatitis, exfoliative, extensive, of hands, arms, shoulders, and neck	1	Rapid pulse	7
Dermatitis, dry, scaly, of face, arms, legs, and body	1	Headache	6
Dermatitis of face, chest, and legs	1	General malaise	3
Macular rash, fine, profuse, generalized	1	Chills	3
Macular rash, fine, generalized	1	Chilliness	2
Macular rash, fine, confluent, of face, neck, and chest	1	Nausea	2
Macular eruption, generalized	1	Edema of eyes	1
Maculo-papular eruption	2	Edema of eyes and lips	1
Maculo-papular eruption over the trunk, axillae, and extensor surfaces of the forearms	1	Edema of skin around the eyes	1
Maculo-papular rash, generalized	1	Edema of eyelids and face	1
Maculo-papular rash on dorsal aspect of hands and ankles	1	Edema about eyes and ankles	1
Maculo-papular rash on hands, face, and trunk	1	Edema of the face	1
Maculo - erythematous rash of trunk, shoulders, and arms	1	Edema of the face and hands	1
Papulo-pustular eruption	1	Edema of arms	1
Erythematous rash, generalized	1	Edema of ankles and lower parts of the legs	1
Erythemo-squamous rash over the body	1	Hair, temporary loss of	1
Erythema, partaking of vesiculo-bullous type, of trunk and both upper and lower extremities	1	Hair, thinning of, on scalp and pubis	1
Pruritic rash, dry, scaly, over face and body	1	Nails, loosening of (no loss of)	1
Pruritic eruption of axillae, groins, waist line, and flexor surfaces	1	Retinitis, congestive	1
Urticarial-like eruption, mildly erythematous, most marked on hands, forearms, and legs	1	Conjunctivitis, moderate	1
Urticarial rash	1	Conjunctiva, injection of	1
Rash, copper colored, over arms, trunk, and legs	1	Sclerae of eyes injected	1
Redness of the entire body	1	Bilateral lesions of eyes	1
		Mouth and pharynx, mucous membrane, inflamed and swollen	1
		Pharynx congested	1
		Pharynx injected	1
		Tongue fissured	1
		Arrhythmia	1
		Soft systolic murmur	1
		Pustules	1
		Vesicles	1
		Jaundice, slight	1
		Stupor	1
		Fatigued easily	1
		Cough	1
		Trichophyton infection of the feet, moderate severity	1

Arsphenamine.—No cases of arsenical dermatitis attributable to arsphenamine were reported in 1932.

Neoarsphenamine (case histories).—(42-1932.) A patient who was last exposed at Norfolk, Va., in January 1932 was given a diagnosis

of syphilis on May 20, 1932, as the result of a primary lesion on his penis, a rash on his body, and 4 plus Kahn blood tests. Arsenical treatment was instituted and the patient was given an intravenous injection of 0.3 gram of neoarsphenamine on May 28. On June 4 he was given a 0.45 gram intravenous injection and on June 11, 18, 25, July 2, 9, and 16, 0.6 gram injections of neoarsphenamine. The total dosage was 4.35 grams administered over a period of 50 days, or an average of 87 milligrams per day. The dilution of the last injection was 0.6 gram of neoarsphenamine dissolved in 20 cubic centimeters of water and the rate of injection was about 1 minute. As concurrent treatment the patient was given 8 intramuscular injections of bismuth, 1 cubic centimeter each dose, between the dates of May 28 and July 16.

About 10 days after the last injection of neoarsphenamine, given on July 16, the patient had dermatitis, exfoliative, which covered his entire body. The secondary infection was extensive and caused pustules which were most marked over the lower abdomen. The patient was given daily intravenous doses of sodium thiosulphate, 1 gram each, from August 15 to September 1, 1932.

On October 11, 77 days after onset of reaction, the patient was considered recovered.

(43-1932.) A patient (Filipino) developed a sore on his lip during the last week of September 1928. The sore did not respond to local treatment and on October 15, 1928, the lesion was definitely indurated. A darkfield examination of the serum was reported positive for *Treponema pallidum* and on October 17 a Kahn blood test was reported 4 plus. Arsenical treatment was instituted and the patient was given a 0.3 gram intravenous injection of neoarsphenamine on October 19 and 0.6 gram injections on October 26, November 2, 13, and 20. The total dosage of this first course was 2.7 grams administered over a period of 33 days or a daily average of 82 milligrams per day. According to case history the patient reported with exfoliative dermatitis (neoarsphenamine) on December 3, 1928, 13 days after the last injection of neoarsphenamine. At this time the condition had existed for about 2 days. The patient was treated for 7 days aboard ship and was transferred December 10, 1928, to a naval hospital for further treatment, at which time he showed a severe dermatitis in the stage of general desquamation. Blood counts were recorded as follows: Red blood count 3,800,000; white blood count 26,200; hemoglobin 65 percent; differential: Polymorphonuclears 70 percent, eosinophiles 11 percent. On January 7, 1929, the patient showed marked alopecia and on January 19 he was placed on a liver diet for anemia. His general condition and blood picture slowly improved and on February 26 he was considered fully recovered and was discharged to duty after 78 sick days at the hospital. During hospitali-

zation the patient received 40 inunctions of mercury and 7 injections of bismuth potassium iodide. On March 20, 22 days after discharge to duty from the hospital, the patient reported to the sick bay with a history of an itching face for the past 24 hours. Examination revealed an edematous condition of the face involving the left eye. The dermatitis condition was mostly generalized but was more marked about the head. Reports of blood counts were as follows: White blood count 20,000; differential: Polymorphonuclears 77 percent, lymphocytes 18 percent, mononuclears 2 percent, eosinophiles 1 percent, and basophiles 2 percent. The patient was readmitted to the sick list with exfoliative dermatitis and immediately transferred to a naval hospital for observation and treatment. During the first few days of hospitalization the edematous condition of the face gradually became worse and involved both eyes. As before, the patient responded to treatment and on May 8 he was discharged to duty after 49 sick days. It will be noted that the patient's condition extended over a period of 156 days as follows:

Admitted December 3, 1928; discharged to duty February 26, 1929, 85 sick days. Interval of 22 days. Readmitted March 20, 1929; discharged to duty May 8, 1929, 49 sick days.

After an interval of 11 months and 1 day, and with no arsenical treatment reported as having been administered, the patient was again admitted to the sick list on April 9, 1930, with a general maculopapular rash which was particularly pronounced about the face, hands, and feet. The patient did not complain of any subjective sypmtoms except very mild itching. He responded to treatment and was discharged to duty on April 18, after 9 sick days. At the time of discharge to duty his condition had cleared with the exception of a moderate exfoliation. After an interval of 5 days he again was readmitted to the sick list with a recurrence of conditions and symptoms. Physical examination showed the patient to be a well-developed and well-nourished Filipino whose face appeared swollen, especially about the eyelids. His entire body, including the scalp, was covered with a marked exfoliating dermatitis. About the mouth, hands, feet, and ears the condition had progressed to a point of fissuring with the denuded areas moist with a clear serum. His pupils were equal and reacted to light and accommodation. His nose and throat were negative. The breath sounds of his chest were clear throughout and the heart sounds were normal. Blood pressure was recorded as 132/90. Some general adenopathy was present with the inguinal glands somewhat edematous. The reflexes were equal and active. Treatment was prescribed and on May 6 blood counts were reported as follows: Red blood count 3,770,000; white blood count 10,400; hemoglobin 70 percent; differential: Neutrophiles 30 percent, lymphocytes 38 percent, eosinophiles 31 percent, and transitionals 1 percent. No cause

for the eosinophilia could be found. By May 8 the dermatitis condition had improved to such an extent that it was considered that he had fully recovered. He was returned to duty after 15 days on the sick list.

As intercurrent treatment the patient received 12 days' treatment of mercury and iodide by mouth between the dates of August 12 and October 9, 1932. He also received 7 intramuscular injections of mercury salicylate, 0.13 gram each, between the dates of August 26 and October 3, 1932.

On October 29, 1932, the patient's second course of arsenical treatment was started and a 0.225 gram intravenous injection of neoarsphenamine was given on that date. The dilution of this injection was 0.225 gram of neoarsphenamine to 5 cubic centimeters of distilled water, and the rate of injection was 30 seconds for the 0.225 gram dose administered.

Twenty hours after this injection the patient's body became covered with a fine confluent macular rash which was dusky red in color and particularly prevalent on his face, neck, and chest. The palms of his hands and the soles of his feet were also involved. The mucous membrane of the mouth and pharynx was inflamed and swollen. The temperature was recorded as 102.4° F. The Dickens' test revealed no arsenic retention. The patient was transferred immediately to a naval hospital for treatment. Examination at the hospital revealed subjective symptoms of swelling of the face and eyelids and a rash covering the entire body and extremities, including the hands and feet, which itched intensely. The patient had a headache and his mouth felt raw inside. Objective symptoms were that the skin gave the impression of being inflamed; that the face was edematous and swollen, especially the eyelids, which were partly closed. The entire trunk and extremities were covered by a great number of dark red, scaly, slightly weeping lesions, more numerous on the chest, shoulders, back, and abdomen and gradually decreasing in numbers toward the hands and feet, although involving both to some extent. There was a large ulcer on the distal third of the right shin which was partly scabbed over. The sclerae of the eyes were injected and moderate conjunctivitis was present. The mucous membrane of the mouth and pharynx was inflamed, swollen, and had a leathery appearance. The blood pressure was recorded as 118/60. The anus was inflamed and leathery in appearance with few excoriations. Reflexes were superficial and deep, present, and active. There was a large dirty ulcer on the coronal sulcus, left side, of the penis. The following treatment was prescribed: October 30, 1932: Sodium thiosulphate, 1 gram, intravenously. Colloidal bath followed by thorough oiling with vegetable oil over the entire body. (When mentioned hereafter colloidal baths include applications of vegetable oil.) October 31:

Colloidal bath. Sodium thiosulphate, 1 gram, intravenously. Fischer's solution, 450 cubic centimeters, intravenously. November 1: Colloidal bath. Sodium thiosulphate, 1 gram, intravenously. Simple syrup, 1 dram, after meals. Sodium thiosulphate, 15 grains, after meals by mouth. Sodium bicarbonate, 20 grains, after meals. November 3: Continued treatment as previously mentioned. In addition, penis was soaked twice a day in a hot 1:5000 solution of potassium permanganate. November 6: Continued treatment as previously mentioned. Assurance was obtained that the patient scrubbed his teeth and washed his mouth with an alkaline mouth wash after each meal. November 10: All other treatment continued. Intravenous injections of sodium thiosulphate were discontinued. November 25: All medications discontinued with the exception of applying oil to the skin. From this date recovery continued and on December 6 the patient was considered fully recovered after 37 days on the sick list.

(44-1932.) A patient was infected at Los Angeles, Calif., on April 7, 1932. Darkfield examinations were positive for *Treponema pallidum* on April 25.

On April 27 arsenical treatment was instituted and the patient was given a 0.4 gram intravenous injection of neoarsphenamine. On May 4, 11, 18, and 25 he was given 0.6 gram intravenous injections. The total dosage was 2.8 grams of neoarsphenamine administered over a period of 29 days, or an average of 97 milligrams per day. The dilution of the last injection, that given on May 25, was 1:20, and the rate of injection was 2 minutes for the 0.6 gram dose administered. As concurrent treatment he was given 4 intramuscular injections of bismosol, 1 cubic centimeter each, between the dates of April 28 and May 23.

On May 28, 3 days after the last injection of neoarsphenamine, the patient was admitted to the sick list with an itching rash over his face and body. He stated that one day following the fourth injection he noticed a scattering pruritic rash on his body but did not report his condition as it caused him little embarrassment. One day following the fifth injection he noticed a recurrence of the rash on his body which seemed to be of a more severe form and which gradually became worse, with itching of the skin as the outstanding symptom; otherwise he stated that he felt well. Physical examination at this time revealed the skin of the face, extremities, and a large part of the trunk covered with a rough exfoliative type of dermatitis. Both eyes showed bilateral lesions resembling psoriasis. The skin around the eyes showed fairly marked edema. The temperature was 99, pulse 90, and respiration 24. Blood pressure 126/70. The remainder of the examination was nonessential. The patient had a history of psoriasis of both legs for the past 6 years but no history of anaphylaxis.

He was transferred to a naval hospital May 28 for observation and treatment. A physical examination at the hospital revealed that the chief symptoms were a dry and scaly skin. The urine was negative for arsenic. He was given an intravenous injection of 1 gram of sodium thiosulphate, and this treatment was continued three times a week until June 20. The patient became worse and the temperature elevated, reaching its maximum on June 6, and then gradually improved. On August 1 sodium thiosulphate was again administered intravenously in 1-gram doses for 3 weeks, and the skin condition improved. The patient at this time seemed otherwise normal. The generalized red macular rash which was accompanied by intense and generalized itching gradually improved, but the skin lesions persisted, especially on the trunk. There was some temporary loss of hair and temporary loosening, but no loss of nails. Absence of sweating was fairly well established until convalescence.

The condition improved gradually and on October 7, 134 days after onset of symptoms, the patient was considered recovered and was discharged to duty.

(45-1932.) A patient was given a diagnosis of syphilis on October 6, 1931, as the result of darkfield examinations which were positive for *Treponema pallidum*. Arsenical treatment was instituted and the patient was given a 0.45 gram intravenous injection of neoarsphenamine on October 13; a 0.3 gram intravenous injection on October 27; and 0.6 gram intravenous injections on November 3, 10, 18, 25, and December 2. The total dosage was 3.6 grams administered over a period of 51 days. or an average of 71 milligrams per day. The dilution of the last injection was 0.9 gram of neoarsphenamine dissolved in 15 cubic centimeters of distilled water, the rate of injection being 2 minutes for the 10 cubic centimeters of solution (0.6 gram dose) administered. As concurrent treatment the patient was given 8 intramuscular injections of bismuth salicylate, 0.1 gram each, between the dates of October 9 and December 2, 1931, and potassium iodide drops from October 9 to October 30.

On January 4, 1932, 33 days after the last injection, the patient was admitted to the sick list with a diffuse maculo-papular eruption over the trunk, axillae, and extensor surface of the forearms. He was given daily intravenous injections of sodium thiosulphate, 1 gram each, and local applications of mineral oil. On January 13 the patient was transferred to a naval hospital for treatment. Upon admission to the hospital it was considered that no severe reaction was present but that the case was in a subacute stage. Examination revealed an erythematosquamous rash generally present over the body, including the face. The skin was thickened and, on opposed surfaces, fissured with considerable moisture present. There was a thinning of the hair on the scalp and pubis, with a patch of alopecia

over the occiput. During the period in the hospital (Jan. 13 to June 22) there occurred four exacerbations, in each of which there was a rise of temperature from 101° F. to 102° F., with corresponding increase of the pulse rate. The skin became more hyperemic and swollen with much serous exudation from the opposed surfaces. Sodium thiosulphate was not given intravenously but was used as a dusting powder in combination with zinc oxide and camphor.

On June 22 he was discharged to duty to his ship with the skin remaining slightly roughened and dry but no other complications. Two days later he was readmitted to the sick list and immediately transferred to a hospital ship for further treatment. Upon admission to the hospital ship the patient showed generalized roughening and desquamation of the skin over the face, anterior trunk, arms, thighs, and legs. The condition was most marked over the extensor surface of the thighs and flexor surface of the legs, where there were numerous papules and considerable weeping of the lesions. The palms and soles were clear and smooth. The patient was given daily intravenous injections of sodium thiosulphate in 1 gram doses for a period of 10 days. The skin cleared rapidly and almost completely.

On July 12, 190 days after the first admission to the sick list, the patient was discharged to duty as cured.

(46-1932.) A patient was infected December 24, 1924, at Philadelphia, Pa. He was given a diagnosis of syphilis because of a sore on his penis. The report did not state whether darkfield examinations had been made or blood test taken. Treatment was instituted and administered as follows:

January 15 to February 17, 1925, 3 injections arsenicals, amounts not stated.
January 15 to February 17, 1925, 7 injections of mercury, dose and type not stated. Potassium iodide mixed treatment given at this time.
November 18, 1931, to January 14, 1932, 8 injections bismosol, 1 grain each.

The second course of arsenical treatment was started on January 12, 1932, and the patient was given a 0.45 gram intravenous injection of neoarsphenamine on that date. On January 19 and 26, February 2, 9, 18, and 23, he was given 0.6 gram intravenous injections of neoarsphenamine. The total dosage of this course was 4.05 grams administered over a period of 43 days, or an average of 94 milligrams per day. The dilution of the last injection of neoarsphenamine was 0.6 gram in 15 cubic centimeters of sterile distilled water. The rate of injection was stated as very slowly.

On March 1, one week after the last injection, the patient reported to receive another injection of neoarsphenamine. At this time it was noted that he had extensive exfoliative dermatitis of the hands, arms, shoulders, neck, and ears. The condition was very slight on the lower extremities. There was a slight amount of pruritus present but no other symptoms were noted. The Dickens' test was normal.

It was the opinion of the medical officer that the acute stage had passed when the patient was seen by him, which was 1 week after the last injection of neoarsphenamine. Sodium thiosulphate was not given as the patient maintained good elimination. Daily applications of cocoa butter were used externally.

On April 12, 42 days after admission to the sick list, the condition had cleared up with the exception of a slight dermatitis of the lobes of both ears.

(47-1932.) A patient was exposed at Brooklyn, N.Y., October 15, 1932. On November 12 he noticed a small firm pimple on the glans penis which broke down to form a small ulcer. Another ulcer which was firm, elevated, punched out with smooth edges and a crater-like base, but not containing pus, was noted on the coronal sulcus. On November 17 the diagnosis of syphilis was established by a darkfield examination of the serum from the lesion which was reported as positive for *Treponema pallidum*.

Arsenical treatment was instituted on November 16 and the patient received a 0.3 gram intravenous injection of neoarsphenamine on that date. On November 19 he received a 0.45 gram intravenous injection and on November 23, 26, 30, and December 3, 0.6 gram intravenous injections. The total amount given was 3.15 grams administered over a period of 18 days, or an average of 175 milligrams per day. The dilution of the last injection, that given on December 3, was 0.10 gram of neoarsphenamine to each 3⅜ cubic centimeters of water. The rate of injection was 20 cubic centimeters every 2½ minutes. As concurrent treatment the patient received 12 injections of bismuth, ⅜ grain to 1 cubic centimeter of water each injection, between the dates of November 15 and December 27.

On December 8, 5 days after the last injection of neoarsphenamine, the patient developed slight jaundice and was treated with calomel and salts. On December 10 he was given sodium thiosulphate. The jaundice cleared up but on December 25, 22 days after the last injection of neoarsphenamine, the patient developed a pruritus of the skin of the body. The pruritus increased in intensity and the skin became dry and showed a tendency to scale. Examination revealed that some small areas had begun to desquamate. There was some slight lymphadenopathy but otherwise the physical examination was essentially negative. Urinalysis and blood counts were reported normal. Dickens' tests showed good arsenical elimination. The patient was given eight intravenous injections of sodium thiosulphate, each injection containing 1 gram of sodium thiosulphate in 10 cubic centimeters of water. Olive-oil rubs and calamine lotion was applied locally. The pruritus gradually subsided and the desquamation gradually cleared up. On January 2, 1933, a Kahn blood

test was reported as negative. On January 26 the patient was placed on liver extract which was given three times daily.

On January 30, 53 days after the symptoms of jaundice, or 36 days after the symptoms of pruritus, the patient was considered to have fully recovered from the arsenical reaction and he was discharged to duty with no complaints.

(46-1932.) A patient was exposed July 13, 1929, at Matagalpa, Nicaragua. A primary lesion was noted August 15 and the diagnosis of syphilis confirmed on August 20 as the result of a darkfield examination of the serum from the lesion which was positive for *Treponema pallidum*. The patient received 6 intravenous injections of salvarsan between the dates of August 2 and October 2 as his first course of treatment. As concurrent treatment he was given 6 injections of mercury. On April 19, 1930, an intravenous injection of neoarsphenamine was given but the course was discontinued for reasons not stated.

On November 18, 1932, the patient complained of headaches, general malaise, and loss of energy. A physical examination revealed generalized glandular adenopathy and his health record revealed that on November 7 a Kahn blood test was reported 4 plus. In view of the findings it was considered that the patient was in need of antiluetic treatment and a 0.1 gram intravenous injection of neoarsphenamine was given. The dilution of the injection was 0.1 gram of neoarsphenamine in 5 cubic centimeters of distilled water and the rate of injection was 3 minutes for the 0.1 gram dose.

Eight hours after the patient had received the injection of neoarsphenamine he complained that "he felt feverish." Examination revealed a temperature of 105.5° F., pulse 130, headache, nausea, stupor, and redness of the entire body. Laboratory findings were: White blood count 21,000; red blood count 4,960,000; urinalysis, negative; and Dickens' test, slight elimination of arsenic. The patient was put to bed and placed on a liquid diet. He was given 20 grains of sodium bicarbonate, by mouth, in a cup of hot water every 3 hours. A hot water bottle was applied to his extremities and an ice cap applied to his head. He was given 2 ounces of magnesium sulphate and palliative treatment. Sodium thiosulphate, 1 gram dose, was given intravenously. At 10:15 p.m. he was given a hypodermic injection of 5 minims of adrenalin.

November 19. The patient's temperature was 99-101° F., pulse 90. A white blood count was reported as 21,978. The patient's fluid intake was 1,300 cubic centimeters and output was 1,400 cubic centimeters. His eyes were swollen and edematous. A smear showed gram positive cocci from the eyes and the Dickens' test was positive for arsenic. The patient was given a 1 gram intravenous injection of sodium thiosulphate. Palliative treatment was continued.

November 20. The patient's temperature was 102.5° F., pulse 130. At 11:05 a.m. he was given Dover's powder, grains X, by mouth. Palliative treatment was continued and the patient was allowed to take hot tea *ad libitum*. A 1-gram dose of sodium thiosulphate was given intravenously. Urinalysis was reported negative for albumin and casts and Dickens' test was slightly positive for arsenic. The patient's fluid intake was 2,200 cubic centimeters and he voided 120 cubic centimeters. A white blood count was reported as 21,000 and a differential count revealed polymorphonuclears as 77 percent.

November 21. The temperature was recorded as 100° F. The patient's entire body surface was red in color, his ankles and lower part of his legs were swollen and edematous. His nausea and stupor had cleared up and he was fairly comfortable. A 1 gram intravenous injection of sodium thiosulphate was administered. The Dickens' test was positive for arsenic. Urinalysis was negative. Laboratory findings revealed a white blood count as 17,000; red blood count 5,000,000; differential count: polymorphonuclears 79 percent, lymphocytes 19 percent. The patient's intake was 1,700 cubic centimeters of fluid and he voided 2,200 cubic centimeters.

November 22. The temperature was 99° F. and the patient seemed to be fairly comfortable. Routine care and palliative treatment was continued. At 11 a.m. he was given 1 ounce of magnesium sulphate by mouth followed later by a 1 gram intravenous injection of sodium thiosulphate.

November 23. Temperature was normal and the patient had no complaints. His systemic condition remained about the same. The skin over his body began to exfoliate and there was some weeping present, the edema of his ankles improving. He was ingesting and eliminating fluids freely. Laboratory findings were reported as follows: White blood count 12,500; red blood count, 4,980,000. The Dickens' test was slightly positive for arsenic.

November 25. The temperature was normal and the patient had no complaints. He was given forced fluids and a free diet. The exfoliation was increasing and a swelling of the face and hands was noted. He was given bath soaks which consisted of one half glass of corn starch and one half glass of sodium bicarbonate. A 1-gram dose of sodium thiosulphate was administered intravenously. Urinalysis was reported as negative.

November 26. The temperature was normal but no improvement in the patient's condition was noted. He was given a milk diet. At 11 a.m. he was given a colloidal bath with symptomatic relief of the pruritus, and hot soaks for 10 minutes in a bath which contained one half glass of corn starch and one half glass of sodium bicarbonate. After the bath the body was rubbed with a 1 percent solution of phenol and olive oil. Dickens' test was reported positive for arsenic.

November 27. The patient's temperature was normal, his appetite was good, and he had no complaints. His eyes were still swollen and there was a marked scaling over his chest and arms. His genitals were moist and there were small denuded areas over his ankles.

November 28. The temperature was 101° F. but the patient's appetite and elimination were good. Palliative and symptomatic treatment was continued. Hot boric acid packs were applied to his face and boric acid drops were placed in his eyes. The colloidal baths were continued. The patient was given a 1-gram dose of sodium thiosulphate intravenously. The Dickens test was reported negative for arsenic. Laboratory findings were reported as white blood count 12,500; differential: Band forms 4 percent, segmented 56 percent, lymphocytes, 36 percent; eosinophiles, 3 percent.

November 30. The temperature was 103.4° F., pulse 120. No improvement was noted and treatment was continued. A soapsuds enema was given. Urinalysis showed a slight trace of albumin and a few pus cells. Laboratory findings were reported as white blood count 12,800; red blood count 4,700,000; hemoglobin 95 percent; differential: polymorphonuclears 64 percent, neutrophiles 51 bands 7, segmented 52, eosinophiles 3, lyphocytes 26, and monocytes 11.

December 1. The patient's temperature was 99° F., his face was swollen and his eyes were closed. Each morning a purulent discharge issued from the eyes. Desquamation over his entire body was noted. Boric acid compresses were applied to the eyes. Sodium thiosulphate, 1 gram in 10 cubic centimeters of sterile water, was given intravenously. Laboratory findings reported white blood count 10,450; red blood count 4,960,000; hemoglobin 95 percent; differential: bands 4, segmented 5–6, eosinophiles 1, lymphocytes 2–8, monocytes 1. Urinalysis was reported as slightly positive for albumin and 2–4 pus cells per high power field.

December 2. The Dickens' test was reported negative for arsenic. Laboratory findings reported white blood count 9,750; red blood count 4,380,000; and hemoglobin 85 percent.

December 5. The patient's temperature was normal, and considerable improvement was noted. The swelling was subsiding and desquamation was improving. Hot packs were applied to his face during the day and cold cream was applied at night. Laboratory findings reported white blood count 8,800; red blood count 4,680,000. Urinalysis was reported negative and the Dickens' test negative for arsenic.

December 7. The temperature was normal, the patient had no complaints, his appetite was normal and his elimination was satisfactory. The skin of the chest, abdomen, and extremities was normal. Large areas of thick skin were falling from his feet, his face was partially clear. Urinalysis revealed few pus cells, Dickens' test was

negative for arsenic. Laboratory findings reported white blood count as 9,950, red blood count 4,810,000.

December 12. The patient was considered to have a satisfactory intake of a high caloric diet. Exfoliation had practically ceased but it did continue over the soles of his feet and the palms of his hands. Urine elimination was normal in quantity and negative in analysis. The Dickens' test remained negative for arsenic.

December 20. The temperature was normal and the patient had no complaints other than ease of fatigue. He was allowed up and about for short intervals during the day. Laboratory findings reported white blood count 9,100; hemoglobin 95 percent; differential: polymorphonuclears 59 percent. A Kahn blood test was reported 4 plus.

December 25. The patient sat up most of the day, his appetite was excellent, and the exfoliation had practically ceased. Laboratory findings reported white blood count 9,100; red blood count 4,890,000; hemoglobin 95 percent; differential: bands 2, segmented 57, eosinophiles 1, lymphocytes 32, monocytes 8. Urinalysis was reported negative. A Kahn blood test was reported as strongly 4 plus.

January 1, 1933. The patient was recovering rapidly, he was up and about all day, and had no complaints. It was estimated that he had lost 20 pounds in weight. Blood counts and urinalysis were reported normal.

The patient continued to improve as exercise was increased and allowed. On January 13, 56 days after the first symptoms of reaction, he was discharged to duty as fully recovered.

(49-1932.) A patient who was infected October 1, 1930, was given a diagnosis of syphilis as the result of Kahn blood tests which were reported 4 plus. Darkfield examinations failed to show *Treponema pallidum* in the serum from the lesion. Arsenical treatment was instituted and administered as follows:

First course, February 12, 1931, to April 14, 1931, — injections, neoarsphenamine, amount not stated.

Second course, August 13, 1931, to October 22, 1931, — injections, neoarsphenamine, amount not stated.

Third course, February 11, 1932, to April 15, 1932, — injections, neoarsphenamine, amount not stated.

On May 25, 1932, the patient started his fourth course of arsenical treatment and he received eight intravenous injections of neoarsphenamine, 0.45 gram each, from that date until July 14. The total dosage of this course was 3.6 grams administered over a period of 51 days, or an average of 71 milligrams per day. The dilution of the last injection, that given on July 14, was 0.45 gram of neoarsphenamine in 15 cubic centimeters of water, the rate of injection being 3 cubic centimeters per minute or 5 minutes for the 0.45-gram dose.

As concurrent treatment the patient was given eight injections of bismuth, 1 cubic centimeter each, between the dates of May 25 and July 14.

On July 18, 4 days after the last injection, the patient complained of an itching eruption in axillae, groins, waist line, and flexor surfaces. He had no constitutional symptoms. The patient was given five 20-grain doses of sodium thiosulphate.

On July 25, 7 days after admission to the sick list, he was transferred to a naval hospital for treatment of a generalized, pruritic, exfoliative dermatitis. His urine was negative for organic arsenicals by Autenrieth's test (Dickens' modification) and by routine and microscopic examination. The patient was given intravenous injections of 1 ampule of sodium thiosulphate daily for 5 days and then twice a week for the remainder of his stay in the hospital. He was also administered a saturated solution of sodium thiosulphate by mouth in 1-drachm doses, three times a day, during his period of hospitalization.

On August 19, 32 days after his first admission to the sicklist, the patient had apparently completely recovered and he was discharged to duty.

(50-1932.) A patient was infected July 26, 1928, and was given a diagnosis of syphilis because of a penile sore and a 4-plus Kahn blood test. Treatment was administered as follows:

First course, September 21, 1928, to December 28, 1928, 11 injections, sulpharsphenamine, amount not stated.

Second course, 1929, — injections, neoarsphenamine, amount not stated.

October 28, 1931, to December 22, 1931, 40 inunctions, 1 dram each, of mercurial ointment.

On January 24, 1932, the patient started his third course of arsenical treatment and he received eight intravenous injections of neoarsphenamine, 0.6 gram each, from that date until March 13. The total dosage for this course was 4.8 grams administered over a period of 50 days, or an average of 96 milligrams per day. The dilution of the last injection, that given on March 13, was 0.6 gram of neoarsphenamine in 10 cubic centimeters of water and the rate of injection was 2 minutes for the 0.6-gram dose administered.

On March 14, 1 day after the last injection, the patient was admitted to the sick list with a papulo-macular eruption. No chills or elevation of temperature was present. The urine was negative in all respects. The patient's condition grew worse and on March 18 he was transferred for observation and treatment because he had an advanced weeping and crusted trichophyton infection of both feet. An examination at the dispensary revealed an erythema multiforme partaking of the vesiculo-bullous type, involving the trunk and both upper and lower extremities. There was evidences of a trichophyton infection of

moderate severity on the feet about both malleoli and between the toes. Except for itching the patient was free from subjective symptoms. The temperature, pulse, and respiration were normal. Laboratory tests were as follows: Kahn blood test, negative; blood counts: red blood count 4,440,000, white blood count 7,850, hemoglobin 85 percent; differential: segmented 51 percent, lymphocytes 45 percent, monocytes 1 percent, basophiles 3 percent; urine: color straw, specific gravity 1.013, reaction acid, albumin negative, sugar negative, leukocytes 3 per high power field, erythrocytes 1 per high power field, crystals amorphous urates. The patient was placed in bed and fluids were forced. He was given sodium bicarbonate baths three times a day and applications of a bland ointment to the affected parts. Intravenous injections of sodium thiosulphate were administered in 0.1 gram doses daily for 5 days, after which he was given 0.1 gram doses twice weekly with the medications continued up to about May 14. The ointment applications were discontinued when the itching ceased. The exfoliation occurred early and the process repeated itself three times. At no time did the patient have constitutional symptoms. Healing occurred first upon the thorax, then upon the arms and hands, and lastly upon the feet.

On May 27, 74 days after first admission to the sick list, the patient had recovered sufficiently to allow performance of his duties and he was discharged to duty to his ship.

(51-1932.) A patient was infected at Norfolk, Va., in February 1932. A primary lesion appeared on his penis and darkfield examinations of the serum were positive for *Treponema pallidum*. Kahn blood tests were reported positive. Arsenical treatment was started and the patient was given a 0.3 gram intravenous injection of neoarsphenamine on February 25. On March 3, 10, 17, 24, and 31 he was given 0.45 gram intravenous injections of neoarsphenamine. The total dosage of his first course of treatment was 2.55 grams administered over a period of 36 days, or an average of 71 milligrams per day. The dilution of the last injection, that given on March 31, was 0.45 gram of neoarsphenamine in 10 cubic centimeters of sterile distilled water and the rate of injection was 5 minutes for the 10 cubic centimeters of solution administered. No concurrent treatment was given.

On April 7, 1 week after the last injection, the patient reported to the sick bay with a generalized macular erythematous rash which was more marked on the trunk, shoulders, and arms. The face was not involved. The Dickens' test was reported as negative. The patient was given 2 intravenous injections of sodium thiosulphate and 3 doses of magnesium sulphate.

On April 12, 5 days after admission to the sick list, the patient was transferred to a naval hospital for further treatment where an

examination revealed that the entire skin surface was covered by a maculo-papular rash which was most marked on the arms and legs. The skin lesions of the upper extremities were distinctly papular in character with slight swelling of both arms. The lesions on the lower extremities were macular in type, did not disappear on pressure, and had a general appearance of purpuric hemorrhages. The lesions varied in size from that of a pinhead to a pea with normal skin between the lesions. The patient was given calomel and a saline purge. Local applications of a saturated solution of boric acid were applied to the entire body surface, followed by applications of cottonseed oil which were applied three times a day. Fluids were forced. The patient was unable to receive intravenous injections of sodium thiosulphate because of the dermatitis and edema. On April 14 the dermatitis had spread to a greater extent with a watery serum exuding from the entire skin surface which dried on the body to form yellowish crusts. The Dickens' test at this time was negative. The patient was very uncomfortable and his temperature was 101° F., pulse 96, and respiration 20. From this date there was a slow but continued improvement. Two subsequent Dickens' tests were reported as showing a trace to a faint trace of arsenic. On April 19 the patient was given sodium thiosulphate three times a day by mouth in 1 gram doses. Sodium bicarbonate tub baths twice daily were substituted for the boric acid applications. These baths were followed by applications of cottonseed oil. On April 20 laboratory reports were as follows: White blood count 6,200; differential: basophiles 2, esoinophiles 7, bands 12, segments 37, lymphocytes 34, mononuclears 8. On May 12 sodium thiosulphate treatment by mouth was discontinued but the remaining treatment was continued.

On June 2, 56 days after first admission to the sick list, the patient was considered recovered from the reaction.

(52-1932.) A patient was infected in March 1931 at Panama City. Darkfield examination of the serum from the lesion was reported positive for *Treponema pallidum*. Treatment was instituted and administered as follows:

First course, April 29, 1931, to July 14, 1931, 10 injections, neoarsphenamine. Total dosage, 4.50 grams. April 29, 1931, to July 14, 1931, 19 biweekly intramuscular injections, one sixth grain each, of mercury bichloride.

Second course, September 8, 1931, to November 17, 1931, 9 injections, neoarsphenamine. Total dosage, 4.05 grams. September 8, 1931, to November 17, 1931, 20 biweekly intramuscular injections, one sixth grain each, of mercury bichloride.

The third course of arsenical treatment was instituted January 12, 1932. The patient was given a 0.45 gram intravenous injection of neoarsphenamine on that date and on January 19 and 26. The total dosage to date was 1.35 grams administered over a period of 15

days, or an average of 90 milligrams per day. The dilution of the last injection, that given on January 26, was 0.45 gram of neoarsphenamine dissolved in 20 cubic centimeters of sterile water. The rate of injection was 45 seconds for the 20 cubic centimeters of solution administered. As concurrent treatment to this third course of arsenical treatment the patient was given 5 intramuscular injections, one sixth grain each, of mercury bichloride.

On January 28, 2½ days after the last injection of neoarsphenamine, the patient complained that the skin of his hands, wrists, feet, and ankles was itchy and reddened. He stated that prior to this time he always became slightly nauseated during the administration of neoarsphenamine and on two occasions had vomited. These symptoms would pass off in about 2 or 3 minutes and no treatment was required. No feeling of nausea was experienced by him during the injection on January 26 and he felt perfectly well until the appearance of the symptoms mentioned above, 2½ days after the injection.

He was admitted to the sick list on January 29 and at this time his face and neck became involved and he developed a headache. The white blood count was reported as 10,500. The patient was placed in bed and he was given a laxative, aspirin for his headache, and zinc oxide dressings applied to his ankles.

On January 30 his face, neck, and extremities were thickly covered with a dermatitis, characterized by a mixture of maculo-papular eruption. The areas of vesicles contained clear serum which later turned grayish. The involved area was quite reddened and inflamed. Blood counts were reported as: red blood count 4,890,000; white blood count 6,200; hemoglobin 90 percent; differential: polymorphonuclears 66 percent, lymphocytes 23 percent, eosinophiles 2 percent. Urinalysis was negative. The patient was given a 0.5 gram intravenous injection of sodium thiosulphate. The pustules were evacuated and the zinc oxide dressings were continued to the ankles.

On January 31 the patient's condition was more aggravated. Many areas of pustules were present over the backs of the hands, ankles, and feet. The temperature was 98.4° F., and pulse 72. He was given a 1 gram intravenous injection of sodium thiosulphate. On February 1 and 2 he was given intravenous injections, 1 gram dose each day, of sodium thiosulphate. On February 2 his temperature was 100.2° F. and pulse 100. This was the first rise of temperature above normal.

On February 5 he had no complaints subjectively except moderate itching over the affected areas. The face and neck presented a typical picture of an exfoliative dermatitis which was scaling quite freely. The extremities had many raised papules, vesicles, and pustules but scaling had not yet begun. The trunk had only a few scattered

vesicles and pustules over it. The patient's condition as a whole seemed improved at this date and he stated that he felt quite comfortable. His temperature was 99.2° F. and pulse 100.

The next day, February 6, the face had peeled and now looked quite clear. The hands were improving. Urinalysis was reported as negative.

On February 7 there was considerable oozing from around the ankles and zinc oxide dressings were applied.

On February 9 the skin condition was rapidly improving, the face had peeled and was now clear; the hands, wrists, and ankles were about cleared up; and the patient stated that he felt well.

On February 11, 1932, he was discharged to duty as greatly improved and his time of recovery was stated as 15 days.

(53-1932.) A patient was given a diagnosis of syphilis in October 1931 as the result of positive Kahn blood tests. He could give no definite date of infection, but it is believed that he contracted syphilis from his wife, who is known to have the disease.

The patient was given the first injection of his first course on October 28, 1931, a 0.3-gram dose of neoarsphenamine injected intraveneously. On November 4, 10, 17, 24, December 9 and 18 he was given 0.6 gram intravenous injections of neoarsphenamine. The total dosage of this course to date was 3.9 grams of neoarsphenamine administered over a period of 52 days, or an average of 75 milligrams per day. The dilution of the last injection was 0.6 gram of neoarsphenamine dissolved in 20 cubic centimeters of sterile distilled water. The rate of injection was 1 minute for the 20 cubic centimeters of solution administered. As concurrent treatment the patient was given inunctions, 1 dram each, of mercurial ointment (stronger) 3 times a week from November 1 to December 18.

On December 26, eight days after the last injection, the patient had symptoms of an arsenical reaction but he did not report his condition until December 29, when he complained of dermatitis of his face, chest, and legs. He also complained of a woody feeling and much itching. No blood counts, Dickens' tests, urinalyses, or other laboratory examinations were made as it was considered too late. The patient was given an intravenous injection of sodium thiosulphate, 0.5 gram in 10 cubic centimeters of sterile distilled water.

Exfoliation began on January 11, 1932, and the patient was transferred to a naval hospital for treatment the next day. An examination revealed an extensive papulo-pustular dermatitis. He was given daily intravenous injections of sodium thiosulphate and local measures were used in treating the dermatitits.

On February 20, fifty sixth days after the first symptoms of reaction, the patient was considered recovered and was discharged to duty.

(54-1932.) A patient was infected at San Francisco, Calif., on October 24, 1931. He was given a diagnosis of syphilis on December 4 as the result of a typical buttonhole lesion on his upper lip, a dark-field examination of the serum from the lesion which was positive for *Treponema pallidum*, a 4 plus Kahn blood test, and the appearance of secondaries. Arsenical treatment was instituted on December 8, 1931, and the patient received an intravenous injection of 0.3 gram of neoarsphenamine on that date. On December 15, 22, and 29 he received intravenous injections of neoarsphenamine, 0.45 gram each day. The patient was returned to his ship, and on January 13, 1932, after an interval of 14 days, his course of treatment was resumed. He received a 0.6 gram intravenous injection of neoarsphenamine on that date. On January 20, 27, February 3, 10, 17, and 24 he received intravenous injections of neoarsphenamine, 0.6 gram each day. The total for the 11 injections was 5.85 grams of neoarsphenamine administered over a period of 79 days, or an average of 74 milligrams per day. The dilution of the last injection, that given on February 24, was 0.6 gram of neoarsphenamine in 10 cubic centimeters of distilled water and the rate of injection was 3 minutes for the administration of the 10 cubic centimeters of solution. As concurrent treatment the patient received 4 intramuscular injections of bismuth from December 7 to December 25, 1931.

On February 25, the day following the last injection, the patient reported to the sick bay with a headache and a copper-colored rash covering his arms, trunk, and legs which seemed to be just under the skin. His temperature was 99.8° F. Blood counts were reported as: White blood count 10,500; differential: polymorphonuclears 64 percent, lymphocytes 34 percent, and large mononuclears 2 percent. The Dickens' test indicated that the patient was eliminating arsenic. He was placed in bed, given a saline catharsis, three eighths grain of ephedrine sulphate, by mouth, every 2 hours for 3 doses, an injection of sodium thiosulphate, and external rubs with a 10 percent solution of sodium thiosulphate. The Dickens' tests, done twice daily for 5 days, were positive until the fifth day when it was reported negative. The injections and external application of sodium thiosulphate were continued for 4 days. After this treatment the rash began to disappear and desquamation started.

On March 4, 8 days after admission to the sick list, the patient was considered completely recovered and was discharged to duty.

(55-1932.) A native of Guam (seaman 2c.) who had no history of infection was under treatment for yaws as the result of positive Kahn blood tests.

The patient was given a 0.25 gram intravenous injection of neoarsphenamine on August 3, 1932, and 0.5 gram intravenous injections on August 10, 17, and 31. The total dosage of neoarsphenamine was

1.75 grams administered over a period of 29 days, or an average of 60 milligrams per day. The dilution of the last injection was 0.5 gram of neoarsphenamine in 10 cubic centimeters of sterile distilled water and the rate of injection was 1 minute (slowly) for the 0.5 gram dose.

On September 13, 13 days after the last injection, the patient developed fever and exfoliative dermatitis. The urine was reported as negative. The patient was given daily treatments with sodium thiosulphate, 2 grams orally and 1 gram intravenously. The skin was treated with applications of cottonseed oil applied every 4 hours. A high carbohydrate diet was prescribed.

On November 15, 63 days after onset of symptoms of reaction, the patient was discharged from treatment with no symptoms.

(56-1932.) A native of Guam (male) who had no definite history of infection was given a diagnosis of yaws because of clinical symptoms and positive Kahn blood tests. Arsenical treatment was administered as follows:

February 24, 1932, 0.3 gram of neoarsphenamine.
March 2, 1932, 0.6 gram of neoarsphenamine.
March 9, 1932, 0.6 gram of neoarsphenamine.
March 16, 1932, 0.3 gram of neoarsphenamine.
March 23, 1932, 0.3 gram of neoarsphenamine.
March 30, 1932, 0.3 gram of neoarsphenamine.

The total amount of neoarsphenamine administered was 2.4 grams over a period of 36 days, or an average of 67 milligrams per day. The dilution of the last injection was 0.3 gram of neoarsphenamine in 6 cubic centimeters of sterile distilled water and the rate of injection was 1 minute (slowly) for the 0.3 gram dose administered. According to the report no concurrent or intercurrent treatment was administered.

On April 2, 3 days after the last injection, the patient noticed that his skin became rough and cracked, which was accompanied by marked pruritus. The temperature was normal in the morning and irregular from 99.2° F. to 101° F. in the afternoon. The urine was negative for albumin and casts. Examination showed a generalized exfoliative dermatitis. The fever lasted about 2 weeks and was normal thereafter. The patient was given a high carbohydrate diet and 20 grains of sodium bicarbonate, three times daily. Cottonseed oil was applied to the skin every 4 hours. Sodium thiosulphate was administered, 1 gram intravenously and 1 gram orally, on alternate days. The urine was normal during the period of hospitalization.

On June 17, 76 days after admission to the sick list, the condition had entirely cleared and the patient was discharged as recovered.

(57-1932.) A native of Guam (female) who had a history of yaws for the past 30 years was given that diagnosis on May 23, 1932, as the result of a 4 plus Kahn blood test. She was given a 0.3 gram

intravenous injection of neoarsphenamine on May 25, and 0.6 gram intravenous injections on June 1, 8, 15, 22, and 29. The total amount for the six injections was 3.3 grams given over a period of 36 days, or an average of 92 milligrams per day. The dilution of the last injection was 0.6 gram of neoarsphenamine in 12 cubic centimeters of sterile distilled water and the rate of injection was 1 minute (slowly). No concurrent treatment was administered.

On July 2, 3 days after the last injection, the patient noticed a skin eruption over her shoulders and arms but did not report this condition until July 17, at which time she was admitted to the hospital with a fever and an extensive exfoliative dermatitis of the shoulders, arms, and chest. A loss of pigmentation in mottled areas of the hands and legs was noted. There were several subcutaneous nodules, the size of a lime, about the elbows and the left knee which were movable and apparently attached to the tendons. Laboratory examination of urine was reported negative but the fecal examination was reported positive for ascaris ova. The patient stated that she had noticed the discoloration of the skin of her extremities and the nodular formations over the tendons for the past 30 years. She was given 1 gram of sodium thiosulphate per day by mouth and local applications of mineral oil and phenol to the skin. On July 28 the dermatitis condition was markedly improved. At this time she was given oil of chenopodium. By August 5 the dermatitis was rapidly clearing and the treatment of oil of chenopodium was repeated.

On August 16, forty-five days after the first symptoms of reaction, the patient was considered fully recovered and she was discharged from the hospital.

(58-1932.) A patient was infected in June 1932 at Norfolk, Va. An indurated ulcer appeared on the penis 2 weeks after his exposure. Darkfield examination of the serum from the lesion was reported positive for *Treponema pallidum*.

Arsenical treatment was instituted and on July 6, 13, 20, 27, August 3, 10, and 17 the patient received 0.45 gram intravenous injections of neoarsphenamine. The total dosage for the seven injections was 3.15 grams. This was administered over a period of 43 days making a daily average of 73 milligrams per day. The dilution of the last injection, that given on August 17, was 0.45 gram of neoarsphenamine in 10 cubic centimeters of water. The rate of injection was 2 minutes for the 0.45 gram dose administered.

On July 14 the patient was given an intramuscular injection of 1 cubic centimeter of bismosol as concurrent treatment.

About 3 hours after the administration of the last injection of neoarsphenamine the patient complained of nausea, rash on body, and chilliness, which was followed by an itching of the skin. A physical examination revealed a congested pharynx, fissured tongue, and an

urticarial-like eruption which was midly erythematous and most marked on the hands, forearms, and legs. A urinalysis examination was reported negative. The patient was given 1 gram of sodium thiosulphate, injected intravenously, and 1 ounce of magnesium sulphate given by mouth. The following day, August 18, he was given a subcutaneous injection of 10 minims of adrenalin hydrochloride. The patient was transferred the next day to a naval hospital for further treatment. At the hospital the condition ran a severe course, but appeared to be improving slowly until September 11 at which time there supervened a chill, a rise in temperature, and an exacerbation of the skin inflammation with a marked burning and itching about the eyes. Tachycardia, arrhythmia, and a soft systolic murmur were also evident. The patient was given daily doses of sodium thiosulphate, 1 gram intravenously and 1 gram by mouth, from August 19 to September 17. He was also given general nursing care and attention and appropriate supportive treatment. All the severe symptoms abated under treatment, but photophobia persisted and on September 20 the ophthalmologist reported the presence of congestive retinitis.

On October 12 the skin condition was about clear with the exception of shallow ulcers which were noted on both corneas. On October 14, 58 days after the first symptoms of reaction, the skin was entirely clear, but the patient was transferred to the ophthalmological ward for treatment.

(59, 60-1932.) Two arsenical reactions were recorded for the same man. The patient was exposed at San Diego, Calif., on March 20, 1932. Diagnosis of syphilis was made as the result of a darkfield examination of the serum from the lesion which was reported positive for *Treponema pallidum* on March 25, 1932. He was given a 0.4 gram intravenous injection of neoarsphenamine on March 26, and a 0.6 gram intravenous injection 4 days later. The total dosage to date was 1 gram administered over a period of 5 days, or an average of 200 milligrams per day. The dilution of the second injection was stated as 1:20, and the rate of injection was 2 minutes. No concurrent treatment was administered.

On April 2, three days after the last injection, the patient had aches in his joints, chilliness, and stated that he did not feel well. He did not report his condition until the following day, when he was admitted to the sick list with a generalized flushing and body rash, malaise, and general body aches. Examination revealed a profuse, fine, macular rash all over his body and extremities. The rash was of a markedly pink color similar to scarlet fever but there was no circumoral color, no strawberry tongue, and no sore throat. His chest and lungs were essentially negative; temperature was 100° F., pulse 85, and respiration 20; and the white blood count was 9,200. The

patient was isolated, placed in bed, and was given a saline cathartic. On April 4, he was resting well and his temperature was normal. The rash was generalized and of a profuse scarlatina form with moderate itching. He was given a 0.5 gram intravenous injection of sodium thiosulphate, and 20 grain doses of sodium bicarbonate every 3 hours by mouth. Calamine lotion was applied locally. On April 5 the patient was resting well and his temperature was 99° F., pulse 80, and respiration 20. He was given a 0.7 gram intravenous injection of sodium thiosulphate on April 6 and the rash began to fade rapidly. The patient was much improved at this time. The next day, April 7, the rash was disappearing and his temperature, pulse, and respiration were normal. On April 8 he was given a 1 gram intravenous injection of sodium thiosulphate. The patient had no complaints at this time and his temperature, pulse, and respiration were normal. Two days later the rash had disappeared and there was no scaling present.

On April 13, 11 days after the first complaints, the patient was discharged to duty, symptom-free. The lesion on his penis had healed and his Kahn blood test was negative. Antiluetic treatment was ordered discontinued for 3 weeks.

On April 23 the patient was given his third injection, a 0.4 gram dose of neoarsphenamine injected intravenously. The total dosage administered to date (plus the third injection) was 1.4 grams administered over a period of 29 days, or an average of 48 milligrams per day. The dilution of this injection was 1:20 and the rate of injection was 2 minutes. The Dickens' test was reported as follows: First hour, no elimination; second hour, no elimination; third hour, poor elimination; fourth hour, fair elimination; fifth hour, good elimination; and the sixth hour, good elimination.

The next day the patient again had complaints of aching in his joints, feeling of chilliness, and a general body rash. He was admitted to the sick list on April 25 and an examination revealed that the rash was not as profuse nor as severe as that in the previous attack. The temperature was 100.5° F., pulse 80, and respiration 20. He was placed in bed; given a 0.5 gram intravenous injection of sodium thiosulphate; and sodium bicarbonate, 20 grain doses, every 3 hours by mouth. The medical officer reported that when the first reaction occurred there was some doubt as to whether it was due to neoarsphenamine. All antiluetic treatment was abandoned for a period of over 3 weeks and then it was decided to try another injection.

On April 26 the patient was resting well; the rash was fading; the temperature, pulse, and respiration were normal; and the pain in his joints had disappeared. This date he was given a 0.75 gram intravenous injection of sodium thiosulphate. He had made no complaints but he was given a 1 gram intravenous injection of sodium thiosulphate 2 days later.

On April 29, 5 days after onset of symptoms, the patient was discharged to duty feeling fine. The Kahn blood test was negative.

(61-1932.) A patient was infected at Canton, China. He was given a diagnosis of syphilis on November 1, 1929, as the result of darkfield examinations which were reported positive for *Treponema pallidum*. Treatment was administered as follows:

First course, November 18, 1929, to January 7, 1930, 8 injections, neoarsphenamine, total dosage, 5.85 grams; December 2, 1931, to December 23, 1931, 17 injections, 1 grain each, bismuth salicylate; [?] to [?], daily inunctions of mercury.

On February 16, 1932, the patient was started on his second course of arsenical treatment and he was administered a 0.45 gram intravenous injection of neoarsphenamine on that date. The dilution of the injection was 0.9 gram of neoarsphenamine to 22.5 cubic centimeters of distilled water and the rate of injection was 2 minutes for the 0.45-gram dose administerd.

One hour after this injection the patient showed a marked rise in temperature, headache, and an urticarial rash; and developed a chill. The next day, February 17, his symptoms were much accentuated; the dermatitis more pronounced; some edema about the eyes and ankles; skin thickening about the face, neck, and arms but no exudation of serum; and the temperature ranging about 102° F. The urine was negative for albumin and the output was normal. The Kahn blood test was 4 plus. The patient showed a history of arsenical reaction (dermatitis, exfoliative) in 1930 when he was returned to duty after 61 days, with a recommendation for light duties. For this reason no further arsenical treatment was given. Daily inunctions of mercury were given from which the patient developed some soreness of gums. As treatment for this reaction he was given 1 gram intravenous injections of sodium thiosulphate daily for 3 days. On February 19 he was transferred to a naval hospital for treatment. An examination revealed a generalized thickening of the skin of the body, red in color, with prominence of the follicles, and some vesicle formation over the malar prominences. There was much itching at contact points and the capillary circulation was sluggish. There was marked painless hypertrophy of the post-cervical, epitrochlear, and inguinal lymph nodes. The spleen and liver were not enlarged or tender. The pupils tended to be oval. The pharynx was injected with prominence of the lymphatic follicles. The blood pressure was 110/50. The icterus index test showed a reading of 4 and a report of urinalysis was: Appearance, very cloudy straw; reaction, acid; specific gravity, 1.015; albumin, 1 plus; sugar, negative; and numerous calcium oxalates. The patient was given 1-gram intravenous injections of thiosulphate daily for 9 days.

On March 12, 25 days after the first symptoms, the patient was considered recovered and was returned to duty.

(62-1932.) A Veterans' Administration patient, who does not know when or where he contracted the disease, was given a diagnosis of syphilis as the result of three positive Kahn blood tests. He was given a 0.2-gram intravenous injection of neoarsphenamine on August 10, 1932, and 0.6 gram intravenous injections on August 17, 24, and 31, and September 7, 14, and 21. The total dosage was 3.8 grams, administered over a period of 43 days, or an average of 88 milligrams per day. The dilution of the last injection, that given on September 21, was 0.6 gram of neoarsphenamine in 20 cubic centimeters of sterile water, and the rate of injection was 2 minutes for the 0.6-gram dose administered. As concurrent treatment, ten 1-cubic-centimeter intramuscular injections of bismuth in oil were administered between the dates of August 19 and September 23.

On September 22, the day following the last injection of neoarsphenamine, the patient noticed a slight skin rash. The rash was mild enough so that the patient gave it no thought and he went on a week-end liberty on September 24. He returned on Sunday because the rash became worse. At this time the patient had a dry scaly dermatitis which involved the face, arms, legs, and body. The condition was more pronounced about the face and there was a slight swelling in that area. The dermatitis was associated with a mild irritation and itching. At no time was the patient a bed patient although he did not feel as well generally as before the onset of dermatitis. An irritating cough and a rise in temperature to 100° and 102° F. from October 11 to October 14, 1932, were the only other symptoms noted. The Dickens' test given on September 21 showed normal elimination of arsenic. While under treatment the dermatitis showed a slow but steady improvement until October 21. The patient was granted leave to attend to financial affairs at home and returned on October 30 with the dermatitis of his face much worse and showing superficial ulcerations of tips of the lobes of both ears. Since that time he has shown continued improvement and when he was discharged to duty the ulcerations of the ears had healed, the dermatitis of the arms and legs had cleared, and that of the face and cervical region had cleared with the exception of a few red raised areas about the size of acne papules. During the period of treatment the following blood counts were recorded:

Date	Red blood count	White blood count	Hemoglobin	Polymorphonuclears	Lymphocytes	Eosinophiles
			Percent	*Percent*	*Percent*	*Percent*
Sept. 30, 1932	5,100,000	8,800	90	68	27	3
Oct. 3, 1932	4,980,000	13,400	90	66	16	16
Oct. 11, 1932	4,680,000	14,050	90	66	21	8
Oct. 18, 1932	4,320,000	11,300	85	62	19	16
Oct. 31, 1932	4,750,000	37,250	85	84	7	5
Nov. 16, 1932	4,030,000	14,100	85	72	21	6

Urinalysis was continually negative. Treatment consisted of keeping the bowels open, forcing fluids, and a soft bland diet. The patient was administered 1-gram intravenous injections of sodium thiosulphate on the following dates: September 28, October 5, 12, 19, and 31, and November 2, 10, and 16. From October 31 he was given potassium iodide by mouth.

On November 23, 62 days after the first symptoms were noted, the patient was discharged from hospital and allowed to go home on leave.

(63-1932.) A patient was last exposed at Shanghai, China, on April 9, 1932. An ulcer appeared on his penis and he reported his condition on May 19. A darkfield examination of the serum from the lesion was positive for *Treponema pallidum* on May 20. On June 5 a Kahn blood test was reported as 4-plus.

The patient was started on his first course of arsenical treatment on May 29 and was administered a 0.3-gram intravenous injection of neoarsphenamine on that date. On June 4 he was given a 0.45-gram intravenous injection of neoarsphenamine. The total amount administered to date was 0.75 gram given over a period of 7 days, or an average of 107 milligrams per day. The dilution of the last injection was 3.3 cubic centimeters of water per each $\frac{1}{10}$ gram of drug used and the rate of injection was approximately 20 cubic centimeters per minute.

On June 6, 2 days after the last injection, the patient reported to the sick bay complaining of an itching rash about his hands and ankles. Examination revealed a thinly scattered maculo-papular rash on the dorsal aspect of the hands and about the ankles. He stated that following the first injection he felt as if he had slight chills and fever that night and the next day he experienced a slight general malaise. Following his second injection he stated that he experienced the same symptoms. No history was elicited of previous dermatitis or renal impairment.

The rash became generalized the next day and in some locations the lesions were confluent, presenting rather extensive, red, slightly raised areas, the color of which disappeared upon pressure. Moderate itching continued at intervals and the patient stated that his hands felt stiff. He was placed in bed, fluids were forced, and he was administered 1½ ounces of magnesium sulphate. At 1 p.m. his temperature was 99.4° F., pulse 95, respiration 16, and at 3 p.m. his temperature was 100° F., pulse 95, and respiration 15. At 8:15 p.m. he was given 1-gram doses of sodium thiosulphate administered intravenously. The following morning, June 8, his temperature was 98.6° F., pulse 85, and respiration 16. The rash and general condition appeared unchanged. He was given a 1-gram intravenous injection of sodium thiosulphate at 8 a.m. and later during the day was trans-

ferred to the tender ship for further treatment. The patient was placed in bed and given forced fluids and injections of sodium thiosulphate and adrenalin. Urinalysis was negative for albumin and sugar.

On June 15 the patient was much improved and the rash was clearing up. The Dickens' test was negative for arsenic.

On June 21, fifteen days after the patient first reported his condition, he was considered sufficiently recovered to be discharged to duty under treatment.

(64-1932.) A patient was infected September 1, 1932, and his case was diagnosed by a typical chancre on the penis and a 4 plus Kahn blood test. He was given a 0.3 gram intravenous injection of neoarphenamine on September 22, a 0.45 gram intravenous injection on September 25, and a 0.6 gram intravenous injection on September 28. The total amount of neoarsphenamine administered to date was 1.35 grams given over a period of 7 days, or an average of 193 milligrams per day. The dilution of the last injection was 0.6 gram in 10 cubic centimeters of sterile water, and the rate of injection was 2 minutes for the 0.6-gram dose administered. No concurrent treatment was given.

On September 29, the day after the last injection, the patient had an elevation of temperature, a maculo-papular rash on his hands and face which gradually spread to the trunk, and an increase in pulse rate. There were no other symptoms, and the patient stated that he felt perfectly well with the exception of the fever. He was then given an intravenous injection of sodium thiosulphate, 1 gram in 10 cubic centimeters of water. On October 1 he was transferred to a naval hospital for further treatment. On admission he had a generalized macular eruption covering the entire body, which appeared to be fading, and which the patient stated was better than on the 2 previous days. The temperature was 101° F., pulse 88, and respiration 18. There were no mucous patches of secondary syphilis and he had no gastro-intestinal symptoms. The patient did not appear to be critically ill and the ulcer on his penis was almost healed. There was no jaundice and no evidence of liver involvement. The case appeared as a mild aresenical dermatitis which was already improving under the treatment given before admission. He was placed in bed and given forced fluids and free saline catharsis. On October 3 the patient was given 1 gram of sodium thiosulphate injected intravenously. The next day he was given another intravenous injection of sodium thiosulphate of the same dosage and the rash then began to disappear. The temperature returned to normal and remained so during the remainder of his stay in the hospital. On October 6 and 11 he was given 1 gram intravenous injections of sodium thiosulphate.

On October 14, fifteen days after admission to the sick list, the patient was considered recovered and was discharged to duty.

(65-1932.) A patient was exposed at San Francisco, Calif. A diagnosis of syphilis was confirmed January 28, 1932, by bilateral painless inguinal adenopathy and a darkfield examination of the serum from the indurated ulcer which was positive for *Treponema pallidum*. Arsenical treatment was immediately instituted and the patient was given a 0.3 gram intravenous injection of neoarsphenamine on January 28, 1932, and a 0.45 gram intravenous injection on February 3, 1932. The total dosage administered was 0.75 gram given over a period of 7 days, or an average of 107 milligrams per day. The dilution of the last injection was stated as 0.45 gram of neoarsphenamine in 20 cubic centimeters of water, and the rate of injection was approximately 5 cubic centimeters per minute. As concurrent treatment the patient was given intramuscular injections of bismuth salicylate, 2 grains each dose, on January 28 and February 1 and 4.

On February 6, three days after the last injection, the patient developed a temperature of 105° F., generalized erythematous rash, swelling of the lips and eyes, injection of the conjunctiva, and a headache. He was given forced fluids; rectal irrigation; saline purge; and sodium thiosulphate intravenously, 1 gram each dose, at 9 a.m., 11 a.m., 3 p.m., and 8 p.m. The next day the urine was negative for arsenic and the patient was given a saline purge and sodium thiosulphate, 1 gram, intravenously. On February 8 and 10 the patient was given sodium thiosulphate intravenously, 1 gram each day.

On February 14, eight days after the first symptoms of reaction, the patient was considered recovered and was discharged to duty.

Miscellaneous Effects Following the Administration of Arsenical Compounds

Damage to the liver.—There were two instances of *mild* reactions with jaundice which were reported during the year 1932 following the use of neoarsphenamine. Case histories are as follows:

(66-1932.) A patient, who was exposed at Shanghai, China, on June 24, 1932, was given a diagnosis of syphilis as the result of a darkfield examination of the serum from the lesion which was reported positive for *Treponema pallidum*. The patient was given his first intravenous injection of neoarsphenamine, 0.3 gram, on July 7. The dilution of this injection was 0.3 gram of neoarsphenamine in 10 cubic centimeters of normal saline solution, and the rate of injection was 1½ minutes for the 0.3 gram dose. As concurrent treatment, the patient was given intramuscular injections of bismuth salicylate, 2 grains each, on July 7 and 11.

On July 15, eight days after the intravenous injection of neoarsphenamine, the patient reported to the sick bay with a generalized jaundice. He was placed in bed and given a soft meat-free diet. On July 23 he was transferred to a naval hospital, complaining of weakness and pain in his right upper abdomen. Slight jaundice was noted at this time and it was considered that the patient was in a convalescent stage.

On August 3 the patient was considered to be fully recovered and was discharged to duty after 19 days on the sick list.

(67-1932.) A patient, who was exposed at Honolulu, T.H., on June 16, 1932, had a primary lesion on the corona sulcus of his penis which appeared July 3. Darkfield examination of the serum from the lesion was reported positive for *Treponema pallidum* on July 5.

The patient was given a 0.3 gram intravenous injection of neoarsphenamine on July 12, a second 0.3 gram intravenous injection on July 19, and 0.6 gram intravenous injections on July 27, and August 2, 9, and 16. The total dosage administered was 3.0 grams. The dilution of the last injection was 0.6 gram of neoarsphenamine in 14 cubic centimeters of sterile distilled water, and the rate of injection was 2 minutes for the 0.6 gram dose administered. As concurrent treatment the patient was given inunctions of mercurial ointment (stronger), 1 dram each, three times a week from July 12 to August 22.

On August 22, six days after the last injection of neoarsphenamine, the patient presented himself to the sick bay with icterus of conjunctiva and buccal membrane. Laboratory examination of urine revealed the specimen as strongly positive for bile with a trace of albumin. On August 24 the patient was transferred to a naval hospital where symptoms of constipation and a conjunctiva of a markedly yellowish tint were noted. The icterus index test was reported as 23. He was given 2 grains of calomel in divided doses. Later, magnesium sulphate was administered followed by daily administrations of 4 ounces of sodium phosphate. He was placed on a fat-free diet. On August 29 the Icterus Index test was reported as 15 plus and on September 13 a similar test was reported as 8.

On September 16 the patient was considered fully recovered after 25 days on the sick list.[8]

Acute renal damage.—During the year 1932 there was one nonfatal case reported from this cause following the use of neoarsphenamine. This reaction was classified as *severe*. Case history is as follows:

(68-1932.) A patient was exposed at San Pedro, Calif. His condition was diagnosed as syphilis on June 23, 1932, as the result of general glandular adenopathy, repeated 4 plus Kahn blood tests, and repeated darkfield examinations which were positive for *Tre-*

[8] The reporting medical officer considered the effects to be of an accumulative nature and that no definite time or relationship could be traced to any one particular injection.

ponema pallidum. The patient was given a 0.3 gram intravenous injection of neoarsphenamine on June 28 and 0.45 gram intravenous injections on July 5 and 13. The dilution of the last injection was 0.45 gram of neoarsphenamine in 20 cubic centimeters of sterile water and the rate of injection was 90 seconds for the 0.45 gram dose administered. As concurrent treatment the patient was given two ⅙ grain intramuscular injections of mercury. Dates of administration were not stated.

Four hours after the last injection of neoarsphenamine (1:30 p.m.) the patient experienced a pain in the epigastrium which was accompanied by nausea. An examination revealed a pallor with dimness of vision, dilated pupils, pulse 170, but no rash of any character. Laboratory examinations were reported as follows: Urinalysis, negative for albumin and sugar; white blood count 13,000; polymorphonuclears 81 percent, lymphocytes 13 percent, eosinophiles 6 percent. The patient was placed in bed with external heat applied. One gram of sodium thiosulphate was given intravenously at this time. The temperature at 2 p.m. was recorded as 100° F. At 4 p.m. he was given a second intravenous injection of 1 gram of sodium thiosulphate. At 5 p.m. his temperature rose to 104° F., and at 5:30 p.m. he was transferred to a naval hospital for further observation and treatment. Upon admission to the hospital the patient's temperature was recorded as 104° F., pulse 90, and respiration 22. At this time he complained of photophobia, headache, slight nausea, and vomiting. Other symptoms found on examination were conjunctival injection of both eyes, edema of both eyelids, palpable anterior cervical glands and oliguria. Shortly after admission the patient was given 1 gram of sodium thiosulphate and 1 cubic centimeter of adrenalin. Two hours later he was given a hypodermic injection of 8 minims of adrenalin and $\frac{1}{100}$ grain of atropine sulphate. He was also given a proctoclysis of 500 cubic centimeters of a 5 percent glucose in normal saline solution. The next day, July 14, the Dickens' test showed elimination of arsenic in both specimens. The urine examinations also showed an acute nephritis. The patient was given 1 gram of sodium thiosulphate intravenously on this and the two following days. On July 17 headache was the only remaining symptom. On July 20 the urine was negative. By August 15 the patient had no symptoms and on August 18 he was discharged to duty as fully recovered after 36 sick days. Prior to discharge he was given three doses of bismuth salicylate, 0.13 gram each, and protoiodide of mercury, ⅓ grain, three times a day.

Aplastic anemia.—During the year 1932 there was one severe reaction reported from this cause and one mild reaction classified under the heading of agranulocytosis following the use of neoarsphenamine.

For statistical purposes these two reactions have been classified and included under the heading of aplastic anemia. Case histories are as follows:

(69-1932.) A patient, who denied any primary lesion and stated he had sexual intercourse only with his wife, was given a diagnosis of syphilis on June 22, 1931, because of iritis of the left eye and generalized secondaries from which darkfield examinations showed positive for *Treponema pallidum*. No blood tests were recorded. Treatment was instituted and administered as follows:

June 26, 1931, to August 11, 1931, 8 intravenous injections, neoarsphenamine (0.9 gram each).

June 26, 1931, to August 11, 1931, 6 intramuscular injections, mercury (1 grain each).

August 18, 1931, to October 6, 1931, 6 intramuscular injections, mercury (1 grain each).

October 13, 1931, to December 4, 1931, 8 intravenous injections, neoarsphenamine (0.6 gram each).

December 10, 1931, to February 10, 1932, 8 intramuscular injections, mercury (1 grain each).

February 16, 1932, to March 29, 1932, 7 intravenous injections, neoarsphenamine (0.6 gram each).

February 16, 1932, to March 29, 1932, 7 intramuscular injections, mercury (1 grain each).

The dilution of the last injection of neoarsphenamine, that given on March 29, was 1:20 and the rate of injection was 30 seconds for the 0.6-gram dose administered. The third course of treatment consisted of 4.2 grams of neoarsphenamine administered over a period of 43 days, or an average of 98 milligrams per day.

About 3 hours after the last injection of neoarsphenamine (March 29) the patient noted that his gums bled freely after he had cleaned his teeth. He was given 15 grains of sodium thiosulphate intravenously at 10 a.m. the next day and a similar injection at 4 p.m. The bleeding from the gums continued and the patient was administered two more intravenous injections of sodium thiosulphate, 15 grains each, on April 1, the first injection being administered at 10 a.m. and the second injection at 7 p.m. Purpuric spots appeared the following day and the bleeding from the gums continued. The patient was transferred to a hospital ship where he was given a blood transfusion of 400 cubic centimeters by direct method. On April 3 laboratory examinations were reported as follows: Red blood count 2,280,000; white blood count 10,150; hemoglobin 55 percent; differential: polymorphonuclears 80 percent, lymphocytes 20 percent; platelets 9,120. Blood type "B" international. On April 4 the patient was weak and was gradually becoming weaker. He had much uncontrollable bleeding from the gums, had vomited some clotted blood and had passed some tarry feces. Purpuric spots now appeared on the inner side of both thighs and right upper arm. Laboratory reports

were as follows: Red blood count 2,025,000; white blood count 10,000; hemoglobin 50 percent; differential: polymorphonuclears 83 percent and lymphocytes 17 percent. Schillings classifications: myelocytes 3, bands 6, normoblast 3, juveniles 7, segmented 84; Peroxidase stain: granulocytes 87 percent, agranulocytes 13 percent; reticulocytes 2.5 percent; platelets 14,145. Bleeding time 31 minutes plus. Coagulation time 6 minutes and 15 seconds. Patient was given a blood transfusion of 500 cubic centimeters by direct method. On April 7 the blood picture was improving and an improvement was noted in the patient. His temperature had ranged from 100 degrees to 102° F. and the pulse rate was from 100 to 120. It was now noted that his tongue was swollen and a more marked bloody stomatitis had appeared which involved the mucosa of the pharynx and the floor of the mouth. The next day, April 8, bleeding had stopped and the patient was given a blood transfusion of 600 cubic centimeters by direct method. From time of first admission to the hospital the patient had been given 6 intravenous injections of sodium thiosulphate, 1 each day; thromboplastin intravenously as indicated; liver extract, daily, intramuscular and by mouth; Blaud's pills; and cod-liver oil wafers. On April 22 the patient was considered to be out of danger. No bleeding from the gums was noted and no purpuric spots were present. The angina condition of the mouth had disappeared. Laboratory reports this date were as follows: Red blood count 2,550,000; white blood count 6,950; hemoglobin 58 percent; platelets 52,600; reticulocytes 3.8 percent; myelocytes 0; segmented 63 percent lymphocytes 28 percent; bands 5 percent; and monocytes 3 percent. After April 25 the patient was up and about the ward and he had slowly gained in weight and strength. No residual symptoms other than general weakness and easy fatigability were noted objectively or subjectively. Improvement continued until discharged to duty. Physical examination and blood pressure were within normal limits. Subsequent laboratory findings were as follows: April 29, red blood count 3,550,000; white blood count 6,000; hemoglobin 80 percent; platelets 98,670; reticulocytes 1.9 percent; polymorphonuclears 70 percent; and lymphocytes 30 percent. May 5, red blood count 3,980,000; white blood count 8,300; hemoglobin 80 percent; platelets 185,040; reticulocytes 1.89 percent; polymorphonuclears 65 percent. May 23, red blood count 4,160,000; white blood count, 6,550; hemoglobin 85 percent; platelets 373,440; reticulocytes 1.1 percent; polymorphonuclears 65 percent. June 13, red blood count 4,800,000; white blood count 7,750; hemoglobin 95 percent; platelets 322,000; reticulocytes 0.5 percent; polymorphonuclears 60 percent.

On June 20 the patient was considered well and was discharged to duty after 83 days on the sick list.

Agranulocytosis.—(70-1932.) A patient was infected at New York, N.Y. He had a history of penile sores in April 1932, and a diagnosis of syphilis was later confirmed by a positive serum Kahn which was reported as 4 plus, 80 units. The patient received a 0.3 gram intravenous injection of neoarsphenamine on November 3, 1932, a 0.45 gram intravenous injection on November 10, and 0.6 gram intravenous injections on November 17, 22, and 29. The total dosage was 2.55 grams of neoarsphenamine administered over a period of 27 days, or an average of 94 milligrams per day. The dilution of the last injection was 0.6 gram of neoarsphenamine in 20 cubic centimeters of sterile freshly distilled water, and the rate of injection was 3 to 5 minutes for the 0.6 gram dose administered. As concurrent treatment the patient was given 1 cubic centimeter intramuscular injections of bismuth salicylate on November 3, 10, 17, and 24.

One hour after the last injection of neoarsphenamine the patient complained of a mild headache. Twelve hours after the injection he had chilliness, malaise, and a rise in temperature. Twenty-four hours after the injection the symptoms persisted and the patient had a temperature of 102° F., pulse 90, and respiration 20. Laboratory findings reported the urine as 4 plus for albumin. The patient was administered antipyretics and cathartics. Sodium thiosulphate was not administered. On December 2 the patient was transferred to a naval hospital, at which time he had a high fever of 104° F., tachycardia (110–120), numerous shallow ulcerations in the mouth and palate, and showed a loss of 25 pounds in weight. From these ulcerations the spirilla and the fusiform bacilli of Vincent's angina were recovered. The patient was placed on expectant treatment and gargle of 0.25 percent solution of chromic acid. His temperature improved immediately and in 3 days reached normal and remained so thereafter. During the convalescent period his appetite returned and he regained his lost weight. A study of the blood picture was interesting in that it indicated a severe involvement of the granulocytic forming cells of the bone marrow without affecting the platelets, erythrocytic or lymphocytic cells. Spontaneous recovery occurred without any of the recent so-called "granulopoietic measures", as transfusion; spray, or nucleotide. It was believed that the patient's granulopoietic cells of the bone marrow were sensitive to benzol-containing compounds, and that no such compound (arsphenamine, neoarsphenamine, sulpharsphenamine, etc.) should be used in the further treatment of his luetic condition. One gram of sodium thiosulphate in 10 cubic centimeters of water was administered intravenously on December 2 and 3.

On December 15, 16 days after the first symptoms of reaction, the patient was considered to have fully recovered and was discharged to duty.

Herxheimer reactions.—It is difficult to determine a line of demarcation between a certain type of vasomotor-phenomena reaction and a Herxheimer reaction. However, two reactions occurred in 1932 that appear to be of the Jarisch-Herxheimer type and are included under this heading. These reactions were of a mild type. Case histories are as follows:

(71-1932.) A patient who was infected at San Francisco, Calif., on August 3, 1932, was given a diagnosis of syphilis because of the appearance of a lesion on his penis on September 28. On October 3 he had a generalized adenopathy, maculo-papular rash, and mucous patches. On October 4 the diagnosis was verified by a 4 plus Kahn blood test. He was given a 0.3 gram intravenous injection of neoarsphenamine on October 5 and 0.45 gram intravenous injections on October 12 and 19. The total dosage administered was 1.2 grams. The dilution of the last injection of neoarsphenamine was 0.45 gram of neoarsphenamine dissolved in 10 cubic centimeters of water and the rate of injection was about 1 minute for the 0.45 gram dose administered.

Two and one half hours after the last injection of neoarsphenamine the patient had a temperature of 102.8° F., severe headache, general malaise, and a generalized pink macular rash. The Dickens' test was negative although the total 6-hour specimen was not available. One gram of sodium thiosulphate was administered intravenously about half an hour after the onset of symptoms of reaction. The following morning, October 20, the patient was much improved. On October 22 he was discharged to duty as completely recovered after 3 days on the sick list.

(72-1932.) A patient was exposed in August 1932, and a lesion developed on his penis on September 1. The darkfield examinations of the serum from the lesion were negative for *Treponema pallidum.* A diagnosis of syphilis was made and the diagnosis was confirmed by 4 plus Kahn blood tests on the following dates: September 28, October 5, 11, and 26. On October 8 the patient was started on his first course of arsenical treatment and was given a 0.3 gram intravenous injection of neoarsphenamine on that date. Following this injection a slight generalized macular rash developed over his body which disappeared before the second injection of neoarsphenamine was administered. On October 12 the patient received his second intravenous injection, which consisted of 0.45 gram of neoarsphenamine. The dilution of this injection was 0.45 gram of neoarsphenamine diluted to 10 cubic centimeters with triple distilled water. The rate of injection was about 2 to 3 minutes, for the 0.45 gram dose administered. As concurrent treatment 1 cubic centimeter intramuscular injections of bismosol were given on October 11 and 14.

Three days after the last injection of neoarsphenamine, that given on October 12, the patient complained of general malaise, fever,

and a skin rash. Examination revealed a temperature of 101° F., pulse 80, and respiration 20. A general examination was negative, except a moderately inflamed pharynx and slight generalized lymphadenopathy. The skin showed a generalized rash consisting of macules and fine papules with some desquamation. No lesions of the mucous membranes were noticed. Laboratory findings were reported as follows: Urinalysis—albumin, negative; sugar, negative; microscopic, no blood cells or pus cells present. Blood—red blood count 4,000,450; white blood count, 6,800; differential, polymorphonuclears 83 percent, lymphocytes 16 percent, and eosinophiles 1 percent. Dickens' tests: specimen 1—trace; specimen 2—faint trace. One gram intravenous injections of sodium thiosulphate were given daily for 8 days. Calamine lotion was applied to the lesions.

On October 24 the patient was considered recovered and was discharged to duty after 9 days on the sick list.

Reactions of minor importance.—During the year 1932 no reactions were reported which could be included under this heading.

HEALTH OF THE NAVY

Statistical returns for the first quarter of 1933 gave a low general admission rate, 354 per 1,000 per annum, as compared with 465, the rate for the corresponding months of 1932. The median rate for the quarter for the preceding 5 years is 419.

The admission rate from disease was 319 per 1,000 per annum as compared with 378, the 5-year median for the corresponding months.

The admission rate from injuries was 35 per 1,000 per annum. The median, or expected rate, for the corresponding quarter of the preceding 5 years is 37.

Common infections of the respiratory type were prevalent at most of the shore stations in the United States during the winter months. As usual, acute catarrhal fever predominated. In January, 518 cases of this disease were notified by forces ashore in the United States; in February, 218 cases; and in March, 112 cases, or a total of 848 cases for the quarter. Fewer cases of this disease occurred in March than has been the experience for the past 5 years. The expected number of cases for this month is 443.

The United States Naval Training Station, San Diego, reported that very good health conditions existed during the quarter. In January, 26 cases of acute catarrhal fever were admitted; in February, 44; and in March, 20. One fatal case of cerebrospinal fever developed at this station in January. The patient, 18 years of age, was transferred to the hospital, San Diego, Calif., and died a few hours after the first symptoms of the disease appeared.

The United States Naval Training Station, Newport, R.I., reported a small outbreak of acute catarrhal fever and influenza, characterized by mild symptoms and averaging about 4 sick days for each case. In January, there were 69 cases of acute catarrhal fever and 43 cases of influenza reported; in February, 23 cases of catarrhal fever and 1 of influenza; and in March, 14 cases of catarrhal fever and no case of influenza.

The United States Naval Training Station, Norfolk, Va., states that the health of the station personnel for the quarter has been very good. In January, 30 cases of acute catarrhal fever occurred as a continuation of the epidemic which existed in November and December of last year. These cases were devoid of serious complications and sequelae. All but one were treated in the sick quarters at the training station. Fourteen cases of German measles were notified by this station in February and 35 in March. They were very mild in type and treated directly on the station.

Reports from forces afloat indicate that morbidity rates for disease and accidents and injuries were a little less than expectancy. The admission rate for the quarter was 326 per 1,000 per annum as compared with the corresponding median for the preceding 5 years, 390.

A total of 984 cases of acute catarrhal fever was notified from all ships for the quarter but as the cases have a considerable time and place distribution, the number is not excessive.

Only 12 ships had 20 or more cases. The U.S.S. *Hannibal* reported the greatest number, 43 in January, 34 in February, and 1 in March. Fifty-eight cases of influenza were notified for the quarter, segregated as follows: 44 in January, of which 18 were on board the U.S.S. *Concord;* 7 in February; and 7 in March. These cases were divided among 11 ships. Six cases of scarlet fever were reported in the following distribution: U.S.S. *Tennessee* and U.S.S. *West Virginia,* 1 each in January; U.S.S. *Pennsylvania* and U.S.S. *Barracuda,* 1 each in February; and U.S.S. *Lexington* and U.S.S. *Rochester,* 1 each in March. There were 2 cases of diphtheria notified—one in February from the U.S.S. *Luzon,* Asiatic Station, and 1 in March from the U.S.S. *Lexington.*

One fatal case of typhoid fever was reported by the U.S.S. *Mindanao* in January. The patient was attached to the U.S.S. *Fulton* when he was hospitalized in Canton, China, with a diagnosis of malaria. On departure of this ship from port, the health records were transferred to the U.S.S. *Mindanao* as the patient had developed typhoid fever while under treatment in the hospital. Death occurred five days from the onset of the disease. His health record indicates that he received three injections of typhoid prophylaxis three years and seven months prior to infection.

TABLE 1.—*Summary of morbidity in the United States Navy for the quarter ended Mar. 31, 1933*

	Forces afloat	Forces ashore	Entire Navy
Average strength	70,411	38,710	109,121
All causes:			
Number of admissions	5,745	3,919	9,664
Annual rate per 1,000	326.37	404.96	354.25
Disease only:			
Number of admissions	5,132	3,568	8,700
Annual rate per 1,000	291.55	368.69	318.91
Communicable diseases, exclusive of venereal diseases:			
Number of admissions	1,633	1,855	3,488
Annual rate per 1,000	92.77	191.68	127.86
Venereal diseases:			
Number of admissions	2,041	482	2,523
Annual rate per 1,000	115.95	49.81	92.48
Injuries:			
Number of admissions	608	344	952
Annual rate per 1,000	34.54	35.56	34.90
Poisonings:			
Number of admissions	5	7	12
Annual rate per 1,000	.29	.72	.44

TABLE 2.—*Deaths reported, entire Navy, during the quarter ended Mar. 31, 1933*

		Navy			Marine Corps		Nurse Corps	Total
		Officers	Midshipmen	Men	Officers	Men		
Average strength		9,344	1,738	81,288	1,179	15,080	492	109,121
CAUSE—DISEASE								
Primary	Secondary or contributory							
Abscess, brain	None			1				1
Abscess, peritonsillar	Septicemia			1		1		2
Appendicitis, acute	Hemorrhage, intra-abdominal.			2				2
Appendicitis, acute	Septicemia			1				1
Appendicitis, acute	Pneumonia, lobar					1		1
Carcinoma:								
Breast	None						1	1
Cecum	None			1				1
Colon	Pneumonia, lobar	1						1
Stomach	None	1						1
Do	Gangrene, right foot	1						1
Angina, pectoris	Myocarditis, chronic	1						1
Cerebrospinal fever	None			1				1
Diverticulitis (Meckel's)	Obstruction, intestinal, from external causes.					1		1
Embolism, coronary artery	None			1				1
Endocarditis, acute	None					1		1
Gallstones	Abscess, lung	1						1
Hemorrhage, subarachnoid	None					1		1
Influenza	Pneumonia, broncho					1		1
Do	Pneumonia, lobar			1				1
Nephritis, acute	Uremia	1						1
Nephritis, chronic	Arteriosclerosis, general					1		1
Pneumonia, broncho	None					1		1
Pneumonia, lobar	None	1		5		3		9
Syphilis	Poisoning, acute, neoarsphenamine.			2				2
Thrombosis, coronary artery	None			1		1		2
Thrombosis, coronary artery.	Arteriosclerosis, general	1		1				2
Tuberculosis, chronic, pulmonary.	Tuberculosis, larynx			1				1
Tuberculosis, chronic, pulmonary.	Tuberculosis, meninges			1				1

TABLE 2.—*Deaths reported, entire Navy, during the quarter ended Mar. 31, 1933*—Continued.

		Navy			Marine Corps			
		Officers	Midshipmen	Men	Officers	Men	Nurse Corps	Total
Average strength		9,344	1,738	81,288	1,179	15,080	492	109,121
CAUSE—DISEASE								
Primary	Secondary or contributory							
Tuberculosis, chronic, pulmonary.	Tuberculosis, peritoneum					1		1
Tuberculosis, general miliary	None			1				1
Typhoid fever	do			1				1
Total for diseases		8		22		13	1	44
CAUSE—INJURIES AND POISONINGS								
Contusion, heart	Dilatation, cardiac, acute			1				1
Drowning	None	1		5		1		7
Do	Psychosis, unclassified					1		1
Fracture:								
Compound, skull	None			2		2		4
Do	Intracranial injury	1		4				5
Do	Hemorrhage, traumatic, intracranial.			1				1
Do	Septicemia	1						1
Simple, pelvis	Hemorrhage, traumatic, intra-abdominal.			1				1
Simple, skull	None			1				1
Do	Hemorrhage, traumatic, intracranial.			1				1
Do	Intracranial injury			1		1		2
Do	Rupture, traumatic, liver			1				1
Injuries, multiple, extreme	None	1						1
Injuries, multiple, extreme	Intracranial injury			1				1
Strangulation, neck	None	1		1				2
Wound, gunshot, head	None	1						1
Poisoning:								
Barbital	Pneumonia, broncho-			1				1
Cresol	None					1		1
Gas, acetylene	None					1		1
Hydrocyanic acid	None			1				1
Potassium cyanide	Psychoneurosis, unclassified.			1				1
Potassium cyanide	None			1				1
Sodium borate and barium carbonate.	None			1				1
Total for injuries and poisonings		6		25		7		38
Grand total		14		47		20	1	82
Annual death rate per 1,000:								
All causes		6.00		2.31		5.31	8.13	3.01
Disease only		3.42		1.08		3.45	8.13	1.61
Drowning		.43		.25		.27		.26
Other injuries		2.14		.74		1.06		.88
Poisoning				.25		.54		.27

ADMISSIONS FOR INJURIES AND POISONINGS, FIRST QUARTER, 1933

The following table, indicating the frequency of occurrence of accidental injuries and poisonings in the Navy during the first quarter, 1933, is based upon all form F cards covering admission in those months which have reached the Bureau:

	Admissions January, February, and March 1933	Admission rate per 100,000 per annum	Admission rate per 100,000 year, 1932
INJURIES			
Connected with work or drill	408	1,495	2,440
Occurring within command but not associated with work	297	1,089	1,675
Incurred on leave or liberty or while absent without leave	250	916	1,900
All injuries	952	3,490	6,015
POISONINGS			
Industrial poisonings	3	11	8
Occurring within command but not connected with work	7	26	68
Associated with leave, liberty, or absence without leave	2	7	17
Poisonings, all forms	12	44	93
Total injuries and poisonings	964	3,534	6,108

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.	
	January, February, and March 1933	Year 1932	January, February, and March 1933	Year 1932	January, February, and March 1933	Year 1932
Percent of all injuries	42.5	40.6	31.2	27.8	26.3	31.6
Percent of poisonings	25.0	8.7	58.3	72.8	16.7	18.5
Percent of total admissions, injury and poisoning titles	42.3	40.1	31.6	28.5	26.1	31.4

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 1933 worthy of notice from the standpoint of accident prevention.

STATISTICS RELATIVE TO MENTAL AND PHYSICAL QUALIFICATIONS OF RECRUITS

The following table was constructed with figures taken from monthly reports submitted by boards of medical survey at naval training stations:

	United States Naval Training Station			
	Hampton Roads, Va.	Great Lakes, Ill.	San Diego, Calif.	Newport, R.I.
Recruits received during the period	240	285	294	198
Recruits appearing before board of medical survey	13	1	1	
Recruits recommended for discharge from the service	13	1	1	
Recruits discharged by reason of medical survey	10	0	0	0
Recruits held over pending further observation	0	0	0	2
Recruits transferred to the hospital for treatment, operation, or further observation for conditions existing prior to enlistment	3	1	33	10

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. The time which elapsed from date of enlistment to date of medical survey is noted in each case. With certain diseases, survey followed enlistment so rapidly that it would seem that many might have been eliminated in the recruiting office. The difficulty in establishing a diagnosis in nervous and mental cases is demonstrated by the time interval in the table. An exception in this group is epilepsy which may or may not diagnose itself promptly. Certain groups, of course, present difficulties in diagnosis at the time of enlistment due to lack of equipment.

Cause of survey	Number of surveys	Number of days between enlistment and survey
Absence, acquired, teeth	1	8
Bronchitis, chronic	1	157
Caries, teeth	1	7
	1	5
Color blindness	1	55
Constitutional psychopathic inferiority, without psychosis	1	143
Constitutional psychopathic state, emotional instability	1	225
	1	28
Constitutional psychopathic state, inadequate personality	1	57
Deafness, unilateral, left ear	1	157
Dementia præcox	1	80
Enuresis	1	20
Epilepsy	1	189
	1	127
	1	118
Flat foot (both feet)	1	202
Gonococcus infection, urethra	2	[1] 7
	1	5
	1	3
Icthyosis	1	160
Intracranial injury	1	27
Malocclusion, teeth	1	6
Syphilis	1	24
	1	23
	3	[1] 17
	1	15
	1	14

[1] Each.

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