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

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Washington D.C.

VOL. XXXIII JANUARY 1935 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
COMMANDER LOUIS H. RODDIS
MEDICAL CORPS, U.S. NAVY, IN CHARGE

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NAVY DEPARTMENT,
Washington, March 20, 1907.

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

TRUMAN H. NEWBERRY,
Acting Secretary.

Owing to the exhaustion of certain numbers of the BULLETIN and the frequent demands from libraries, etc., for copies to complete their files, the return of any of the following issues will be greatly appreciated.

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PREFACE

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

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

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

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

The Bureau does not 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. XXXIII JANUARY 1935 No. 1

SPECIAL ARTICLES

MOTOR-VEHICLE DAMAGE TO MEN OF THE NAVY

By LUCIUS W. JOHNSON, Captain, Medical Corps, United States Navy

For several years past the number of Navy men injured and killed in motor-vehicle accidents has shown a steady increase. A constantly growing proportion of all those killed or invalided from the service has come from this source. It has supplanted drowning which, for many years, stood at the top of the list of causes of death. This has long been the subject of official notice and many regulatory devices have been adopted. Yet the cost to the Government and the wastage of carefully selected manhood continues.

TABLE 1.—*Motor-vehicle injuries—whole Navy*

Calendar year	Admissions	Sick days	Invalided from service		Deaths		Percent injured while on leave or liberty
			Number	Percent of all I. S.	Number	Percent of all D. D.	
1924	598	19,972	32	1.43	23	4.62	68.40
1925	677	22,766	27	1.65	23	5.54	70.40
1926	757	25,171	36	1.6	31	8.98	76.66
1927	719	24,348	28	1.12	32	4.75	87
1928	759	26,037	24	1.13	52	10.70	85
1929	978	31,921	32	1.56	48	12.18	83
1930	1,138	41,631	54	2.54	58	14.9	92.95
1931	1,216	43,297	69	3.97	81	20.71	86
1932	1,149	41.362	39	3.09	70	18.59	89.55

This study is an attempt to analyze the cases of the patients admitted to our largest naval hospital for motor-vehicle injuries during the 6-month period from May 1 to October 31, 1933. It is hoped that facts will be disclosed that will help to indicate what administrative measures should be taken to reduce the loss from this cause.

In April 1933 two striking examples were observed in a single day. The first was a recruit at the local training station who was involved in a motorcycle accident while on liberty and suffered a fractured skull, for which he was under treatment 128 days. Twelve days after returning to duty he suffered a similar accident and was returned

to the hospital, once more with a fractured skull, for which he was eventually invalided from the service. Here was a man who had rendered no service to the Government but had cost many hundreds of dollars and will probably be pensioned for the rest of his life. The second was a fireman, second class, who, within the space of 10 months, had been three times admitted to the hospital, once for automobile and twice for motorcycle injuries, with a total of 103 sick days. Another poor investment for Uncle Sam.

My interest was sufficiently aroused so that I started to collect data. Forms for reporting motor-vehicle injuries were mimeographed and distributed to the wards. To those ward officers who so faithfully did the work of recording the cases, I desire to express my thanks and appreciation. All enlisted patients who were admitted to the Naval Hospital, San Diego, for such injuries during the 6-month period from May 1 to October 31, were recorded. They numbered 121. Six men who were instantly killed in this vicinity during that period are not included, since their bodies went directly to the undertaker instead of coming to the hospital. But their cases should be considered in estimating the total damage in this neighborhood. During the same period three officers were admitted for motor-vehicle injuries. They are not included in this group, but will be separately discussed.

The United States Bureau of the Census compiles statistics of mortality from automobile accidents by 4-week periods from 86 cities of the United States. A curve is plotted showing the death rate per 100,000 of estimated population. This curve is quite constant, from year to year, in showing a low rate from February to July, when it begins to rise and reaches its peak in the period from November to January. The time covered by this present study covers a portion of the low curve and of its ascending limb. But climatic conditions are different in California and there is less seasonal variation in the use of cars and the mileage driven. The statistics compiled by the California Highway Patrol for 1932 show that 49.1 percent of the reported motor-vehicle accidents, 41.1 percent of the injuries, and 51.4 percent of deaths due to such accidents occurred within the period, May to October, inclusive. These percentages indicate that our figures may be considered as being, roughly, one-half of a year's damage.

It must be remembered that this report covers only the patients admitted to the hospital. It does not include those who were slightly injured and able to return to their ships or homes. Nor does it include those instantly killed. The total injuries can only be conjectured.

Several times in the last few years San Diego has held the unenviable distinction of having the highest death rate of all the reporting cities. Just how much the presence of a large Navy personnel

has to do with this high death rate nobody can state with precision. I have been able to get very little help from police records, the State traffic bureau, health department, or the local automobile club in determining this point. Enlisted men are accustomed to wear civilian clothes while on liberty and to conceal their service connection, if possible, when involved in traffic accidents, say the police. From the newspaper accounts one gets the impression that there are few serious accidents in which Navy men are not, in some way, involved.

Official figures, furnished by the California Department of Motor Vehicles, for the first 6 months of 1934 indicate that the men of the Navy are not such an important factor in automobile accidents as many have supposed.

Reportable motor-vehicle accidents for the period, January 1 to June 30, 1934, show an increase of 532, or 3.6 percent, over the first half of 1933. There were 195 more persons killed in traffic crashes this year than last, an increase of 18.8 percent. Thus the increase goes on in spite of the absence of the fleet from the Pacific area for 3 of the 6 months under consideration.

Drunkenness as a factor has also increased during the absence of the fleet. The combined figures on intoxicated drivers and pedestrians involved in motor-vehicle accidents show for the first 6 months of 1933 a total of 1,653 cases; in 1934 there were 2,113 cases, an increase of 27.8 percent. The 184 fatal accidents which involved liquor show an increase of 47 over the previous year.

Analysis of our cases shows the following facts.

Age.—Table 2 shows the distribution by age groups of all our cases and of drivers only, compared with the California Highway Patrol figures for drivers involved in reported accidents during 1932.

TABLE 2

Age	All cases		Drivers only		State of California [1]
	Number	Percent of total	Number	Percent of total	
20 or under	11	9.09	6	11.76	7.61
21 to 30	83	68.59	35	68.62	26.22
31 to 40	25	20.66	10	19.61	21.22
41 or over	2	1.66			25.22

[1] Does not total 100 percent because of 9,978 accidents in which age of driver was not stated.

The average age of all our patients was 24.69, of drivers only, 24.78. It will be noted that two-thirds of all our cases were in the age group of 21 to 30, closely paralleling the age distribution of the enlisted personnel of the Navy. The California figures show a contrasting distribution which is nearly equal in the three higher age groups.

Table 3 shows the age grouping of automobile passengers and drivers, motorcycle passengers and drivers, and also pedestrians.

TABLE 3

Age	Automobiles			Motorcycles			Pedestrians	Total
	Drivers	Passengers	Total	Drivers	Passengers	Total		
20 or under	----	3	3	6	1	7	1	11
21 to 30	22	42	64	13	5	18	6	88
31 to 40	10	8	18	----	----	----	3	21
41 or over	----	1	1	----	----	----	----	1
Total	32	54	86	19	6	25	10	121

The ages of those injured in automobiles closely parallel those shown in table 2, while the ages of those injured on motorcycles indicate the predominance of youth among the followers of this sport.

The average age of automobile drivers was 27.1. More than half were 26 or over and none was under 21. The average age of motorcycle drivers was 22. One-half of them were 21 or under and one-third were 20 or under, showing again the distinct prevalence of youth among riders of motorcycles. The average age of pedestrians was 29.5.

Rates.—Table 4 shows the distribution according to rates held by those injured.

TABLE 4

	A. S.	S. 2c	S. 1c	F. 3c	F. 2c	F. 1c	Pvt.	Cpl.	Sgt.	P. O. 3c	P. O. 2c	P. O. 1c	C. P. O.	Total
Auto drivers	----	1	6	----	1	3	1	----	2	2	9	5	2	32
Auto passengers	1	3	12	2	6	3	5	1	----	5	10	5	1	54
Motorcycle drivers	----	2	4	3	4	4	----	----	----	1	1	----	----	19
Motorcycle passengers	----	1	2	----	2	----	----	----	----	1	----	----	----	6
Pedestrians	----	----	3	1	----	----	----	----	----	1	3	1	1	10

A considerable preponderance of rated men among the automobile drivers will be noted. Many of them are men of several years' service who own their own cars and drive to and from their duty. Several of those in the lower rates were driving rented cars. Automobile passengers show a general distribution through all rates. Some were passengers in taxis, some were hitch hikers, and others riding with friends or shipmates. Several of those listed as passengers were probably drivers but declared themselves as passengers to avoid responsibility for the accident and possible punishment for reckless driving while drunk. Motorcycle riders are practically limited to the lower rates. No marines were found in this group. No reason can be given for the predominance of seamen first-class and petty officers second-class among the pedestrians.

Length of service.—The average length of service agrees, in a general way, with the figures for ages and rates. Automobile drivers averaged

4 years 3 months of service and their passengers 6 years 6 months. Motorcycle drivers averaged 3 years 7 months and their passengers 4 years. Pedestrians averaged 8 years 4 months of service. The higher ages and rates of pedestrians indicate that the older men are either more careless or less agile.

Duty status.—Only 2 of our 121 patients were injured while performing duty. A seaman second-class, driving an official car, had a collision at a street intersection. He sustained contusions about the knees which caused 17 sick days. A chief petty officer on patrol duty was answering an official call when his car collided with another at an intersection. He was in the hospital 7 days for lacerations of the scalp. The last column of table 1 shows the proportion of motor-vehicle injuries in the whole Navy which were incurred while on leave or liberty.

One of the most striking facts of this whole subject is the contrast between the slight damage done while on duty and the enormous amount done while on leave or liberty. The Government pays the huge bill but gets no return in service. Reference to table 5 and the discussion on cost to the Government will emphasize this point.

This feature, injury while absent from station and duty, would appear to offer the most promising field for restrictive regulation, yet it is beset with difficulties. Those injured cannot be deprived of the benefits of hospitalization and none of us would wish to do so. We have accepted this responsibility and could not relinquish it if we would. Hospitalization costs are more than half the total and these injury cases are the most costly to treat. Deprivation of pay, for men involved in accidents while absent from duty, would probably have some effect on those with family responsibilities because of the probability that their dependents would become objects of public charity if their pay were stopped because of injury.

But the service man is by no means always at fault in these accidents. Many of them are perfectly innocent victims of civilian drivers who are drunk, reckless, or careless. It would be most unjust to punish the man who was not at fault, by stopping his pay. To separate the sheep from the goats by stopping the pay of those who are blameworthy offers serious obstacles. A visit to the traffic court of any large city will convince you of this. The inherent difficulty of securing clear honest statements of what occurred from participants or spectators of an accident is common knowledge. Very seldom can the clear-cut fault of one party and the complete innocence of the other be established. So most of us who would have such decisions to make would give the injured man the benefit of the doubt. The more closely we examine this subject, the more obscure it becomes.

Cost to the Government.—An accurate estimate of this would be beyond my resources. I have attempted to approximate it by taking the base pay of the man's rate for the number of days he remained

in the hospital and adding to it $3.483 for each day, that being the per diem cost of this hospital for the last fiscal year. These figures represent the minimum cost, an indeterminate fraction of the total. This method is, of course, full of inaccuracies. It takes no note of increases in pay for longevity, sharpshooter, or expert, commuted rations, aviation pay, or other special allowances. In cases invalided from the service it does not include the cost of hospitalization for an indefinite period after separation from the service or the lifelong pension that may follow. Also, it includes no costs beyond December 31, 1933, although nearly 20 percent of all the cases will be still in the hospital at that time. They will remain there indefinitely and many of them will be surveyed from the service and pensioned. The exact cost is probably within the realm of the unknowable but an approximation can be made which will be of some value.

TABLE 5

	On duty	Percent	Leave or liberty	All cases
Sick days	24	0.0041	5,737	5,761
Average sick days per case	12		48	47.61
Cost	$133.39	.0042	$31,301.39	$31,434.78
Average cost per case	66.69		263.03	259.79

Table 5 shows the cost for those injured on duty, those injured on leave or liberty, and the totals, also the sick days for each group. The contrast cannot fail to be impressive. What relation do the figures from this one hospital bear to those of the whole service? Or those of all Government services? That would be an interesting study.

The absurdity of the relation between the two groups is increased by the fact that all the duty cases have been discharged from the hospital and are closed while many of the other group will remain for weeks or months, with constantly increasing costs which cannot be estimated.

The essence of the whole problem lies in control of the pastimes of the men while they are on leave or liberty. No priority is claimed for that idea. It has long been recognized by those in authority, but it is given added force by the figures presented here.

TABLE 6

	Number	Sick days		Cost		Disposition		
		Total	Case average	Total	Case average	D.	I. S.	D. D.
Auto drivers	32	1,554	48.56	$8,578.18	$268.06	29	3	0
Auto passengers	54	2,120	39.25	11,745.43	217.67	49	3	2
Motorcycle drivers	19	1,324	74.27	6,750.45	355.28	16	2	1
Motorcycle passengers	6	378	63	2,046.17	341.03	6		
Pedestrians	10	385	38.5	2,505.55	250.56	9		1
Total	121	5,761	47.61	31,434.78	259.79	109	8	4

Type of vehicle.—There is a general impression that the driver of an automobile is less liable to suffer serious injury than his passengers; also that motorcycle injuries average more serious than those sustained in automobiles. Table 6 throws some light on these points. The average automobile driver stayed about one-fifth longer time in the hospital and cost the Government about $50 more than the average automobile passenger. On the other hand, two passengers died but no drivers. Motorcycle riders spent more than 50 percent more time in the hospital than did the automobile riders. One bizarre fact was brought out, that motorcycle drivers injure their right legs 5 times out of 8, while their passengers injure the left leg twice as often as the right. Experienced drivers say that is because most accidents happen while passing an automobile and the driver's right leg is crushed between the car and the clutch of his cycle. When the brake is applied suddenly the rear wheel skids to the right and the cycle falls to the left, catching the left leg of the passenger.

Approximately one-fifth of our patients were injured on motorcycles. They were mostly youngsters of short service and little sense of responsibility. On one liberty day two patients who were in the hospital because of motorcycle injuries went out on their machines and both crashed again, receiving more serious injuries than before. The commonest motorcycle injury is compound fracture of the leg. This accounts for the prolonged stay in the hospital of motorcycle riders.

An important fallacy lies in the number of passengers and drivers. Undoubtedly, some of those classed as passengers were really drivers. Both civil laws and fleet orders penalize drunken or reckless driving and make it undesirable to be caught doing so. Even though newspaper reports and police records show the patient to be the driver, by the time he is able to make a statement he has become an innocent passenger. The police, many of whom are ex-service men, are always willing to concede that they might be wrong about who was the driver. A passenger, no matter how drunk he may be, is always a guiltless victim and the misconduct rule does not apply. So there is strong inducement for the driver to make a misleading statement and such statements, unless supported by other evidence, are to be taken with a grain of salt.

Alcohol and misconduct.—California Highway Patrol statistics for 1932 show that, in accidents where the driver was at fault, 31.7 percent had been drinking. Of pedestrians injured, 11.5 percent had been drinking. In our series, 12 automobile drivers and 2 pedestrians were stated to have been drinking. None admitted taking anything stronger than 3.2 beer.

There are numerous fallacies connected with any figures concerning alcohol as a factor in accidents. When the patient is obviously

drunk, the decision is simple. When he has had a little alcohol, not enough to affect his station or speech, but sufficient to produce slight changes in muscular tension, mental acuteness or reaction time, it is a more difficult problem. Also, an accident has an immediate sobering effect. When conditions are such that a blood alcohol estimation can be made, it gives a firm basis for decision. In many cases hours elapse after the accident before the patient reaches the hospital and there is no evidence to controvert his statement that he was perfectly sober. When a man is killed or very seriously injured, there may be nothing to show whether he was drunk or sober, and he is given the benefit of the doubt.

In driving, one must make many split second decisions, most of which become automatic after one has driven for some time. Estimates of speed and direction of other cars approaching an intersection, for example, soon come to be made without conscious effort. Hundreds of such estimates and actions based on them are made in the course of a day's driving. If these estimates are accurate and the actions correct, there is relative safety. Otherwise there is trouble.

A small amount of alcohol will affect the rate of cerebration, the muscular tension and the reaction time; also the judgment, attention, and sense of responsibility. Coordination of all these factors is essential for safe driving. Any dissidence among them will produce faulty timing or altered degree of muscular response and so make accidents more likely. Alcoholic exhilaration and the suppression of the inhibitions also favor excessive speed which, the police say, is responsible for 80 percent of accidents.

So it can be understood that alcohol may be a factor in accidents even when its presence is not manifest. For this reason, all statistics or statements on the importance of alcohol as a factor in motor-vehicle accidents are meaningless. A good guess is as likely to be accurate as the most carefully compiled estimate.

Disposition.—One hundred and nine of our cases were, or will be, returned to duty, 8 surveyed from the service, and 4 died. To the last group should be added the six others who were killed in the San Diego area during this period. The ultimate disposition of those who still remain on the sick list can be quite accurately predicted, although the length of their stay in the hospital cannot. The 8 who will be invalided from the service are still on the sick list, as are 16 who will probably be able to return to duty. No sick days or costs, after December 31, 1933, for those who remain in the hospital after that time are included in the tables.

Causes of accidents.—Patients injured in automobiles gave the following causes of the accidents: Collision with another car, 38; soft shoulder, 5; ran off road, 10; collision with street car, 1; driver asleep, 6; falls while entering or leaving car, 7; collision with fixed

objects, 8; cranking car, 2; fire under hood, 1. Injuries due to mechanical defects of the car were blow-outs, 6; wheel came off, 3; steering-gear trouble, 1; faulty brakes, 1; no lights, 1. Four collisions were blamed on fog, in spite of the literature issued by the local chamber of commerce which prates blithely about the fog-free climate of San Diego.

Patients injured on motorcycles gave the following causes: Collision with automobile, 19; with other motorcycles, 3; with fixed objects, 2. Those due to mechanical defects were 1 blow-out and 1 defective light. One case was blamed on fog.

All pedestrians were struck while crossing roads.

Traffic officers with whom I have talked say that fully 80 percent of accidents of the classes shown above are really due to excessive speed at intersections, in passing other cars, or rounding curves. Running off the road onto a soft shoulder, or a blow-out, will nearly always cause a car to overturn if it is traveling at high speed. Mechanical defects are quite likely to be present in rented cars and in those purchased for a small first payment. The seller or renter knows that the accident risk is very high and that, if an accident happens, the driver will simply abandon the car. So there is little incentive to keep the car in good mechanical condition.

It may be of interest to give a few incidents which illustrate the care-free attitude in which these youngsters incur their injuries.

A seaman, returning to his ship sober, saw a drunken shipmate about to cross the road in heavy traffic. He ran to give assistance and both were struck by an automobile. The drunken man had only slight injuries, while his sober helper received a compound fracture of the leg for which he was invalided from the service.

A man was returning to his ship, running because he was late. A shipmate, passing on a motorcycle, stopped and offered him a ride. At the next intersection they ran into an automobile. One, named East, lost his left leg. The other, named West, lost his right leg. Both were invalided from the service.

A man who had never driven an auto essayed to do so at 4 a. m. when he was full of 3.2 beer. He collided with another car and suffered a fractured skull.

A patient in the hospital with severe motor-vehicle injuries, on his first liberty took another patient for a ride on his motorcycle. When they were returned, by ambulance, one had a fractured pelvis and dislocated hip. The other had a fractured skull.

It all comes under the head of good clean fun. Fear of physical injury has little force in deterring healthy young men from reckless driving. This statement is supported by the fact that in a single week four patients in this hospital with motor-vehicle injuries went on liberty and crashed again, sustaining even more serious injuries.

While I was in the police station one day, a report was brought in of a youngster who had just been in a motorcycle crash and had been taken to the hospital with a fractured spine. In recording this accident it was discovered that it was his third collision within 10 days and during that same period he had been booked for two other traffic violations.

Whatever inhibitions the driver may have are quickly deadened by a little alcohol, which is never far to seek when the fleet is in. If a girl is along, that also stimulates recklessness, and a considerable number of girls were seriously injured in the accidents that sent our patients to the hospital.

Officers.—Three officers were admitted for motor-vehicle injuries during the period of this report. An ensign, aged 23, was a passenger in a car which ran off the road on a turn, because of excessive speed. Four teeth were knocked out and his right hand was badly cut. He was in the hospital 140 days and will return later for corrective operation on his hand. A lieutenant (j. g.), aged 31 went to sleep while driving a car. He was in the hospital for 5 days with minor injuries. A retired lieutenant commander, aged 51, while driving a car collided with another automobile at an intersection. He had several ribs fractured and was in the hospital 45 days.

The United Services Automobile Association figures show that officers have a much lower accident rate than the general public. They are much more careful than the average driver and injuries to them do not constitute a serious problem.

The facts so far given demonstrate the magnitude and importance of this drain on the man power of the Navy. The 41,362 sick days during 1932 mean the loss of 114 men for a year or 1,378 men for a month. A statement issued on December 9, 1933, by the chairman of the House Naval Affairs Committee says that the lack of men for the new ships is a most serious problem. Anything that can be done to reduce the number of noneffective men will be of value. If a man who obviously will be on the sick list for a long time could be immediately discharged from the service, a vacancy would be created which could be filled at once by a recruit, thus maintaining the full quota of effective men.

Efforts at control.—Bureau of Navigation Circular Letter No. 56–30 of September 3, 1930, quotes a circular letter issued by a force commander. This mentions the deaths and time lost by motor-vehicle injuries, also the many instances of indebtedness or insufficient financial support of families because of ill-advised purchase of automobiles. It is noted that naval personnel enjoy certain advantages, such as death benefits, free hospitalization, and freedom from attachment of pay to cover damages. These are privileges which should not be abused. Cars are not to be allowed to enter or leave ships,

stations, or bases without passes or insignia which are issued only after compliance with certain rules. These cover financial ability of the owner to keep the car in proper mechanical condition, proper licensing and ownership, property damage and liability insurance, character and reliability of the operator, agreement not to use the car for hire or to loan it to other enlisted men, and not to dispose of it without official permission. Similar regulations have, no doubt, been adopted at most shore activities.

Combatfor Letter P6–1/(9) of January 2, 1932, states that deaths and injuries to personnel of the battle force have increased alarmingly. The return of the fleet to the base means a jump of 70 to 80 percent in automobile accidents. Commanding officers are directed to impress on the personnel the necessity for observance of the laws and regulations governing the operation of motor vehicles.

I have heard from several sources that there is a fleet order requiring discharge from the service of men convicted for reckless driving or driving while drunk. I have been unable to secure a copy of the order or to discover that any men have been discharged for such offenses.

The circular letter given below seems to have accomplished definite results and it appears worthy of reproduction in full.

P13–1 05/Dy(0):

UNITED STATES FLEET,
DESTROYERS, BATTLE FORCE,
U. S. S. "MELLVILLE", FLAGSHIP,
San Diego, Calif., May 15, 1933.

DESTROYERS, BATTLE FORCE, CIRCULAR LETTER NO 4–33

From: Commander Destroyers, Battle Force.
To: Destroyers, Battle Force.
Subject: Reckless driving and driving while drunk.

1. The large number of deaths and nonfatal injuries to enlisted men involving hundreds of sick days per annum, resulting from motor vehicle accidents ashore, is a serious drain on naval efficiency both in a military sense and from an economic standpoint. The action taken by the civil courts in cases where naval men are convicted of subject offenses does not appear to have resulted in any substantial diminution of the number of such offenses.

2. The acts involved in subject offenses not only constitute offenses against the civil law but also constitute offenses against naval law. Both "driving while drunk" and "reckless driving" are considered to be "conduct to the prejudice of good order and discipline" (carelessly or negligently endangering the lives of others). In "driving while drunk" the additional naval offense of "drunkeness" is involved.

3. In view of paragraph 2 above it is directed that in every case of "reckless driving" or "driving while drunk" that is sustained by proper evidence, regardless of the fact that the offender may have been tried, convicted, and punished by the civil courts, the man concerned also be subjected to one of the following disciplinary measures:

(*a*) Trial by court martial or deck court.

91829—34——2

(*b*) Punishment by commanding officer at mast. If the latter action is considered sufficient, it is directed that the punishment awarded be sixty (60) days restriction to the ship. Such action, the authority for which is conveyed under the twenty-fourth article for the Government of the Navy, is deemed necessary as a military punishment to the offender, as an inhibitory warning to other personnel, and to remove, temporarily at least, from the highways a menace to the public safety. The latter considerations govern Commander Destroyers, Battle Force, in his decision radically to curtail the shore liberty of these offenders, a step which he views with disfavor in all cases except where the public safety is endangered or the good name of the Navy is disgraced.

4. In this connection it has come to the attention of commander destroyers, battle force, that the civil authorities sometimes change an original charge of "driving while drunk" to "reckless driving." The basis for such change appears to be the degree of insobriety evinced by the accused upon examination by the police surgeon. This latter circumstance should have no bearing upon the action taken by naval authority. A conviction of the offense of "drunkenness" can be established before a court martial provided there is sufficient evidence to show incapacity for the proper performance of duty due to impairment of the faculties through indulgence in intoxicating liquor.

5. Upon receiving a report that an enlisted man has been charged with "driving while drunk" or "reckless driving", the commanding officer shall, regardless of the civil-court action, cause a thorough and independent investigation to be made into all the facts of the case. To facilitate such investigation, commander destroyers, battle force, has directed the senior patrol officer to furnish the commanding officer, in addition to the regular patrol officer's report, with a copy of the police surgeon's report of the examination for the state of sobriety, when such examination has been held on an enlisted man.

6. Minor violations of traffic regulations shall not be included in the categories herein considered.

7. Attention is directed to the additional, but independent action, required by article D-9110 (1) (d), Bureau of Navigation Manual, that all cases of trial and conviction by civil authorities shall be reported to that Bureau.

E. C. KALBFUS.

The medical officer (Capt. W. L. Mann) of that force states that these measures have not only checked the progressive increase of traffic accidents but have served to reduce their number. Energetic treatment of the condition has improved it in this command, undoubtedly it would work equally well on a large scale if universally adopted. Knowledge that accidents would be promptly investigated and that drastic action would be taken against offenders would undoubtedly cause the men to be more careful.

Restrictions on cars entering or parking on naval property have had a good effect both on driving and care of cars. I am informed that in one city the dealers protested that an order prohibiting driving motorcycles on the station had the immediate effect of stopping all sales of motorcycles to enlisted men and glutted the second-hand market with motorcycles recently purchased.

It is difficult, if not impossible, to restrict ownership of cars driven by men outside the limits of naval stations. Here the traffic regulations are the only limitations. Cooperation of shore patrols with the

local traffic courts and a knowledge that the naval authorities favored immediate and severe punishment of traffic violations would surely have a salutary effect.

The Comptroller's decision not to pay the death gratuity for death due to recklessness and to make such deaths not in line of duty may have some deterrent effect on those mature enough to have some sense of responsibility. It apparently means nothing to the type of young man who furnishes most of our accidents.

Repeated admissions to the sick list for traffic accidents indicate that the individual is habitually careless or reckless or unlucky. In any case he is likely to return little service for what he costs. It would be a saving if such a man were discharged as soon as possible, either by survey or as undesirable.

Are private airplanes to be the next great menace to sailors? We have recently had two crashes involving Navy men piloting their own planes, which were described as unlicensed and uninspected. One man was killed and three injured. Antiquated planes can be bought for a small sum and, if quartered below the border or on someone's ranch, the troublesome Department of Commerce regulations can be dodged, at least for a time. Any action taken to reduce motor-vehicle accidents might well be made to include privately owned airplanes.

RECOMMENDATIONS

The problem having been outlined, suggestions for its correction would appear to be in order. These are submitted, not as a matured program, ready for adoption, but rather as ideas for discussion or marks for everybody to shoot at. If sufficient interest and discussion can be aroused, perhaps some good ideas may crystallize out of it.

(1) Broadcast to the service the magnitude of the problem and its effect on the man-power of the Navy. Make it known that the naval authorities are determined to act energetically to control this menace. Instruct all those in authority that every motor-vehicle accident and every instance of reckless or drunken driving must be investigated and the blame determined if possible. This to be made the subject of an official report, entered on the guilty one's record or made the basis of punitive action.

I am convinced that the moral effect of such action would be very great and that most of the battle would be won if service men believed that energetic action would be taken in every case. Close cooperation with civil authorities would be necessary in carrying out this program.

(2) Men to be discharged from the service as undesirable or court-martialed if a charge of reckless driving or driving while drunk can be supported, even though no one be injured.

(3) A modified interpretation of line of duty that would better protect the interests of the Government. A review of the decisions

on the subject will show many absurdities which are detrimental to Uncle Sam and his Treasury.

(4) Men seriously injured while drunk to be discharged from the service, whether drivers or not.

(5) Men so seriously injured as to be kept from duty for a month to be discharged from the service unless the injury was incurred while in the performance of official duties. Numbers 4 and 5 would not affect hospitalization but would stop the pay and make it possible to fill the vacancies created by such discharge with effective men.

(6) Exclude motorcycles from all ships and stations. Class as misconduct all injuries received while riding motorcycles, unless on official business.

(7) Extend to all naval establishments the regulations requiring that cars parked on the station have proper licenses, ownership certificates, efficient brakes and lights. Drivers to have the necessary licenses and carry insurance.

(8) Apply the same requirements to all cars purchasing gasoline at Navy filling stations. Here is a powerful lever that can be used to control both cars and drivers.

REPORT OF INJURIES ADMITTED TO THE HOSPITAL SHIP IN ONE YEAR

By Arthur H. Dearing, Lieutenant Commander, Medical Corps, United States Navy

The injuries sustained by naval personnel are a matter of interest to all medical officers. The treatment of such peace-time injuries afford us the best possible opportunity of training for care of war-time casualties. The nature of the wounds and injuries may vary but the broad principles of treatment and their application remain the same.

This report is presented to bring to the attention of the service the nature of the injuries sustained by fleet personnel, the place or manner in which the injury was suffered, something of the treatment, and an end-result study, showing final disposition, sick days, etc. Although this report covers the period from July 1, 1932 to July 1, 1933, during 2 months of this period the *Relief* was undergoing overhaul in a navy yard and received no patients. Therefore a period of 10 months only is covered. During this time the ship was in the San Pedro-Long Beach area and served as hospital for all ships of the fleet in that area. There were 418 patients received for treatment of injuries, an average of 41.8 per month. The average personnel of the fleet dependent on the *Relief* for hospital facilities was about 28,500.[1]

[1] Obtained from fleet surgeon of battle fleet and force surgeon of scouting force.

It includes only those cases of traumatism which were injured to such an extent as to necessitate their transfer to the hospital ship. It does not include the probably larger number of minor injuries which were cared for aboard their own ships. It does not include those patients who were injured while their ships were away from the vicinity of the hospital ship, either at navy yards or at other ports. However, it is believed that during the period covered, and serving such a large personnel afloat, it does represent a fair picture of the injuries sustained among such a force. It might be said to represent a cross-section of the peace-time casualties of the fleet.

TABLE 1.—*Classification of traumatic cases admitted to hospital ship from July 1, 1932, to July 1, 1933*

Type of injury	Injured aboard ship, in boats, or on duty ashore	Injured in athletic contests or training	Injured while riding in or on motor vehicles, liberty or leave	Injured when struck by motor vehicles as pedestrian, liberty or leave	Miscellaneous trauma ashore	Total
Fracture	80	19	65	9	29	202
Wound, punctures, lacerated, etc	34	0	35	1	10	80
Intracranial injury	5	2	24	2	7	40
Dislocation	8	6	1	1	2	18
Sprain of joint	15	8	1	1	2	27
Strain of muscle	4	3	0	0	1	8
Contusion	15	3	5	3	4	30
Burn	9	0	0	0	2	11
Unclassified	0	0	1	0	1	2
Total	170	41	132	17	58	418
Percent of total	40.67	9.81	31.58	4.06	13.88	100

Table 1 is a classification of all cases of injury admitted, showing the nature of their injury and where sustained. A number of patients of course received more than one severe injury at the same time, e. g.: "multiple fractures" with "intracranial injury." In this table such a case has been classified under the heading of that injury causing the longest disability.

The first column represents all those injuries sustained aboard ship, in boats, or on shore patrol or other duty ashore. No attempt has been made to separate those due to misconduct, negligence, or actually due to work. One hundred and seventy, or 40.67 percent, come under this heading. Forty-one, or 9.81 percent, were incident to athletics, whether ashore or afloat. These 2 classes, totaling 50.48 percent, account for approximately half of all injuries. They might be considered the normal hazard of sea duty in the fleet. Of the remaining half, 149 cases, or 35.64 percent of the total, are the result of vehicular injuries, the patient being either in or on the vehicle or hit by a motor vehicle while a pedestrian. This figure is of interest because there is a feeling among many officers in the fleet that vehicular accidents are a cause of a large majority of the in-

juries sustained by naval personnel. However, these injuries tend to be of greater severity, resulting in more loss of time from duty and separation from the service. This is borne out by the figures in table 2 which will be discussed later.

TABLE 2.—*Follow-up or end-result study of all cases (418) including sick days per case*

	Ship	Athletics	Motor vehicle, passenger	Motor vehicle, pedestrian	Miscellaneous trauma ashore	Total
Number returned to duty	160	41	114	17	52	384
Invalided from service	8	0	15	0	5	28
Died	0	0	1	0	1	2
Still under treatment, Mar. 1, 1934	2	0	2	0	0	4
Percent to duty	94.1	100	86.3	100	89.6	91.8
Percent died	0	0	0.75	0	1.7	0.47
Percent invalided	4.8	0	11.4	0	8.6	6.6
Total sick days [1]	6,235	1,047	4,929	878	2,349	15,483
Average number of sick days per patient [1]	38.96	25.53	43.25	51.65	45.17	40.32

[1] Includes only those patients returned to a duty status. Does not include those invalided from the service, died, or still under treatment on Mar. 1, 1934. Sick days include time spent in hospitals after transfer from hospital ship for completion of treatment.

Under the heading "Miscellaneous trauma ashore" are included all those cases who received injuries while on liberty or leave which were in no way connected with motor vehicles. Such trauma as falls, fights, burns, etc., compose this class.

Table 2 is an end-result study of all the cases reported in table 1; 384 were returned to duty; 28 had been invalided from the service; 2 were dead; and 4 were still under treatment on March 1, 1934. It should be borne in mind that the hospital ship acts largely as an evacuation hospital for the fleet. Limited bed capacity does not permit of keeping all patients till their final disposition. Those patients whose injuries are obviously of such a nature as to make their return to duty impossible, are transferred to a naval hospital as soon as practicable. Those requiring prolonged treatment and who will not be ready for duty within 3 months, are transferred for convalescence after their treatment has been well established. In the estimation of sick days the time spent in hospitals before final return to duty are included as well as time spent on the hospital ship.

It may be noted that both of the deaths in this series resulted from injuries ashore while on liberty or leave. In addition to those two deaths, figures obtained from the force surgeons of the Battle and Scouting Forces, show there were 32 other deaths among the personnel of the fleet in the San Pedro-Long Beach area due to injury. These 32 deaths occurred in individuals who would ordinarily have been transferred to the U. S. S. *Relief*, had their injuries not been fatal, immediately or within a short time. The mortality rate of 0.47 percent applies only to those patients who reached the hospital ship. Six and six-tenths percent of all cases were invalided from the

service, and again the greatest percentage occurs among the motor-vehicle injuries. A study of this table shows that injuries sustained on liberty or leave were the cause of all the deaths, a greater percentage of patients invalided from the service, and a higher average of sick days per patient.

TYPES OF INJURIES

Wounds include all types—lacerated, punctured, incised, gunshot, etc. These cases usually reached the hospital ship from 2 to 24 hours after injury and often had received appropriate treatment before transfer, so that the only treatment necessary on the hospital ship was rest and dressings. A majority of those sustained in accidents ashore had received treatment in a civilian hospital or emergency clinic and were already sutured when such treatment was indicated. Many of the patients with severe injuries also sustained wounds of some nature. In such instances the case is listed in the tabulation under "wounds" only when the wound was the outstanding and major trauma, e. g.: A case with a fractured femur and a lacerated wound of the scalp is listed only under fractures and is *not* listed again under wounds. One patient in this group was invalided from the service because of the residuals of a gunshot wound of the spine and abdomen. The injury was sustained ashore, the patient being shot when mistaken for a prowler. The missile, a 38-caliber revolver bullet, caused an injury to the third lumbar vertebra and the cauda equina with resulting paresis and sensory changes of the right lower extremity.

DISLOCATIONS

There were 25 dislocations among the cases admitted; 5 of these occurred in patients having more serious injury and tabulated under a different diagnosis, thus accounting for the fact that only 18 are shown in table 1. The dislocations were divided as follows:

Shoulder:	
Recent or acute	2
Chronic recurrent	1
Elbow	2
Hip	3
Clavicle:	
Acromial end	9
Sternal end	2
Knee, semilunar cartilage	6

The dislocations at the acromio-clavicular joint comprise the largest series and are of particular interest. This displacement is almost invariably due to a fall on the tip of the shoulder. The acromion process of the scapula is thus thrust violently downward from its articulation with the outer end of the clavicle. The ligaments holding the outer end of the clavicle in place are torn and this portion of the bone is

carried upward. Muscular action then prevents its replacement. The deformity is easily seen, for it is usually very marked. As a rule there is very little discomfort after the first few days and slight disability. These dislocations are easily reduced but not easy to maintain in reduction as, in our experience, it has been almost impossible to obtain permanent complete reposition of the clavicle as an end-result. The displaced end is easily pressed into position and can be held there with splints and pads. However, after immobilization for 6 to 8 weeks, when the splints are removed and the weight of the arm is allowed to pull the shoulder girdle downward, the deformity recurs at least partially. All of these patients have had good functional results with no disability, in spite of persistent deformity. We have not felt justified in submitting them to surgery merely to correct the deformity. Four of the patients have been carefully followed and all state definitely that their shoulder is as strong as ever and they have no weakness or limitation of motion.

Of the 6 patients with dislocation of a semilunar cartilage of the knee, 5 were chronic with histories of repeated incidents of "giving way" of the knee or "locking." The original injury to the joint was often minor and one patient could not recall any definite injury. Yet at operation a "bucket-handle" fracture of the internal semilunar cartilage was found and after operative removal of the offending cartilage, he was completely relieved of symptoms and returned to duty. One patient in this group did not obtain complete relief from the removal of a displaced cartilage and was subsequently invalided from the service. One patient was not operated on. He was admitted to the hospital ship soon after the first incident of "locking" due to a fall and twist of the knee. Following manipulative reduction he was free from symptoms and declined operation. He returned to duty and when last heard from, about a year after the injury, had had no further trouble.

It may be noted that most bluejackets with this disability have trouble over a relatively long period of time. The man quickly learns the proper maneuver to carry out when the knee locks and is usually able to carry it out himself. He is usually loath to seek medical aid and when he does so there is very little to be seen or felt objectively. He is usually given some baking and massage with a few days of light duty and the residual soreness quickly disappears. He is not referred for proper surgical treatment until he has been seen several times by the same medical officer or some serious fall or accident results from an incident of locking. An unstable knee is a serious handicap aboard ship and may result in a more serious injury to the individual when he is on a ladder, getting in or out of a boat or near machinery. It is our belief that every effort should be made to correct this disability in a sea-going man as early as possible.

The following case history is so typical that it is considered worth reporting:

L. M. L., Seaman 1c. (Apr. 20, 1933). About 18 months ago while in a football scrimmage, he was hit on the right knee. He does not know just what happened or what position he was in. As he plunged into the line somebody hit him across the knees and when he attempted to get up the knee was painful. He had to leave the game but was able to limp about. The knee was sore and swollen and he limped for about 10 days but was not on the sick list. He thinks that the inner side of the knee was the point of the greatest pain and tenderness. Since then there have been frequent occurrences of locking of the joint. These always occur when the knee is half flexed and the body weight is thrown on the outer side of the right foot. When in this position there will sometimes occur a snap or feeling as if the knee was out of joint. He is then unable to move the joint actively. However, if he forcibly straightens out his leg with his hands, twisting it slightly, there will be a snap and motion will be normal. The locking of the joint is always corrected by pulling the leg forward and making pressure over the patella. At first there would be soreness of the knee and swelling would develop after an episode of locking, but this is rather infrequent now. He states that he has never had any serious fall because of the locking of the knee, because he has learned not to depend upon it. He goes down ladders slowly and never jumps into a boat. He has never been on the sick list before because of the knee but has been treated by baking, hot towels, diathermy, massage, and liniments.

Past history.—G. C. infection of the urethra 2 years ago. Denies lues. Injured right knee 7 years ago, when a small baggage hand truck fell on it. The joint was swollen and painful for several days, but then caused no further trouble. No other injuries.

Physical examination.—Negative except for the right lower extremity. There was a normal range of motion of the right knee joint and no abnormal mobility of the joint could be demonstrated. There is a point of tenderness along the edge of the proximal end of the tibia anterior to the internal lateral ligament of the knee. This area appears slightly more depressed than the left. Laboratory examinations were negative. X-ray examination of the knee was reported as negative.

April 28, 1933, operation under spinal anesthesia. Jones incision to expose the internal semilunar cartilage showed it detached from the capsule in the lateral two-thirds of the cartilage. This portion of the cartilage would slip under the internal condyle of the femur into the intercondyloid notch. The anterior three-fourths of the cartilage was removed and the joint closed. The patient had an uneventful convalescence and returned to duty on June 6, 1933. He was again admitted to the hospital ship in November 1933 for treatment of an acute tonsillitis. He stated that his right knee was as "good as ever."

SPRAINS

There were 27 admissions for sprains of a joint, classified as follows:

Sprain of—	
Knee	12
Ankle	6
Elbow	2
Sacro-iliac	5
Lumbo-sacral	2
	27

Twenty-three were sustained either aboard ship or in athletics. As a rule the disability was not long and the patient returned quickly to duty. One man, however, was invalided from the service as the result of a severe sacro-iliac sprain received in a motorcycle collision.

STRAINS AND CONTUSIONS

Strain of muscles and contusion which resulted in 38 admissions were likewise due most often to injuries aboard ship. Those injured in vehicular accidents were often violently contused, but more serious coincident injuries disabled the individual long after the contusions were healed.

BURNS

The nine patients burned aboard ship suffered injuries of a relatively minor nature due to such miscellaneous agents as burning gasoline, hot liquids, electric flare, signal rockets, steam, etc. The average sick days was 24.5. The two patients burned ashore sustained extensive severe burns due to the explosion of illuminating gas from leaking stoves. One was so badly burned that there was extensive scarring of the head and face and gangrene of the left hand which necessitated amputation. The other patient died as a result of his burns 11 hours after admission to the hospital ship. Following is an abstract of the clinical history and autopsy findings in the fatal case:

F. D. H., chief commissary steward.—Admitted to the hospital ship at 1:45 a. m., August 22, 1932. The patient was semiconscious on admission. When roused he had difficulty in talking because of burns inside of mouth and was incoherent. Information obtained later from board of inquest revealed that he was injured at about 11 p. m., August 21, when he lighted a match in the kitchen of his house in Long Beach, Calif. Gas which had escaped from the kitchen range exploded, causing his burns. He rushed out of the house and fell on the lawn. Was taken by a police ambulance to a hospital, in that city, where his wounds were dressed and was then transferred to the U. S. S. *Relief*. There were extensive deep cutaneous burns over the face to the hair line, neck and ears and anterior surface of the thorax and abdomen to the belt line, and extending to the mid-axillary line laterally. Both forearms and hands from elbow to finger tips were likewise burned. All burns were covered with ointment dressings. The patient was placed in a warm air bath and the burns were sprayed with tannic acid solution. Given 1,000 cc. of normal saline intravenously. Patient died at 12:45 p. m., August 22—11 hours after admission and about 14 hours after being burned.

Summary of autopsy findings.—1. Second degree burn over face, neck, mouth, nose, forearms, hands, and anterior chest wall and abdomen.

2. Intense congestion and inflammation of the bronchial tree and to a lesser extent, the lung parenchyma. Moderate degree of hypostatic pneumonia.

3. Acute parenchymatous changes in all solid viscera.

4. Acute renal irritation, congestion, and inflammation.

UNCLASSIFIED

Two cases are included under this heading, both due to trauma received while on liberty. One man with a traumatic rupture of the small intestine, received in an auto accident, died. The other received an intraspinal injury from a knife wound in a street fight. He was subsequently invalided from the service. Abstracts of the clinical histories are given:

R. M. D., seaman 1c.—Admitted to the hospital ship at 12:45 p. m., July 27, 1932. Conscious and rational on admission but has no memory of events since the accident in which he was injured. History received with patient states he was in an auto collision at 5:45 a. m. on July 25, 1932. Taken to a hospital in Los Angeles where he has been under treatment until transferred to this ship. While he was in that hospital he was irrational and noncooperative. He now complains chiefly of abdominal pain and inability to void. Temperature 99.8°, pulse 106, respiration 28.

Physical examination.—Head: Face flushed, eyes bright, pupils react to light and accommodation. Mouth, nose, throat negative. There is a sutured, lacerated wound of the chin. There is some ecchymosis over the right mastoid and some old blood in the left ear. Neck is normal. Chest moves freely on respiration. Breathing is costal in type; breath sounds normal.

Abdomen.—Distended, tender, and rigid, especially in the upper half. Tympanitic throughout the abdomen. Liver dullness completely obliterated. There is no dullness in the flanks. No peristalsis on auscultation. Light percussion or palpation causes pain. No dullness over bladder region. Genitals normal. Extremities normal, moves legs freely. Back not examined. Patient unable to void, catheterized and 200 cc. of amber urine obtained. No free blood in urine. White blood count 11,700. Blood pressure 126/72. X-ray shows presence of large amount of free gas in the abdomen.

Impression.—Ruptured viscus with peritonitis. Operation 2 hours after admission. Spinal anesthesia reinforced by nitrous oxide and ether. Seven-inch left rectus incision. Abdomen contained a considerable quantity of free blood and a great deal of odorless gas. All blood possible removed by sponging and suction. Small intestines show signs of general peritonitis. No evident pathology seen or felt in the right upper quadrant or in the lower half of the abdomen. No adhesions in the pelvis. There was a large area of fibrinous peritonitis in the left upper quadrant, with numerous adhesions between the colon, spleen and gastro-splenic-mesentery. On separating the adhesions, a transverse rent in the jejunum was found. This was on the antimesenteric border, about 3 inches from the duodeno-jejunal flexure and was transverse, occupying one-half the circumference of the gut. Tear was sutured with double row of Lembert sutures, great omentum fastened over the suture line. Examination after suturing showed lumen of gut about the diameter of the index finger. Abdomen closed with cigarette drain to the upper left quadrant. Patient given 850 c.c. of intravenous saline during operation. 10 p. m. Transfusion of 480 cc. of whole blood. The patient seemed to improve for 48 hours but then weakened and died of general peritonitis at 7:15 p. m., July 30, 1932.

Autopsy findings.—Anatomical findings: (1) linear fracture of the skull and small extradural hemorrhage; (2) hemorrhage into pleural cavity; (3) peritonitis upper abdomen; (4) superficial rupture in spleen; (5) superficial rupture in the right kidney; (6) rupture upper pole left adrenal gland; (7) hemorrhage into esophagus, stomach, and upper part of small intestine; (8) suture of jejunum (12 cm from ligament of Treitz); (9) toxic parenchymatous changes in solid

viscera; (10) clostridium welchii organism found in the direct smears from the liver and blood stream.

Cause of death.—1. Multiple injuries (rupture small intestines, spleen, left kidney, and left adrenal gland). Skull fracture extending into middle fossa. 2. Infection, peritonitis. Blood stream infection. Hemorrhage into intestine. Clostridium welchii.

INTRASPINAL INJURY

Case A. P. E., coxswain.—Admitted to the hospital ship at 5:30 p. m., April 13, 1933. Conscious and rational but unable to move left extremities and rather dyspneic.

Present illness.—At about 2 a. m., this date, patient was attacked by a gang of negroes on the street in Los Angeles. He, in company with a shipmate and his wife and another girl, was leaving a cabaret when the attack occurred. Patient was stabbed in the neck and immediately fell to the street unable to rise. Taken to a hospital in Los Angeles where he was given emergency treatment. Chief complaint is pain in the left side of the back and inability to move arms or left leg. There is some difficulty in breathing.

Physical examination.—White male, age 25; height, 68½ inches; weight, 155 pounds. Well developed and nourished. Scalp negative. There is a lacerated wound three-fourths of an inch long on outer side of right elbow which has been sutured. Abrasion of auricle of right ear. Eyes normal, pupils are dilated, the right larger than the left. They do not react to light and accommodation. Patient is able to move head slightly. There is a lacerated wound three-fourths of an inch long 1 inch below and posterior to the tip of the left mastoid. This wound has been sutured. Small wound over the left suprascapular region. There is tenderness along the back of the neck. The head is tilted to the right. The chest is symmetrical, but there are no respiratory movements on the left side. Respiration Cheyne-Stokes in character with the accessory muscles of respiration being used. The heart sounds are normal. Breath sounds are diminished on the left side but are apparently normal. Blood pressure 130/96. Abdomen shows no masses or tenderness. Genitals normal. There is complete paralysis of the left side of the body and left extremities. Partial paralysis of the right upper and lower extremities. Patient has been unable to void since injury and has required repeated catheterization. X-rays of cervical spine taken at the civilian hospital have been examined and no evidence of fracture of the cervical vertebrae can be seen. In view of the lack of spasm and pain on movements of the head, it is believed that there is no bony injury. The wound of the neck probably was made by a stiletto which penetrated between the skull and cervical vertebrae or lower, wounding the cervical cord.

April 14, 1933, neuropsychiatric examination.—Pupils, mydriatic, rigid. No reaction. Other cranial nerves apparently normal, both sides (eye grounds negative). Reflexes, upper deep, active to diminished, upper arm and left shoulder to left side. All absent. Abdominals and cremasterics absent both sides. Patellars, absent, both sides. Achilles, present and active, both sides. Sensorium, right side, deep and light touch present only. Left side, pain and temperature, deep touch, light touch present. Some parasthesia also present. Vibration and position absent on left side. Muscle movements, right leg moves but is paretic. Right arm movements weak and paretic. Left leg and arm show a flaccid paralysis. The sensory disassociation begins at the level of second and third cervical cord or approximately second or third vertebra. Apparently it is a stab wound with a pointed instrument whose point has gone deep into the cord, giving an atypical Brown-Sequard Syndrome. Spinal fluid examination shows clear fluid.

April 16, 1933, no change in condition.

April 20, 1933, patient shows no evidence of motion of muscles of left upper or lower extremity. Fluoroscopic examination demonstrates normal excursion of the left diaphragm. No change in the paresis on the right side.

April 23, 1933, moves left leg slightly. Movements are very disassociated and purposeless. Reflexes still absent in left abdomen and left arm. Left Achilles and patellar present. No clonus. Reflexes are not abnormal in the left leg. Some sensory improvements in left leg. Vibration is good at pelvis, but this may be transmitted to the right side by bony structure. Right pupil mydriatic. Left normal.

April 24, 1933, patient now able to move left leg partially. Lumbar puncture shows a clear fluid under normal pressure. Improvement in sensation over left leg and thigh. No change in left upper extremity. Laboratory examination of spinal fluid shows no increase in globulin or cells. In view of this, it is believed that there has been no appreciable intraspinal hemorrhage which would indicate operative exploration of the injured area.

May 2, 1933, transferred to Naval Hospital, San Diego, Calif., for further treatment and disposition. Condition unchanged. (Eight sick days.)

Follow-up on August 2, 1933, about 3 months after the injury, the following information was received to an inquiry regarding his condition:

"Patient is able to sit up in bed and looks perfectly well. He is able to get on his feet and walk with a little support. He has very good function of the right side and is constantly improving on the left. There is still some spasticity and clonus can be obtained in all extremities but it is less marked than formerly.

INTRACRANIAL INJURIES

As shown in table 1, a large majority of these patients were injured in vehicular accidents. Twenty were injured by automobiles and six by riding motorcycles. Two were injured in football games. Those occurring aboard ship were due to falls, except one patient who hit himself in the face with a sounding lead while swinging it. Those injuries ashore, aside from vehicular accidents, were due to falls or assault. One patient was hit by falling material during an earthquake.

In addition to the 40 listed, 12 others with intracranial injury also suffered other severe injuries. These consisted mainly of fractures of the bones of the extremities or bones of the face. There were no deaths in this series, although undoubtedly a number of the 32 deaths referred to above, were due to severe cranio-cerebral injuries. One patient was invalided from the service because of a diplopia resulting from head injury.

In this group of injuries the symptoms have varied from mild to severe. The period of unconsciousness is difficult of estimation as patients injured ashore are necessarily delayed in reaching the ship. The longest period of unconsciousness observed was 5 days. The average period of unconsciousness, of the 19 patients who were unconscious on admission, was 24.7 hours.

Treatment of these cases has been conservative and consisted of measures aiming to control the increased intracranial pressure. Rest, dry diet, and restriction of fluid intake, and dehydration by

magnesium sulphate solution orally have proved to be the most valuable therapy in this series. Glucose (50 percent) intravenously has been given to those patients with symptoms of marked intracranial pressure as shown by deep unconsciousness and very low pulse. A dosage of 50 cc has seemed to be efficient, and this was repeated once in seven cases. Lumbar puncture was done on those patients showing the most severe symptoms, a total of 26. The pressure, as recorded in millimeters of mercury, has varied from 10 to 30. The average pressure in the 26 cases was 18.6 mm of mercury. Twenty-three of the patients who had lumbar punctures showed varying amounts of blood in the spinal fluid. Lumbar puncture for drainage and repeated puncture has not been used as a means of overcoming increased intracranial pressure. It seems reasonable that if capillary subdural hemorrhage has occurred, possibly this hemorrhage is stopped or controlled by the high pressure of the cerebrospinal fluid. If sudden reduction by drainage is then instituted, it is rational that fresh hemorrhage may again be started by such sudden reduction of pressure.

One patient was subjected to operation on the eighth day after injury for the removal of subdural hematoma. Focal symptoms and convulsions indicated local pressure on the right side of the brain. Through a trephine opening in the right subtemporal region a large amount of blood clot was removed from the subdural space. The trephine button was replaced and the wound closed. The patient made an excellent recovery and returned to duty. It is of interest to note that although he appeared mentally clear and lucid for several days before operation, after complete recovery he had no lucid memory of any events occurring until 10 days after the injury and 48 hours after the operation.

FRACTURES

Two hundred and two patients were admitted for fracture. Table 3 shows the type and site of fracture and also the final disposition. Twenty, or about 10 percent, were invalided from the service. Of these, 14 were injured in vehicular accidents, 5 were injured aboard ship, and 1 was injured during an earthquake while on liberty. Four patients were still under treatment on March 1, 1934. The remainder, 178, had been returned to duty prior to that date. There were no deaths in this series.

Table 4 shows how the fractures were sustained. It will be noted that by far the greater proportion of serious fractures was due to vehicular accidents. Fractures sustained while on liberty or leave account for 53.3 percent of the total admissions for fractures and 75 percent of those surveyed from the service because of fracture.

The following list shows the diagnosis of those patients who were invalided from the service:

Compound fracture of the tibia and fibula (6 vehicular accidents, 1 earthquake) 7
Simple fracture of the tibia and fibula (on board ship) 1
Simple fracture of os calcis (1 vehicular, 2 ship board) 3
Compound fracture of bones of hand (crush; ship board) 2
Compound fracture of radius and ulna (vehicular) 2
Simple fracture of femur (vehicular) 2
Simple fracture of humerus (vehicular) 1
Compound fracture of mandible (vehicular) 2

TABLE 3.—*Type and site of fractures*

Fracture of	Simple	Compound	Total	Invalided from service	Still in hospital
Mandible	25	11	36	2	1
Maxilla and bones of face	3	5	8	0	0
Clavicle	14	0	14	0	0
Scapula	3	0	3	0	0
Humerus	3	2	5	1	1
Forearm	11	3	14	2	0
Wrist [1]	7	0	7	0	0
Metacarpals and hand	12	11	22	2	0
Ribs	9	0	9	0	0
Spine	8	0	8	0	0
Pelvis	7	0	7	0	0
Femur	6	0	6	2	1
Patella	1	1	2	0	0
Tibia and fibula	11	10	21	8	1
Ankle [2]	18	0	18	0	0
Tarsus	6	1	7	3	0
Metatarsals and foot	12	2	14	0	0
Total	156	46	202	20	4

[1] Fractures involving lower end of radius and, or, carpal bones.
[2] Fractures involving lower end of tibia and fibula and ankle joint.

TABLE 4.—*Classification of fracture, showing where or how sustained*

	Injured aboard ship, in boats or on other duty ashore	Injured in athletic contests or training	Injured by motor vehicle either as passenger, driver, or pedestrian	Miscellaneous trauma ashore	Total
Mandible	10	3	7	16	36
Maxilla and bones of face	2	1	4	1	8
Clavicle	1	4	8	1	14
Scapula	0	0	3	0	3
Humerus	0	0	5	0	5
Forearm	5	0	7	2	14
Wrist	2	2	3	0	7
Metacarpals and hand	21	0	2	0	23
Ribs	3	2	3	1	9
Spine	4	0	4	0	8
Pelvis	2	0	4	1	7
Femur	1	0	5	0	6
Patella	1	0	1	0	2
Tibia and fibula	5	0	15	1	21
Ankle	4	5	7	2	18
Tarsus	5	0	2	0	7
Metatarsals and foot	11	0	3	0	14
Total	77	17	83	25	202
Percent of total	38.1	8.4	41.2	12.3	100

A detailed discussion of types of fractures and treatment will not be attempted in this report. A few outstanding points are, however, worthy of comment.

Fractures of the mandible comprise the largest group in this series. The majority were sustained by the time-honored method of a blow of the clenched fist. This large series offers silent evidence of the combativeness and pugnacity of the modern bluejacket which is so necessary for the elan and effectiveness of a fighting man. He is not afraid to "take it on the chin" as long as he has an opportunity to deliver his blow also.

The fractures of metacarpals and bones of the hand are interesting in that they present the largest single group of cases which were injured aboard ship. These fractures are often serious, always disabling and require careful and early treatment to prevent prolonged or permanent disability. They offer a fertile field for preventive work in industrial surgery. Those injuries aboard ship were sustained as follows:

Hand caught in machinery	7
Hand carried into a block on a moving line	4
Hatch cover fell on hand	4
Hand crushed between boats or boat and ship	2
Miscellaneous	4

TABLE 5.—*Anesthesia employed*

Anesthesia for—	Local or regional	Spinal	General, gas or ether	Total
Closed reduction of fracture	50	12	6	68
Open reduction of fracture	7	9	5	21
Suture of wound, debridement, etc	19	7	7	33
Operation on joint or reduction of dislocation	8	6	3	17
Operation on head (skull)	1	0	0	1
Total	85	34	21	140
Percentage of total	60.8	24.2	15	100

Fractures of both bones of the lower leg comprise another large group in this series. Seventy-five percent occurred while the individual was on liberty or leave, and again vehicular accidents are the most common cause. All of the compound fractures in this group were sustained ashore, 6 while riding a motorcycle and 3 in automobile accidents. The vulnerability of the lower leg to serious injury in motorcycle accidents can be readily understood. Most of these fractures were severely compounded and often comminuted. When these patients are received on the ship early after injury, debridement and primary closure has given most gratifying results. Those compound fractures received on the ship too long a time after injury for primary closure and with wounds showing gross infection have received the Orr type of treatment. The convalescence of these patients has been prolonged, and although the ultimate result may be good, as far as function is concerned, the protracted nature of their disability often leads to separation from the service.

SUMMARY

1. A review of the injured naval personnel treated on the U. S. S. *Relief* for a period of 1 year is presented.

2. The injuries sustained were about evenly divided between those occurring aboard ship or in athletics and those occurring while on liberty or leave.

3. Vehicular injuries comprise a large percentage of those injured ashore and were also responsible for more average sick days and more men invalided from the service.

THE PRACTICE OF MEDICINE IN AMERICAN SAMOA

By JOSEPH L. SCHWARTZ, Lieutenant Commander, Medical Corps, United States Navy

It is not the purpose to present a detailed treatise on medical and surgical diseases in American Samoa, but, rather, to present a general survey as well as a summary of the more interesting surgical conditions and entities that were encountered. The race of natives that inhabit these islands have such an interesting environment that it is proper to first consider this phase, and then to present some idea of how they live, and to consider the various factors that influence them in health and disease.

The group of five islands that comprise American Samoa is located some 4,200 miles west of San Francisco and some 500 miles south of the Equator. All the islands are volcanic in origin, and all are small and all mountainous. The island of Tutuila, on which is located the naval station, is the largest of the group and is about 18 miles long, 5 miles wide, and cut almost in two by the Bay of Pago Pago. The estimated area of this island is about 40 square miles, very little of which is level land, yet the produce of its soil supports a population of over 8,000.

The climate is tropical but equable with variable winds, occasional gales, and infrequent hurricanes. The average daily temperature is 81 degrees. From a seasonal standpoint the year may be divided into a wet season with an average rainfall of 20 inches per month, and a dry season with an average rainfall of 10 inches per month. The highest recorded rainfall for a year is 284 inches, and the lowest is 130 inches.

The Samoans are pure Polynesians, and they are perhaps the finest physical specimens of the race. This aboriginal race still maintains to a great degree most of their ancient habits and customs. They rarely migrate, and only infrequently do they come in contact with other races. By reason of their isolation they have not been affected to any appreciable extent by those social and economic adversities that have influenced our own civilization.

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In demeanor they are light-hearted, happy, and gay, given to feasts, fascinating (Siva) dances, and untruths. Rarely does the Samoan engage in very intensive physical effort, or even in the intensive or methodical cultivation of his land, since such efforts on his part are not altogether necessary, as nature is prodigal in her favors and a simple planting produces a ready and abundant growth. In appearance the Samoan is reddish brown in color, well formed, and well developed, the race having many of the characteristics distinctive of the European, with a firm chin, straight nose, and a high forehead. The men are tall, muscular, and rarely corpulent. The women are not particularly beautiful, but in young womanhood they have beautiful figures.

Food.—The diet of the native is essentially high carbohydrate and low residue, consisting of such food as taro, breadfruit, bananas, coconuts, yams, papayas, etc. Fish and shellfish, as well as chicken and pigs, are also eaten, but the latter two are mainly reserved for feasts.

From a practical standpoint the native does not consume any alcoholic beverage. The national drink, known as kava, is an aqueous emulsion made from the powdered root of the *Piper methysticum*. Kava is not an intoxicant and has no injurious effects, but may produce some weakness of the legs if drunk in large quantities, and if taken in excessive quantities over a period of time may produce a peripheral neuritis of the lower extremities.

Communicable diseases.—Tuberculosis and pneumonia are the leading causes of death. As in other aboriginal races the incidence of tuberculosis coincides with the coming of the white man, who undoubtedly introduced the causative organism among a people lacking racial immunity. Pneumonia may be attributed to the wearing of clothing on the upper part of the body, and repeated drenchings by rain during the day is apt to produce a chilling of the body which is probably the predisposing factor. Typhoid, paratyphoid fever, and filariasis are endemic. Acute tonsillitis and the exanthema are very uncommon. Filariasis is extremely common and begins to manifest itself after the age of 20, and from that age on the incidence rises very abruptly. The filarial embryo can be found in the fresh blood smear of about 20 percent of the natives, regardless of the presence or absence of symptoms.

Yaws is principally a disease of children, and more than 90 percent of the Samoans living under native conditions develop or have had yaws. Tertiary yaws is extremely uncommon, undoubtedly as a result of the introduction of the arsenicals in the treatment of yaws for which it acts as a specific. In scanning the laboratory data we find that in over 90 percent of Samoans the Kahn test is positive, yet the entity syphilis such as we see it in other parts of the world is

unknown here. Aneurysm, aortitis, paresis, tabes dorsalis are not seen. Gonococcus infection of the urethra in the male is very uncommon, and no cases of gonococcus salpingitis were observed in the Samoan women during a period of about 1 year and a half.

Intestinal parasites, ascaris, hookworm or whipworm in any and all combinations are found in over 75 percent. Although there is a high incidence of hookworm infestation the Samoan does not present the devitalizing effects or the anemia that is usually observed in other races when this parasite is found. Dental caries as well as albumin and casts in the urine are common findings in the adult.

Mosquitoes of the varieties *Aades variegatus*, which is the vector of filariasis and *Aades aagypti* and *Albopictus* which are the vectors of dengue fever are in abundance. Dengue fever, however, is not of common occurrence. Flies are numerous, and in the breadfruit season when they are particularly prevalent they are probably the disseminating factor in the occurrence of so-called "Samoan conjunctivitis", which involves both the conjunctiva and the cornea. Blindness and corneal ulcer is a common sequel of this infection, especially as a result of neglect or from the native Samoan treatment, which consists of abrading the cornea by various means.

Surgical conditions.—From a standpoint of surgical pathology there are many features of interest. During a period of less than 18 months about 164 patients were admitted to the Samoan Hospital for operation. The usual routine laboratory tests were made on all patients, and where indicated, X-ray study. The anesthesia used in all but two of these operative cases was either local novocain infiltration, caudal or spinal anesthesia. Spinal anesthesia, often combined with local novocain infiltration, was used in all abdominal explorations. Local infiltration combined with caudal anesthesia was used in operating for elephantiasis of the scrotum and local infiltration alone for practically all other operative procedures. The Samoan is a perfect subject for these types of anesthesia, and is extremely cooperative. The average adult dose of procaine for spinal anesthesia was 100 mgm. No ill effects were noted in any case.

Since the entire officer personnel of the medical department was composed of three medical officers, no anesthetist was available. A friend of the patient, usually a Samoan pastor, would accompany the patient to the operating room and act in the capacity of a pseudo-anesthetist by conversing with the patient or consoling him, and the intonation of these voices was used as one guide to the patient's condition. Glucose saline solutions intravenously or subcutaneously were used freely, and blood transfusions where necessary. Seventeen patients were transfused and no difficulty was ever encountered in obtaining sufficient volunteers to act as donors.

The following is a résumé of some of the surgical procedures and conditions that were noted:

Skull fracture.—The patient, a male of about 40 years of age, was struck on the head with a bush knife. The weapon apparently penetrated to a great depth as brain substance was escaping rather freely. Nevertheless, the patient was conscious, rational, and no motor or sensory changes were observed. The wound was thoroughly cleansed with saline. Hemorrhage from the scalp wound only was present and that was controlled. The wound was dressed and the patient returned to bed. After a period of about 24 hours the patient lapsed into coma and a generalized spastic paralysis ensued. About 72 hours later the patient had regained consciousness, the motor functions were returning, and a well-developed hernia cerebri was present. For the following 3 days the patient's condition was improving rather rapidly, with the herniation of brain increasing daily. On the eighth day there was a sudden elevation of temperature, the patient again lapsed into coma, and death ensued. On post-mortem examination the fracture was found to be a quadrilateral section of the parietal bone about 3½ by 1¾ inches. The middle meningeal artery was not involved, and only a very moderate amount of extradural hemorrhage was noted. There was extensive destruction of brain tissue. Plastic exudate covered all the cerebral hemispheres. A smear showed gram positive diplococci.

Colloid adenoma.—Two cases of colloid adenoma of the thyroid were operated. These two cases are mentioned because of the opinion of most authorities that goiter is not found along the seacoast, nor in those who eat seafood, while the reports of other workers indicate that goiter may be the result of toxins produced in the intestines by bacteria introduced in impure drinking water and that a high fat or high carbohydrate, vitamin-poor diet will also result in goiter.

Branchial cleft sinus.—One case of branchial cleft sinus and two cases of osteomyelitis of the body of the mandible were operated.

Empyema.—Empyema in a child aged 2 was treated by intercostal drainage with a catheter.

Bronchogenic carcinoma with suppuration.—The patient, a male aged 70 years, was admitted complaining of cough, expectoration, and pain in the right chest. Extensive dullness involving the right middle and lower lobes was found. There was only a moderate leucocytosis. On X-ray there was a marked increase in density of the right lung extending as high as the fifth rib posteriorly. On aspiration a very small amount of pus was obtained from the lung. Under local novocain infiltration the eighth rib in the posterior axillary line was exposed and a section of the rib removed. The lung was found to be solid and no respiratory excursions were observed. The parietal and visceral pleura including the lung were incised with actual cautery.

ELEPHANTIASIS OF SCROTUM AFTER OPERATION.

ELEPHANTIASIS OF SCROTUM BEFORE OPERATION.

Multilocular areas of necrosis and suppuration were broken into, but no free pus was found. Drainage was established. The patient lived about 6 months, finally returning to the bush to die. No other metastasis were noted on repeated physical and X-ray examinations. No post mortem was obtained.

Cholecystectomy.—Cholecystectomy was done in three patients with symptoms suggestive of gall-bladder disease. Under X-ray examination the gall bladder in all these cases failed to visualize after the use of oral dye. However, on examination of the gall bladder none of them showed sufficient pathological changes in the gall bladder to account for the symptoms, and we surmise that a hepatitis resulting from repeated attacks of filarial fever may have been the pathological basis for the symptoms in these cases.

Ulcer of the duodenum.—Ulcer of the duodenum was encountered in two cases. The first case, that of a cook boy working on the naval reservation, was admitted with an acute perforated ulcer. The other case, a Samoan of 40 years of age, came from a distant island and presented a typical duodenal ulcer history and X-ray findings. Suture of the first case and gastro-jejunostomy in the other resulted very satisfactorily.

Appendectomy.—Appendectomy was performed in 20 cases. The pathology varied from the acute gangrenous type to the so-called "chronic" appendicitis. All the patients in this group were young adults. All lived in close proximity to the naval reservation or worked as domestics on the reservation, and all of them had ready access to a diet containing a far greater proportion of protein than the diet of the Samoan living under native conditions.

Intestinal obstruction.—Intestinal obstruction was found in four cases. In two, the ileum was involved, one as a result of post-puerperal pelvic peritonitis, the other resulting from post-operative adhesions. The third case presented an angulation of the hepatic flexure of the colon as a result of adhesions between the transverse and ascending colon, for which no basis could be assumed. The fourth case was that of a Samoan woman of 27 years of age who was admitted to the hospital after an illness of 10 days, during which time she was ineffectually treated by means of Samoan cartharsis, which consists of huge doses of magnesium sulphate or else administering a thick brown residue that is obtained after prolonged boiling of the coconut husk. An annular carcinoma of the sigmoid that had produced a complete stricture was the cause of obstruction.

Disease of female pelvic viscera.—Operation for disease of the female pelvic viscera was done in 24 cases, the pathology including fibroid uterus, ovarian cyst, chronic salpingitis, retroversion, prolapsed uterus, and ectopic pregnancy. The cases of salpingitis resulting from gonococcus infection were not Samoan women.

Hernia.—Hernia in the male is rather common. Twenty-eight cases were operated, and the types of hernia included 2 epigastric, 3 femoral, 1 of which involved a loop of strangulated ileum in an emaciated Samoan of 60 years of age, who had been ill for over a week. A combined femoral and abdominal approach with the release of the strangulated, bluish loop by severing Gimbernat's ligament, resulted in very satisfactory recovery.

Elephantiasis and hydrocele.—Elephantiasis of the scrotum and hydrocele are very common and are the result of filariasis. Repeated attacks of lymphangitis and obstruction of the lymphatics by the action of the microfilaria produces a marked thickening of the tunica vaginalis in the cases of hydrocele and a marked thickening of the integument with a blubbery edema of the subcutaneous tissues in the cases of elephantiasis of the scrotum or the extremities. The occurrence of hydrocele as either unilateral or bilateral is of about even distribution. In about 15 percent of cases an haematocele is noted. The association of hydrocele with elephantiasis of scrotum is common. The size of the filarial scrotum varies from that of a large grapefruit to that of a huge watermelon or larger. The largest one observed weighed 37 pounds. The results of operation for both hydrocele and elephantiasis of the scrotum are very satisfactory. However, there is always a recurrence of the elephantiasis of the scrotum after a period of years.

Inguinal adenitis.—Extensive inguinal adenitis without involvement of the extremities is frequently observed. No operative procedure is indicated in these cases since a removal of the enlarged glands will result in elephantiasis of the extremities.

Abscesses.—Deep-seated abscesses, at times multiple and often retroperitoneal are of common occurrence, and the result of filariasis. These heal readily after incision and drainage.

Elephantiasis of the extremities.—Elephantiasis of the extremities presents no surgical problem, nor is treatment often necessary, except local treatment during an attack of lymphangitis. In only one case of elephantiasis of the lower extremity was amputation of the leg necessary, and this was on account of the extensive foul-smelling ulcer encircling the lower third of the leg. It was rather surprising to note that healing of the remaining blubbery tissues was by first intention.

Ascaris and the acute surgical abdomen.—The symptoms produced by infestation with ascaris are usually negligible. However, serious pathology may occur as a result of their migrating into the bile ducts, the appendix or gall bladder. Occasionally they produce a mechanical obstruction of the small intestine. Of more rare occurrence is the perforation of the walls of the intestine by the ascaris, and whether the perforation results from the mechanical action or chemical action

of the worm can only be surmised. Sakaguchi notes that after injecting guinea pigs with a quantity of so-called ascaris toxin, death occurred in from 5 to 14 hours and degenerative changes were noted in the stomach, intestines, liver, adrenal glands, etc. Two cases of perforation of the intestine by the ascaris were operated, one in a Samoan woman of 40 years of age, who presented a picture typical of the acute surgical abdomen. On opening the abdomen, purulent exudate was found, the omentum was gangrenous, and was adherent to a large retroperitoneal tumor lying to the left of the vertebral column. Edematous omentum, turgid and violaceous, capped this tumor, and then came down to become adherent to a high loop of ileum plugging up the perforation in this part of the intestine. An ascaris was found lying between the omentum and the tumor.

The tumor was about 5 inches in diameter, and was a dermoid containing hair. The other case was that of a girl aged 12 years, who was admitted complaining of moderate abdominal pain, fever, and constipation. Her relatives stated that she had complained of abdominal pain for the past 5 days, and that during 3 days previous to her admission to the hospital was given repeated doses of castor oil and enemas without result. Her temperature was 103.2, pulse 120, and respiration 32. She appeared very toxic. The action of accessory muscles of respiration and alae nasi was noticeable. The throat and lungs were clear. The abdomen was distended and only very moderate guarding was present. There was no audible peristalsis. White blood count was 14,600, pmn's 84 percent, haemaglobin 70 percent. She was given intravenous glucose saline, hot applications were applied to the abdomen, and she was put up in Fowler's position. The following day her temperature was 101.8, pulse 116, and respiration 32. Operation was done under spinal anesthesia, using 75 mgm of procaine. On opening the abdomen, pus, gas, and dark bloody fluid escaped. The upper loops of the ileum were red and covered with a plastic exudate. Gas and feces were seen escaping from the left side of the pelvis in which location was found a perforation in the ileum about three mm across, and an ascaris lying free between the loops of ileum. The ascaris was removed, and ileostomy was done inserting the tube through the site of the perforation in the ileum, and the abdomen drained. Her general condition was fairly good and she continued to hold her own. Two days following operation it was notices that the drainage bottle contained no solution, and an order was left to put a 1 percent lysol solution in the bottle. The Samoan nurse who carried out the written instruction took the ileostomy tube and instilled about 2 ounces of 1 percent lysol solution into the ileotory tube and so into the intestinal tract.

The following day the patient's bowels moved, and she passed seven dead ascaris. Her temperature was 100, and she was apparently improving nicely without evidence of any toxic action from such unusual treatment. Twelve days after admission her temperature was 98.8, pulse 90, and respirations 18. General condition was good. The urine showed 3 plus albumin, hyaline casts and 12 to 15 pus cells per field. Hb. was 60, red blood cells 2,500,000, white blood cells 9,650. Seg. 72, band 13, juvenile 2 L. 11, E. 2. We were interested as to the possibility of typhoid or paratyphoid being the basis for her perforation and laboratory results were as follows: Kahn test was positive, of course. Urine culture showed B. coli, Widal was negative. Blood culture was negative and stool culture negative. During her convalescence she developed a bronchopneumonia from which she eventually recovered, and finally left the hospital in good condition.

Malignancy.—Malignancy in the Samoan seems to be rather common. Besides the carcinoma of the sigmoid and lung there were noted carcinoma of the stomach and primary carcinoma of the gall bladder on which exploratory laparotomy was done, extensive epithelioma of the face, melanotic sarcoma of the foot with numerous metastasis to the leg and thigh, carcinoma of the head of the pancreas and a primary carcinoma of the liver. The last case presents such interesting findings that it is also reported in detail.

Primary carcinoma of the liver.—A male, aged 38 years, was admitted April 20, 1932. Chief complaints were abdominal pain, anorrexia, nausea and occasional vomiting, and loss of weight. The patient had been ill for 4 months. No history of previous illness. On examination the patient was definitely emaciated, the sclera jaundiced, teeth poor. Head, neck, and throat showed no abnormality. There was a very moderate adenopathy of the posterior cervical glands. The lungs were clear, the heart normal. The abdomen was moderately distended, and the liver nodular and palpable about four fingers below the costal arch. There was shifting dullness in the flanks. There was a moderate hydrocele. The extremities showed no particular abnormality other than elephantiasis. Temperature, pulse, and respirations were normal. R. B. C. Hb. 80, W. B. C. 6,300, P. 77, L. 18, E. 4 M. 1. No microfilaria. Stool, flagellate and pus cells. Negative for occult blood. Urine showed albumin two plus, hyaline casts and granular casts. Fasting meal 35 cc. Free hcl. 4, T. A. 18, lactic none, pus cells and bacteria. Test meal (Ewald)—free hcl.—none, T. A. 10, lactic none, pus cells and bacteria present. X-ray G. I. series, no 6-hour residue, cap somewhat hazy, but no definite pathology in the upper gastro-intestinal tract. The patient continued to have pain and he vomited occasionally. On April 27, 1 week after admission he complained of sudden, very severe abdominal pain.

ELEPHANTIASIS OF EXTREMITIES.

The abdomen was more distended and rigid, and the sclera showed a more intense jaundice. The Hb. was 65, R. B. C. 3,030,000, W. B. C. 5,000, P. 82, L. 16, and E. 2. Operation was done under spinal anesthesia. The abdomen was found to be filled with blood and bloody fluid. The liver was considerably enlarged and nodular, and on the under surface of the right lobe was a large friable bleeding mass. On post-mortem examination a careful search failed to reveal evidence of malignancy in any organ other than the liver. The liver was markedly enlarged, and studded with numerous masses, and on the under surface of the right lobe there was a large fungating friable mass. The pathological report was hepatoma.

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GASTRIC RETENTION IN PEPTIC ULCER[1]

By Clarence C. Kress, Lieutenant Commander, Medical Corps, United States Navy

A study of a group of cases of peptic ulcer showing gastric retention is made with a view of determining the incidence of the various factors which may be responsible for the gastric stasis.

Many authors have emphasized the frequency with which gastric stasis in duodenal ulcer is found to clear up after a prolonged period of medical management without operation, indicating that the retention in many instances does not depend upon cicatricial changes.

Sippy states that—

operative experience shows that of all cases of duodenal and pyloric ulcer presenting definite clinical evidence of obstruction at the outlet, less than 10 percent are found at operation to be associated with actual tissue narrowing of serious grade.

He differentiates two types of pyloric obstruction in peptic ulcer:

(1) Pyloric obstruction due to conditions easily removable by 2 weeks of the accurate medical management advocated; more than 90 percent of all cases of pyloric obstruction due to ulcer are of this type. (2) Pyloric obstruction due to anatomic narrowing from infiltration tissue, some of which may be cicatricial tissue in varying stages of development. The probable cause of the first is pyloric spasm, inflammatory swelling, and at times perhaps local peritonitis.

Cases of the second type, where after giving a full motor meal, 100 cc or more of food is found at the end of 7 hours, were formerly subjected to gastroenterostomy, I am now convinced that unless the opening is so very narrow as to threaten starvation, the size of the opening will practically always increase; and none has yet failed to open up sufficiently to allow a motor meal to be emptied in 7 hours. Operation is useless in such a case.

[1] From the Gastrointestinal Service of Dr. H. L. Bockus, Graduate Hospital, Philadelphia. Thesis submitted to the faculty of internal medicine of the Graduate School of Medicine of the University of Pennsylvania in partial fulfillment of the requirements for the degree of master of medical science (M. Sc. (Med.)) for graduate work in gastroenterology.

Crohn states that it is because of the large element of accompanying spasm and edema, that medical treatment of pyloric stenosis in peptic ulcer has still a functional and important usage, and remarks how successfully some cases of pyloric stasis can be handled by means of systematic lavage and dietary regimen. Hurst and Stewart report recovery of a person suffering with pyloric obstruction who was still well after 16 years without operation. Rafsky reports 12 cases of peptic ulcer with pyloric obstruction. Of these, 9 have been perfectly well since the medical treatment, 1 of the other 3 was past 60 years and remained symptom free as long as medical treatment was continued. Two did not improve under medical treatment and required the aid of surgery. He describes two stages of obstruction—although it is clinically and radiographically impossible, at times, to differentiate between them. The first stage of spasm, edema, congestion, and lymphoid hyperplasia is amenable to medical measures. The second stage of fibrotic changes, cicatricial contraction and a permanent closure or stenosis of the pylorus requires surgery for relief, and results in the second stage are very favorable. Hypertrophy or dilitition of the stomach may occur in either the first or second stage but usually the latter, depending upon the duration of the ulcer and the amount of obstruction present. There was no alkalosis in any case. All were ambulant cases except one. Rafsky concludes from study of his series of 12 cases that the only way to discover whether the patient is suffering from the first or inflammatory stage (congestion, pylorospasm) or from the second or cicatricial stage (stenosis) is by the therapeutic test of 4 to 6 weeks of observation—if the condition gradually subsides but X-ray fails to show appreciable improvement then stenosis is present. Friedenwald and Morrison report five cases of peptic ulcer in which pyloric obstruction of a rather aggravated type existed, which definitely yielded to medical treatment. All five were of long standing, many with typical signs and positive X-rays. There must have been actual tissue narrowing present. Shay and Schloss present a case of peptic ulcer with pyloric obstruction relieved by medical treatment and advise trial of medical treatment of pyloric obstruction (stenosis), watching the blood chemistry for signs of approaching alkalosis. Scriver reports a case of pyloric obstruction in peptic ulcer responding to medical treatment.

Bockus reports two cases of the rarer causes of obstruction near the pylorus easily mistaken for (marked) pyloric obstruction, in the Negro. Both cases developed alkalosis, both were syphilitics. One was a case of enormous calcified tuberculosis of the mesenteric glands causing extrinsic pressure and stasis in the second and third parts of the duodenum, the basis of some of the gastric retention. At the same time there was also present a callous duodenal ulcer playing a part in the gastric retention. Both cases accentuated the difficulty of diagnosis in the Negro, with subjective symptoms so far exceeded

by the pathology present. He remarks that it is common to find syphilis and tuberculosis with less classical peptic ulcer in the Negro. Bockus, Glassmire, and Bank, in a review of 200 cases of duodenal ulcer, report that 54 percent had motor delay as determined by fractional gastric analysis, while 31 percent gave evidence of gastric retention by the barium meal. Fifty of the 200 cases of duodenal ulcer with delayed motility had a second gastric analysis performed at periods varying from 2 months to 5 years after medical treatment was first instituted. The last gastric analysis showed improvement in stomach emptying in 15 cases and normal motility in 15. It was their experience that no further improvement in stomach emptying occurs in cases with retention after the first 3 months of medical treatment. If the gastric analysis at that time reveals retention of grade 2 or more, which can be attributed to the ulcer, a permanent cure should not be expected by medical treatment. The element of pylorospasm has been largely eliminated and the residual delay in emptying is dependent upon some degree of pyloric stenosis, and surgical treatment must be given consideration. Konjetzny states that—

> what must next be determined is the differentiation between spastic and organic pyloric stenosis. Continued spasm in chronic gastroduodenitis can develop into hypertrophic pyloric stenosis, a true organic stenosis of the pylorus. A typical clinical ulcer case brought to operation for pyloric stenosis is found at operation to be one of gastroduodenitis, instead of ulcer, as the cause of the pyloric obstruction.

Not infrequently even at the present time in some quarters patients with peptic ulcer associated with gastric retention are subjected to gastroenterostomy without a careful preliminary survey and period of stomach rest. Many such cases develop recurrent symptoms. As Jenkinson has indicated, a gastroenterostomy for duodenal ulcer with stasis not due to stenosis is not physiologic. The tone of the stomach improves for a while following gastro-enterostomy and becomes smaller, as does also the stoma due to contraction of the stomach. While the stoma functions, the ulcer improves, spasm and edema disappear, and food again begins to pass the pylorus freely. Resumption of the normal channels in preference to its passage through the stoma, not only tends to further closure of the stoma, but reactivates the ulcer inflammation, edema, spasm, and symptoms. Retention and decompensation of the stomach supervene, due to the closure of the gastroenterostomy, and secondary operation must be done. Jenkinson further remarks that if the stomach is dilated markedly at beginning of treatment, preparation for operation should include small frequent feedings, aspiration and lavage at night and aspiration in the morning, thus eliminating or lowering hypersecretion, reducing tension, and improving the tone of the stomach, so making the patient much more comfortable and a better surgical risk.

TABLE I

	Name	Age	Sex	Race	Duration of symptoms	Duration of vomiting	Alkali, grade of	Assoc. Spastic Colitis	Gastritis	Lues	Grade of initial retention, G. A.	Grade of initial retention, X-ray	Type and duration of interval treatment	Grade of check retention, G. A.	Grade of check retention, X-ray	Accurate diagnosis	Follow-up: Operative: Symptoms, free	Follow-up: Operative: Recurrent	Follow-up: Nonoperative: Recurrent	Follow-up: Nonoperative: Symptoms, free	Stenotic	Nonstenotic
34958	J. S.	30	M	W	5 years	2 weeks	0	0	0	+	2	2	HS, 3 weeks; P, 12 days; B, 8 days; Atr, 10 days; T_{12} AntL.	2	2	D. U. and G. U.	---	rec.	---	---	+	---
29174	I. R.	59	M	W	12 years	3 months	4	0	0	+	2	2	HS, 4 weeks; P, 7 days; B, 3 weeks; A, 18 days, AntL.	1	2	D. U	---	---	---	---	+	---
103011	A. M.	41	M	W	20 years	---	0	0	3	0	1	2	HS, 4 weeks; P, 8 days; A&L, 10 days; B, 12 days.	1	2	D. U	+	---	---	---	+	---
100898	W. G.	35	M	W	2 years	---	0	0	3	0	1	1	HS, 3 weeks; P, 6 days; A&L, 8 days; B, 2 weeks.	0	0	D. U	---	rec.	---	---	---	+
	P. L.	29	M	W	2 years	4 days	0	0	3	0	2	1	HS, 3 weeks; P_2, 7 days; A&L, once day, 7 weeks; B, 3 weeks; twice day, 2 weeks.	2	2	D. U	---	---	---	---	+	---
88330	H. R.	70	M	W	8 years	2 months	3	0	+	0	3	---	HS, 6 weeks; P, 6 days; A, 2 weeks; B, 20 days; Atro, 8 days.	2	3	D. U	---	rec.	---	---	+	---
19466	A. Q.	61	M	W	2 years	1 month	2	0	0	0	3	3	HS, 4 weeks; P, B, A	2	2	D. U	---	---	---	---	+++	---
69490	D. B.	46	F	B	8 years	3 weeks	0	0	0	0	3	1	HS, 3 weeks; P, 8 days; $T4_2$	3	1	D. U	---	---	---	---	+++	---
91998	F. B.	38	M	W	12 years	---	0	0	0	+	1	2	HS, 2 weeks, Atr, 8 days; A, 14 days; P, 8 days.	0	1	D. U	---	rec.	---	---	---	---
196403	M. B.	42	M	W	20 years	1 year	2	0	0	0	2	3	HS, 5 weeks; A&B, 12 days, p. m.; A&L, 8 days, a. m.; Atr, 8 days.	3	1	D. U	---	rec.	---	---	+	---
105923	R. C.	53	F	W	1 year	3 months	0	0	0	0	3	3	HSB, 4 weeks; A&L, once day, 3 weeks.	2	3	D. U	---	---	---	---	+	---
29281	G. K.	53	M	W	25 years	Occasional	0	0	0	0	2	2	HSB, 2 weeks	1	2	D. U	---	rec.	---	---	+++	---
190253	R. P.	66	M	W	1 year	5 months	2	0	0	0	0	2	HSB, 9 weeks; P, 6 days; T_{34}	---	3	G. U	---	---	---	---	+++	---
95600	S. C.	59	M	W	7 years	1 month	4	0	0	0	---	3	HS, 4 weeks; P, 23 days; A&L, 23 days; B, 10 days; T_{34}.	1	0	D. U	---	---	---	---	---	---
198900	I. K.	45	M	W	3 years	---	0	0	0	0	2	1	Cl, 3 weeks; #2, A, B, L	2	1	D. U	---	---	---	++	+	---
	E. Mc.	23	M	W	1 year	---	0	0	2	0	1	2	Cl, 10 weeks; A, B, #2, #3, #9	---	1	D. U	---	---	---	+++	---	++
108479	B. W.	55	F	W	8 years	---	0	0	0	0	1	1	HSB, 3 weeks	---	0	G. U	---	---	---	+++	---	---
108477	G. B.	31	M	W	5 years	6 months	+	0	0	0	2	1	HS, 4 weeks; A&L, 10 days; B, 4 weeks	1	0	D. U	---	---	---	+++	---	---
89738	R. S.	74	M	W	7 months	---	0	0	0	0	0	2	HS, 5 weeks; B, 7 days; T_3	---	1	G. U	---	---	---	---	---	---
106926	H. L.	63	M	W	3 years	2 months	0	0	0	0	2	2	HS, 2 weeks; A&L, a. m. and p. m.; and P_2, 10 days.	3	3	D. U. and G. U.	---	---	+	---	---	---

122997	L. C.	30	M	W	3 months		0	0	0	0	2	0	Cl, 4 months; #2, L, B, #3	0	0	G. U				+		+
100304	F. L.	55	F	W	12 years	3 months	0	0	0	0	1	1	HS, 5 weeks; B, 3 weeks; Atr, 2 weeks	1	1	G. U			+	+		++
86575	J. C.	31	M	W	4 years		0	+	0	0	2	0	HS, 4 weeks; A, 12 days; B, 3 weeks	0	0	G. U				+		+++
70995	L. B.	41	M	W	2 months		0	0	0	0	2	0	HS, 10 days; BAL, 2 weeks	0	0	G. U				+		++
81623	G. H.	37	M	W	20 months		0	2	0	0	2	2	HS, 4 weeks; B, 10 days	0	0	G. U				+		++
100260	B. K.	53	M	W	8 years	4 weeks	+	0	2	0	3	3	HSB, 26 days; A, a. m., 25 days; A&L, p. m., 20 days.	2	2	D. U				+	+	
89741	A. L.	60	M	W	6 years	6 days	0	0	0	0	0	2	HS, 3 weeks; B, 4 weeks; A&L, once day, 10 days.	1	0	G. U			+	+	+	
40361	V. W.	26	F	W	18 months	10 weeks	0	0	0	0	1	3	Cl, 1½ years; S, A, #2	2	1	D. U. and G. U				+	+	
34120	J. M.	52	M	W	10 years	1 week	+	0	0	0	1	0	HS, 18 days; B, 3 weeks	1	0	G. U. and D. U			+	+		+
102136	M. L.	35	M	W	5 years	2 months	0	0	0	0	1	3	HS, 5 weeks; Atr, 8 days; B, 4 weeks; A, 2 weeks.	0	0	D. U				+		+
81969	J. G.	31	M	W	11 years	1 month	2	0	4	0	3	2	HS, 5 weeks; P, 5 days; Atr, 8 days; A, 5 days.	0	0	D. U. and G. U			+	+		+
73251	E. F.	32	M	W	5 years	Occasional	0	0	0	0	1	0	HS, 2 weeks in 1928; 2 weeks in 1933	1	1	D. U			+	+		+
	D. A.	29	M	W	10 years		0	3	0	0	2	0	Cl, #2, #3, #4; A, B, 8 weeks	0	0	D. U			+	+		+
96972	M. L.	47	M	W	3 years	3 days	+	0	+	0	2	0	HS, 18 days; Atr, 6 days; P, 4 days; A, 17 days.	1	0	D. U			+	+		+

Grade 1. Alkalosis.—55 to 60 percent CO_2.
Grade 2. Alkalosis.—60 to 65 percent CO_2.
Grade 3. Alkalosis.—65 to 70 percent CO_2.
Grade 4. Alkalosis.—More than 70 percent CO^2.
Grade 1. Retention by G. A.—50 cc or more fasting juice, no food; 51 to 65 cc, 2-hour juice, with or without 10 cc food.
Grade 2. Retention by G. A.—51 to 150 cc fasting juice, microscopic food; 51 to 200 cc, 2-hour juice, 21 cc or more food.
Grade 3. Retention.—101 to 650 cc, fasting juice, gross food.
Grade 1. Retention by X-ray.—Up to 10 percent, 6-hour barium residue.
Grade 2. Retention by X-ray.—From 11 to 25 percent, 6-hour barium residue.
Grade 3. Retention by X-ray.—26 percent or above 6-hour barium residue.

Abbreviations:

A—Daily fasting residue by aspiration.
A&L—Aspiration and lavage.
AntL—Anti Luetic treatment.
Atr—Atropin sulphate for night pain.
A—Alkalies.
B—Belladonna.
Cl—Clinical observation: Ambulatory.
HS—Modified Sippy treatment in hospital.
P—Parenteral fluids and glucose.
P_g—Parenteral glucose alone, no other nourishment.
T—Transfusion.
O—Recurrence from which recovered.
x—times.
G. A.—Gastric Analysis.
G. U.—Gastric Ulcer.
D. U.—Duodenal Ulcer.

Diets in use at Gastrointestinal Clinic, Graduate School Medicine, University of Pennsylvania:

#2—Diet in second stage ulcer.
#3—Diet in third stage ulcer.
#4—Diet in Fourth stage ulcer.
#9—Smooth Diet. Narrowing of bowel lumen. Post-operative anti-peristalsis.

In the preparation of this thesis, the records of 140 cases of peptic ulcer were reviewed. Thirty-four cases of pyloric obstruction were found in which the clinical data and follow-up management were sufficient to justify inclusion for analysis. This does not truly indicate the incidence of pyloric disfunction in peptic ulcer as some cases with retention could not be included because of lack of follow-up. By consulting table I, the degree of stasis by gastric analysis and by X-ray, before treatment was started, can be seen in each case. The type of treatment used and the degree of resulting stasis, if any, at the termination of the treatment, is given. The 34 cases of stasis in this series comprise 20 patients with duodenal ulcer and 5 with both gastric and duodenal ulcer. Of the 9 cases with gastric ulcer alone, the gastric ulcer was located near the lesser curvature on the posterior wall, pars media in 4; just distal to the incisura angularis in 2; on the lesser curvature near the pylorus in 1; proximal to incisura angularis in 1; and in 1 case the ulcer was on the greater curvature near the cardia. In some of the cases with gastric ulcer alone, gastroptosis played a part in association with the ulcer as a factor in the stasis.

There were 5 women in the series, 1 of them a Negro. The average age of the entire group was 45.3 years. The ages varied between 23 and 74 years. There were 6 between 26 and 30 years of age; 8 between 31 and 40 years; 6 between 41 and 50 years; 8 between 51 and 60 years; 5 between 61 and 70 years; and 1 over 70 years of age. The average duration of digestive symptoms was 6.9 years. All, except 3 of the 34 cases in this series, had a period of hospitalization on a strict medical ulcer regimen of from 3 to 4 weeks. Three received only 2 weeks' hospitalization, while 10 were hospitalized from 6 to 8 weeks. Following the period of hospitalization, all patients were reexamined 1 to 10 years later. The check examination averaged 1½ years following the initial study.

Stenotic pyloric obstruction.—In 20 of the 34 cases (59 percent) the retention was considered to be dependent upon actual stenosis at the pylorus. Fourteen in this group were proven by operation. In the remaining 6 cases not operated upon, the criteria for the diagnosis of organic stenosis consisted of the persistence of large fasting residua as shown by gastric analysis, and 6-hour barium residue and enlargement of the stomach by X-ray after 3 or 4 weeks of a strict medical ulcer regimen in bed in the hospital, or under a much longer period of observation as an ambulant patient under the care of the gastrointestinal clinic of this institution. Fourteen stenotic cases came to operation, with a mortality of 50 percent. Four, of the seven surviving operative patients, remain comfortable the greater part of the time and are able to do light work. Operation was only partially successful in three others, having corrected the hyperacidity but failed to

establish complete gastric compensation due to imperfect functioning of the stoma. Recurring discomfort is controlled in these three by diet, belladonna, and alkaline powders. One has recently developed a recurrence of symptoms apparently due to a marginal ulcer.

The following case is cited as one typical of stenotic retention:

I. R., white male, 59 years of age, presented a duodenal ulcer syndrome of 12 years' duration, during which time he required 4 periods of hospitalization. In 1923 at the start of his gastrointestinal treatment, X-ray examination showed a 6-hour barium residue of 80 percent; in 1927 this was 15 percent; in 1932, 20 percent; in 1934, 25 percent. He was luetic, receiving antiluetic treatment in 1930–32. Gastric analysis persistently showed a marked 2-hour gross food retention. Toward the end of this period of clinical observation under a careful medical regimen, protracted vomiting of a retentive nature and high grade alkalosis supervened. This led to a period of Sippy treatment in the hospital in March 1932 during which the gross food 2-hour retention by gastric analysis, was reduced to 20 cc, and the X-ray showed a 6-hour barium residue of 25 percent. Duodenal ulcer with second degree pyloric stenosis was diagnosed and this diagnosis was confirmed at operation. A jejunal ulcer just distal to the stoma of the gastroenterostomy was observed by X-ray on February 14, 1934, during the course of a routine check-up without recurrence of symptoms.

Nonstenotic pyloric obstruction.—Disappearance of pyloric obstruction (gastric stasis) on a strict medical regimen was taken as evidence of noncicatricial stenosis. Fourteen of the thirty-four cases (41 percent) thus fell into the classification of the nonstenotic type. Among the 14 nonstenotic cases, practically all remain symptom free under periodic supervision. Two of this group strongly suspected of being malignant, remain symptom free after 2 to 3 years.

The following case is cited as one typical of nonstenotic retention:

M. L., 102135, a 35-year-old white male, gave a history of appendectomy and hernia repair in 1927 with relief for a short period, followed 2 months later by attacks of periodic epigastric pain following meals. The duodenal ulcer syndrome recurred off and on for 5 years, and in the past 2 months vomiting has developed, becoming retentive recently. Gastric analysis at this time, October 12, 1932, showed grade 1 retention, with unusually high sustained acid curve, no gross food, blood, or bile, but considerable detritus microscopically (gastritis), while X-rays showed a grade 3 retention with obstruction due to a duodenal ulcer with attendant pylorospasm and antiperistalsis. The duodenal cap was very irritable, constantly deformed, irregular, and tender. Gastritis was present although the stomach was normal in size. Sippy treatment in hospital with daily aspiration and lavage, atropin for night pain, belladonna during the day, constituted the treatment. Pain and vomiting stopped after a week's treatment. The fasting resuduum, after 3 weeks was reduced from 100 cc to 60 cc, and after 4 weeks to 30 cc, while X-ray showed no 6-hour barium residue on November 19, 1932. Foci of infection were cleared up by extraction of abscessed teeth. Antral lavage and treatment of an ethmoiditis gave relief of frontal headache.

The following survey of findings is made in the attempt to reveal a clue to the differentiation of the stenotic from the nonstenotic type in the management of cases of gastric retention in peptic ulcer.

TABLE II

	Cases of organic stenosis, 20 cases (59 percent)	Cases of noncicatricial stenosis, 14 cases (41 percent)
Age	50.4 years	38 years.
Sex:		
Male	19	12.
Female	1	2.
Race:		
White	19	14.
Negro	1	
Luetic	MW-3	None.
Duration of ulcer symptoms (average)	7.6 years	5.6 years.
Severe hemorrhage	4 cases	None.
Persistent bleeding with anemia	3 cases	5 cases.
Incidence of gastritis	7 cases	2 cases.
Associated spastic colitis	None	4 cases.
Duration of vomiting (average)	11 weeks, in 15 cases—½ to 52 weeks.	5 weeks, in 5 cases—½ to 12 weeks.
Original degree of retention	G. A.: Grade 1, 2; grade 2, 9; grade 3, 5. X-ray: Grade 1, 5; grade 2, 8; grade 3, 6.	G. A.: Grade 1, 7; grade 2, 6; grade 3, 1. X-ray: Grade 1, 3; grade 2, 3; grade 3, 1.
Enlarged stomach, by X-ray	15 cases. Marked in 7 cases	None.
Visible peristalsis or sucussion splash	5 cases	None.
Alkalosis	9 cases	1 case.
Arteriosclerosis	6 cases	None.

Gastric analysis on November 5, 1932, after 4 weeks of medical treatment shows no retention and definite improvement, in the quantity of microscopical detritus or gastritis findings. Blood chemistry, blood count, serology, biliary drainage, and stools were all negative. On November 28, 1932, he was discharged free of ulcer symptoms with relief of pyloric obstruction after 5 weeks on a medical ulcer regimen. He has remained sympton free.

In this series of 34 cases of peptic ulcer with stasis, 59 percent were stenotic or organic in type, but together with the stenosis in eight there was an associated functional condition about the pylorus as shown by the definite lessening of the retention (accounted for by relaxation of pylorospasm, or reduction of inflammation edema, etc.) after 2 to 4 weeks of strict ulcer regimen. So it is fair to assume that in many cases of pyloric cicatricial stenosis the degree of obstruction is augmented by a functional stenosis or inflammatory swelling. It is obviously impossible to determine without a period of strict medical management what percentage of the retention is dependent upon actual organic stenosis. A comparison between the stenotic and nonstenotic cases (table II) serves to emphasize a number of clinical observations which should help in some measure to differentiate functional from stenotic obstruction due to ulcer. In the stenotic group: (*a*) The average age was considerably greater; (*b*) the duration of ulcer symptoms was longer; (*c*) the incidence and duration of vomiting was greater; (*d*) severe hemorrhage occurred more frequently; (*e*) there was a higher incidence of associated gastritis, arteriosclerosis and alkalosis; (*f*) the degree of gastric retention as revealed by gastric analysis and X-ray study was greater, and (*g*) enlargement of the stomach was decidedly more marked. Obviously the existence of many of the above factors in any given case favor cicatricial stenosis. High grade retention sufficient to cause marked

enlargement of the stomach and gradual onset of retention vomiting in association with alkalosis will usually be found to be dependent upon some degree of organic stenosis. However, there is no clinical syndrome which makes an absolute differentiation.

SUMMARY

Thirty-four cases of peptic ulcer with gastric retention were analyzed. After a period of strict ulcer management, the gastric stasis, as measured by gastric analysis and X-ray, disappeared in 14, or 41 percent. In 59 percent retention persisted, indicating some degree of organic cicatricial stenosis at the pylorus. Fourteen of these were proven at operation. In the cases with cicatricial stenosis the average age, average duration of symptoms, the degree of retention, severity of symptoms, incidence of alkalosis, size of the stomach, and the occurrence of concomitant gastritis and arteriosclerosis exceeded that present in the cases of gastric stasis not dependent upon stenosis.

CONCLUSIONS

An attempt to ascertain, as promptly as possible in every case, the relative degree of stenosis, if any, and of functional closure of the pylorus is of great importance in the selection of therapy. Most clinicians favor a preliminary period of strict medical management in all cases of peptic ulcer with retention.

It has been observed that every case must be subjected to a period of 3 to 4 weeks of strict medical management, unless the retention is of such a high grade that nourishment can only be maintained by continuous parenteral introduction of fluids, as there is no short cut available in the detection of the degree of stenosis. It is the experience in this clinic that no further improvement in stomach emptying occurs in cases with retention after the first 3 months of medical treatment. If the gastric analysis at that time reveals retention of grade two or more, which can be attributed to the ulcer, a permanent cure should not be expected by medical treatment. The residual delay is dependent upon some degree of pyloric obstruction, and surgical treatment is indicated.

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THE PHYSIOLOGY, PATHOLOGY AND DIAGNOSIS OF NEPHRITIS [1]

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The classification of nephritis is one of the most perplexing problems in medicine. There have been a great many classifications set forth by the various students of Bright's disease that one becomes confused when reading the literature due to the various terms employed. It is unfortunate that the various authorities cannot come to a more unified classification in order that the students and practitioners of medicine might read literature on nephritis without becoming confused and be able to follow the various authors.

The principal difficulty lies in the nomenclature. Bright divided his conditions into dry and wet nephritis which was satisfactory from a clinical standpoint. But as time went on pathologists began to discover various types of kidneys, as large white kidneys and small red kidneys, and began using terms as primary contracted kidneys, secondary contracted kidneys, chronic interstitial nephritis, and parenchymatous nephritis.

A clinical classification is difficult due to the fact that in nephritis we are dealing with signs and symptoms which are extrarenal, as edema and high-blood pressure. Clinically some cases run a short fulminating course with rapid destruction of the kidney, in others the course is prolonged and many cases with great destruction live to ripe old age. Our knowledge of the clinical side of nephritis dates back 100 years when Richard Bright made such a clear-cut analysis of his cases. He was aware of all the clinical facts that we know of today. Blood chemistry, the sphygmomanometer and the hemo-

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globinometer have helped little in solving the problems that confront us.

A classification cannot be based on etiology, as the etiology of some of the nephritides is still unknown. A classification based on etiology would be ideal but until the etiological problems are solved such a classification must be abandoned.

There is no doubt that the pathological classification at the present time is the most satisfactory. Because in understanding it we know fairly well what is going on in the kidney. A pathological classification is taught in the United States Naval Medical School; but before the classification is discussed in detail a physiological and histological review is in order.

The unit structure of the kidney is the nephron which consists of the afferent arteriole, the glomerulus, and the proximal and distal convoluted tubules. The number of nephrons have been estimated at 2,000,000. Richards, in studying the frog's kidney, discovered that the number of functioning glomeruli varied at different times, sometimes as many as 10 were seen in a 2 mm field, at other times 2 or 3. From this observation we can understand why such large numbers of nephrons can be destroyed without producing symptoms of renal failure and also why certain glomeruli are in the path of bacteria or toxic substances and others are not. Richards also found that glucose and sodium chloride were present in the glomerular filtrate and not present in the bladder urine; that glucose and sodium chloride were reabsorbed in the tubules.

Glomeruli consist of a capillary bed, being lined by an inner vascular endothelium and an outer epithelium. This epithelium is one of the most important functional structures of the kidney. It permits the passages of sodium chloride and glucose but does not permit the passage of proteins. The functioning of the glomeruli depends on several factors. First, on the pressure—the filtering pressure is 80 mm of hg. The plasma proteins exert an osmotic pressure of about 40 mm which tends to hold the fluids back inside. Filtration can only take place when the ureteric pressure is below 80 mm. If the ureter is ligated and the pressure recorded above the ligation, filtration ceases when the pressure reaches 80 mm of hg. Secondly, blood flow—when the renal vein is ligated the glomerular pressure is raised but the blood flow through the glomerulus is greatly reduced. The epithelium covering the glomerular capillaries suffers from asphyxia and we have albumin and red blood cells escaping into the capsular spaces. At night the rate of flow is reduced, and the blood pressure lowered, consequently the night urine does not exceed 450 cm.

The tubules are lined with cuboidal cells which have a brush border. The tubules modify the glomerular filtrate by a process of differential absorption. The various constituents of the plasma are

not treated alike. Sugar is completely absorbed, creatine and urea sulphate are concentrated about 80 times; this is due entirely to the absorption of water from the lumen of the tubules back into the plasma. If the tubules absorbed water alone, then all of the products of the glomerular filtrate would be concentrated to the same extent. In 24 hours there is excreted in the urine about 30 grams of urea. The average blood urea is 0.03 percent, which means that there is contained in 100 liters of plasma 30 grams of urea. In order to have 30 grams of urea in the urine 100 liters of plasma must pass through the glomerular epithelium in 24 hours. On an average, 1.5 liters of urine is passed in 24 hours so 98.5 liters of glomerular filtrate must be absorbed by the tubules. Sugar, chlorides, sodium, and bicarbonate are spoken of as threshold substances and are more or less completely reabsorbed depending on the needs of the body. On the other hand, creatinine, sulphate, and for practical purposes urea are not threshold substances and are excreted so long as any are present in the body.

The kidney regulates the fluid content of the body. If a large amount of water is consumed there is immediately set up a diuresis (urine of low specific gravity) until fluid volume becomes normal. On the other hand, when no fluid is consumed the kidneys hold back the fluids of the body and very little urine is passed; the passed urine has a very high specific gravity. The dilution-concentration kidney function test is based on ability of the kidney to dilute and to concentrate the urine.

The kidney also under abnormal conditions aids in the regulation of the hydrogen-ion concentration. The kidney can excrete the equivalent of 60 to 70 cm of normal acid solution in 24 hours.

The functions of the kidney may be enumerated as:

1. The maintenance of the normal osmotic pressure of the plasma and tissues.
2. To maintain the normal levels of the threshold substances.
3. Eliminates waste products, the nonthreshold substances, drugs, and poisons.
4. Maintains the water balance.
5. Helps maintain acid base balance.

Just what relation there exists between the pituitary and the kidney is not definitely understood. Pituitrin evidently acts directly on the epithelium of the tubules increasing the absorption of water. Diabetes insipidus patients drink and excrete large quantities of water, sometimes up to 40 liters daily. If water is withheld serious symptoms develop. If pituitrin is given without limiting water intake grave manifestations develop from excessive fluid content of the tissues.

The pathology of nephritis is confined to the cortex of the kidney. It must be understood that as the blood vessels, glomeruli, and tubules are so closely related that it is impossible to have injury to

one without resultant injury to the other two, but to a less extent. If the glomerulus is injured the corresponding tubule suffers as it receives its blood supply from the afferent arteriole as it leaves the glomerulus. On the other hand, the narrowing or sclerosis of the afferent arteriole will result in decreased blood supply to both the glomeruli and the tubules. The kidney being one of the most intricate structures of the body, the different components are so dependent on one another that damage to one part will result in damage to the other parts. From an anatomical standpoint we may have three types of nephritis, namely, that affecting the glomeruli, the tubules, and the blood vessels. The following classification is taught at the United States Naval Medical School:

- Glomerulonephritis
  - Acute.
  - Subacute.
  - Chronic.
- Nephrosis
  - Amyloid.
  - Toxic.
  - Chemical.
  - Lipoid.
- Arteriolosclerosis
  - (A) Without renal insufficiency:
    1. Cerebral symptoms.
    2. Cardiac symptoms.
  - (B) With renal insufficiency.

In comparing this classification with that of Christian, Addis, Van Slyke and associates, Fishberg, O'Hare and Volard, and Fahr, we find that most of them include under vascular nephritis either one or both arteriolosclerosis and arteriosclerosis. There is some question as to whether or not arteriosclerosis should be included in the classification of nephritis. In this condition there is a generalized sclerosis of all of the larger blood vessels of the body and the kidney is no more affected than other organs of the body. Usually there is no hypertension or evidence of kidney failure aside from a little albumin in the urine and an occasional hyaline cast. It matters little what classification we go by for when they are carefully analyzed they more or less all refer to the same condition only using a different nomenclature.

Glomerulonephritis—Syn.—Acute nephritis, hemorrhagic nephritis, Bright's disease acute, glomerulonephritis diffuse acute.

Etiology.—Unknown. This type of nephritis is associated with certain types of infections, namely, scarlet fever, tonsillitis, erysipelas, and rheumatic fever. The causitive organism is probably streptococci. The condition does not come on during the height of the disease but during the convalescent period, which points to the fact that it may be an allergic phenomenon.

Pathology—Gross.—The kidney is enlarged, capsule strips with ease leaving a gray surface which may contain small hemorrhages. The cut surface is moist with bright red blood.

Microscopic.—There is a proliferation of the epithelium of the tuft and the endothelium of the capillaries and intracapillary hyalinization. This accounts for the avascular glomeruli. The capsular space contains albumin, leucocytes, and red blood cells. Round cell infiltration is usually present. Not all of the glomeruli show this change, for even in the chronic stages of this disease some glomeruli are found with little or no pathological changes.

Cases of glomerulonephritis acute are not frequently encountered in the service. This condition occurs frequently during childhood. There is no doubt that a great many of the acute cases are not recognized as the only symptons may be a little puffiness of the eyes, and if the urine is not examined at this time the diagnosis is not made. Cases of glomerulonephritis acute do occur in the service and as these cases are rarely fatal it is only rarely that a case comes to autopsy.

Glomerulonephritis, subacute—Syn.—Subacute nephritis with edema, chronic parenchymatous nephritis, large white kidney.

Etiology.—Same as acute glomerulonephritis.

Pathology—Gross.—The pathological process is a continuation of that described under glomerulonephritis acute. The kidney in most cases is enlarged and referred to as the large white kidney. The capsule strips with ease, the surface of the kidney is smooth and pale. The cut surface is pale and soft. The cortex is thicker than normal and may be spotted with yellow areas.

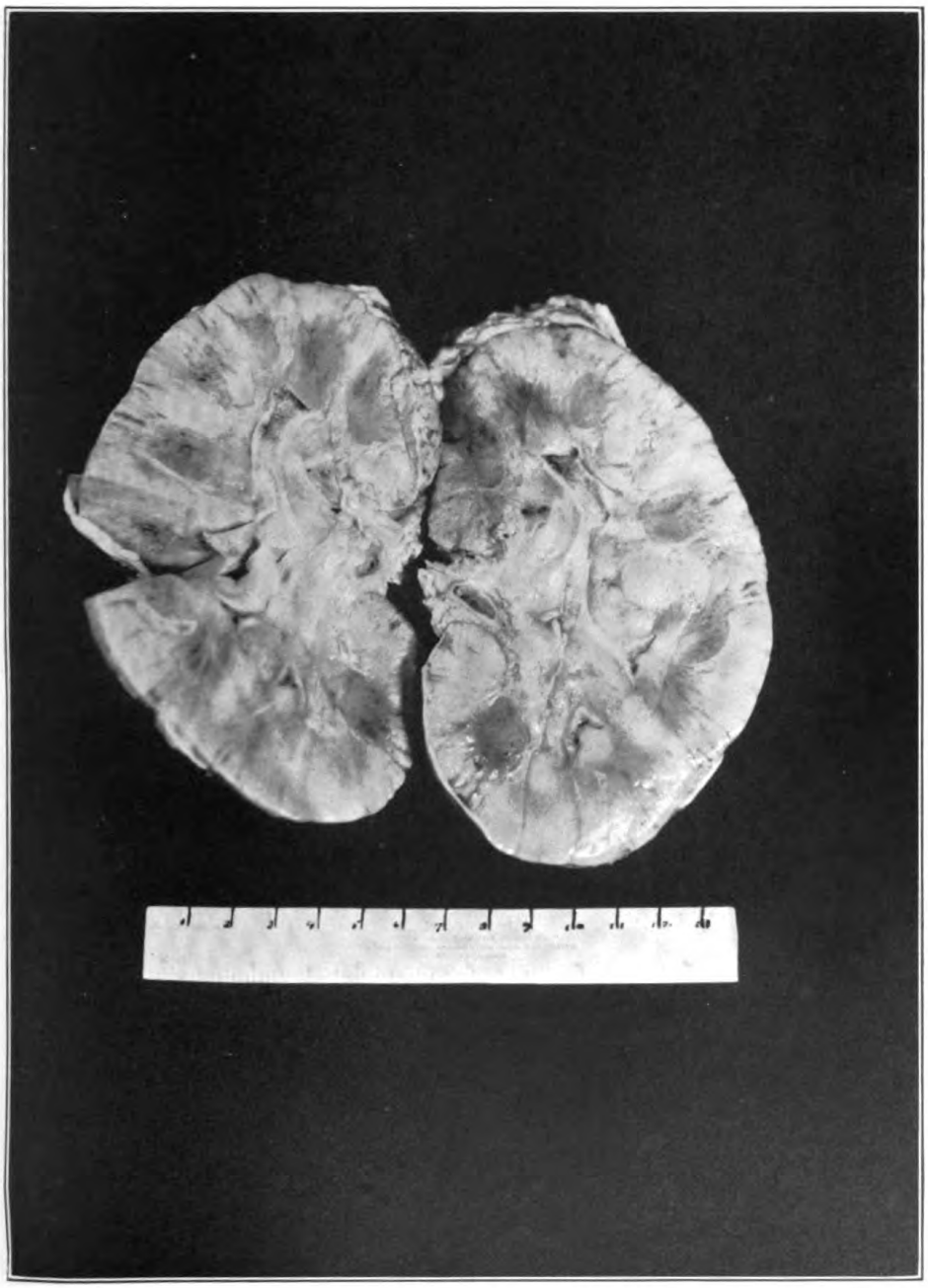
Microscopic.—The glomeruli are greatly enlarged; some show marked proliferation of the epithelium lining of the tuft and also the capsular epithelium. These cells may mass in one part of the glomerular space in crescentic forms and are known as the "epithelial crescents." In the glomerular space are seen albumin, red blood cells, and white blood cells. The glomeruli show various stages of hyalinization, some being completely hyalinized. The tubules show parenchymatous and fatty degeneration and even necrosis. Scattered throughout the interstitial tissue are collections of round cells. Some of the blood vessels may show thickening of the intima.

Glomerulonephritis, chronic—Syn.—Chronic nephritis without renal edema, Hemorrhagic nephritis chronic, Hemorrhagic Bright's disease, diffuse glomerulonephritis chronic, and chronic interstitial nephritis.

Etiology.—Same as acute glomerulonephritis.

Pathology—Gross.—The kidney is small and referred to as the small white or red kidney, the granular contracted kidney and the secondary contracted kidney. The capsule is firmly adherent so that pieces of the cortex are torn in removing the capsule. The surface is covered with fine granules. The cut surface reveals a thin irregular cortex, normal markings are obscure.

Microscopic.—Large numbers of the glomeruli are completely replaced by fibrous tissue, others undergoing proliferative changes previously described. An occasional normal glomeruli may be seen.



LARGE WHITE KIDNEY OF AMYLOID NEPHROSIS

The convoluted tubules are atrophic, some undergoing compensatory hyperplasia, others are dilated and are filled with detritus. There is great increase of the fibrous tissue. Blood vessels are thickened.

This is the most frequent type of kidney found at autopsy in cases of nephritis which die in the naval service. Glomerulonephritis progress is gradual from the acute form to the subacute form ending in the chronic form. It is estimated that between 90 and 95 percent of all of the acute glomerulonephritis recover and that the remaining few percent give us our subacute and chronic glomerulonephritis. A great many of the acute cases are not recognized and in taking a history it is impossible to elicit a history of previous attacks.

Case no. 1.—J. E. N., fireman third-class, age 34 years, admitted November13, 1933. Diagnosis, nephritis chronic. Chief complaint: Poor eyesight, severe headaches. Past history: Scarlet fever, age 10, otherwise negative. Present illness: About 2 weeks ago patient noted his vision was failing and suffered from severe headaches. No history obtained of previous attacks. Physical examination: Face puffy, heart enlarged, blood pressure 190 over 110, pulse full and regular. No edema present. Eye grounds showed marked neuroretinitis. Urine: Specific gravity 1.014, reaction acid, albumin heavy trace, few granular casts. R. B. C. 1,770,000, Hgb. 50 percent, W. B. C. 5,900, blood urea nitrogen 75, creatinin 5.6. Kahn negative. The specific gravity was fixed between 1.004 and 1.015, with trace of albumin and hyaline and granular casts. December 8, 1933, blood indican 3 plus; December 9, 1933, patient died in uremia. At autopsy a contracted kidney was found. Microscopic diagnosis: Chronic glomerulonephritis.

Case no. 2.—M. G. S., pensioner, age 27 years, admitted July 28, 1933. Diagnosis, nephritis chronic. Chief complaint: Frontal headaches, blurred vision, swelling of eyes and ankles. Family history: Negative. Past history: Scarlet fever 1923, developed nephritis following it and was surveyed 1924. In July 1933 had influenza. Present illness: Following influenza his vision began to fail, developed severe frontal headaches, edema of eyes and legs, vomited following meals. Physical examination: Skin pale waxy like, edema of lids, lungs clear except for rales in dependent portions, heart slightly enlarged, pulse regular. Blood pressure 180 over 108. Eye grounds showed marked neuroretinitis. Urine: Acid, specific gravity 1.011, albumin heavy trace, many hyaline and granular casts. Blood urea nitrogen 45, creatinin 4.5. The condition progressed rapidly. Specific gravity of urine fixed between 1.004 and 1.012, with trace of albumin, hyaline and granular casts. Occasional showers of red blood cells. Blood urea nitrogen varied between 37 and 80 mg for 100 cc of blood. September 14, 1933, patient died in uremia. At autopsy a contracted kidney was found. Microscopic diagnosis: Chronic glomerulonephritis.

Nephrosis—Syn.—Tubular nephritis, degenerative Bright's disease, nonhemorrhagic nephritis.

Fredrick Muller introduced the term nephrosis in 1905 to indicate tubular degeneration. Since that time many students of nephritis have applied the term to many conditions in which edema and albuminuria were the chief symptoms. Four types of nephrosis may be recognized:

(*a*) *Toxic nephrosis.*—This type occurs chiefly in cases of pneumonia, typhoid fever, and syphilis.

(*b*) *Chemical nephrosis.*—This type results from the injection of heavy metals as mercury, potassium, silver, and bismuth.

(*c*) *Amyloid nephrosis.*—This type occurs in chronic infectious states as osteomyelitis and tuberculosis.

(*d*) *Lipoid nephrosis.*—This condition has been a subject of dispute. Many authorities doubt if such a condition as lipoid nephrosis ever occurs. Christian does not include it in his classification of nephritis. The clinical findings of lipoid nephrosis are so closely allied to subacute glomerulonephritis that if these cases are followed over a long period of time they usually turn out to be subacute glomerulonephritis. Bannick reports 30 cases in which clinically a diagnosis of lipoid nephrosis was justified, but in subsequent examinations 7 developed chronic glomerulonephritis, 4 patients died from uremia.

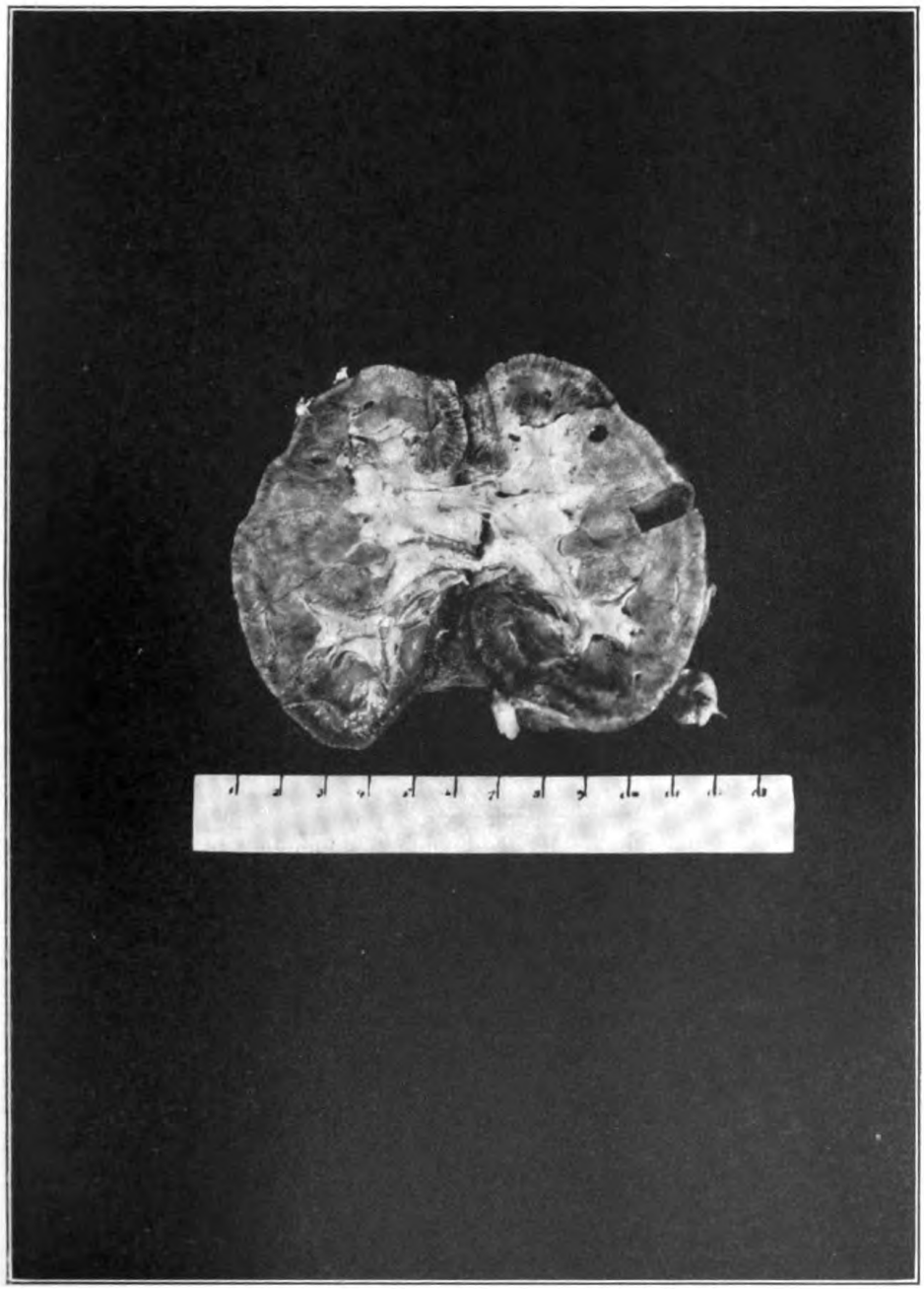
The distinguishing feature between subacute glomerulonephritis and nephrosis is the finding of red blood cells in the urine. Epstein laid stress on the finding of the bright doubly refractive bodies (ester of cholestrol) in the urine. These are also found in subacute glomerulonephritis.

Pathology—Gross.—The kidney is large and pale. Spoken of as the large white kidney, this kidney cannot be distinguished from the kidney removed from a case of subacute glomerulonephritis. The capsule strips with ease, leaving a smooth surface. The cortex is swollen and pale; frequently yellow areas may be seen.

Microscopic.—The tubules show marked degeneration. The tubular cells contain fat droplets. The glomerular capillaries show some changes but only show partial obstruction. The proliferation, hyalinization found in subacute glomerulonephritis is not present.

Case no. 3. J. H. R., machinist's mate first class, age 30 years, admitted September 6, 1933, with diagnosis arthritis, acute, right foot. Chief complaint: Pain and swelling, right foot and ankle. Family history: Irrelevant. Past history: Usual childhood diseases, appendectomy 1925, tonsillectomy 1926, iritis right eye 1931. No history of previous kidney disease. Physical examination: Essentially negative except for swollen right foot and knee. Blood pressure 144 over 90. Temperature 98.8 degrees, pulse 96, respiration 20. Urine: Cloudy, neutral reaction, specific gravity 1.010, albumin 3 plus, sugar negative, leucocytes few. Red blood count 3,600,000, Hgb. 77 percent. White blood count 10,400. Differential: Polys. 72 percent, lymph. 26 percent, mono. 2 percent. Kahn negative. Blood urea nitrogen 11. The patient gradually developed renal failure. October 12, 1933, 800 cc of fluid removed from abdomen. The specific gravity of the urine was fixed between 1.008 and 1.014 and contained albumin 3 to 4 plus. October 13, 1933, patient died. At autopsy a large white kidney was found. Microscopic diagnosis: Amyloid nephrosis.

Case no. 4. F. C., fireman first-class, age 28 years, admitted December 7, 1933. Diagnosis, poisoning neoarsphenamine. Chief complaint: Nausea and vomiting, pain in stomach. Family history: Irrelevant. Past history: Chancroid 1926, tonsillitis acute 1926, and syphilis 1927. Present illness: 24 hours before



SMALL WHITE KIDNEY OF CHRONIC GLOMERULAR NEPHRITIS.

admission received his fourth injection of neoarsphenamine; 1 hour following injection patient suffered from pain in legs, back, hands, and feet. Physical examination: Icteric tinge on conjunctivae, heart essentially negative, liver enlarged to 3 fingers below the costal margin. Pulse of good quality and regular. Blood pressure 130 over 60. W. B. C. 8,000, polys. 61, lymphs. 26, mono. 3, eos. 8. During first 4 days patient passed only small amounts of urine. It was highly colored, specific gravity 1.024, albumin 4 plus, many hyaline and granular casts. December 14, 1933, patient markedly edematous unable to retain nourishment. Blood urea nitrogen 100, creatinin 7.5. Urine, alkaline, specific gravity 1.011, albumin 4 plus. Patient gradually became worse and died on December 18, 1933. At autopsy a large white kidney and a large liver were found. Microscopically the picture of nephrosis was present. The liver showed fatty degeneration.

Arteriolosclerosis—Syn.—Essential hypertension, malignant hypertension, chronic interstitial nephritis, and primary contracted kidney. This condition is a primary affection of small arterioles of the kidney, spleen, pancreas, brain, retinae, and heart. The condition is referred to as essential hypertension because usually the primary finding is high blood pressure, which may be present for a long time without any symptoms and may be discovered accidentally during routine examination. It is referred to as malignant hypertension when there is cardiac, renal, or cerebral damage. Benign or malignant hypertension are subdivisions of the same disease. This condition is primarily vascular. Persons afflicted with arteriolosclerosis may run a benign course for years and finally develop renal insufficiency, cardiac failure, or suffer a cerebral accident, depending on the organ which is most affected. In about 20 percent of the cases the kidney function is impaired, about 8 percent die in uremia.

Etiology.—The etiology of arteriolosclerosis is unknown. It is possibly a hereditary condition.

Pathology—Gross.—The kidney is usually smaller than normal. The capsule strips with ease, leaving a granular surface. The cut surface shows the cortex thinner than normal and irregular.

Microscopic.—The striking thing is the intimal thickening of the afferent arterioles. Some of the afferent arterioles are completely occluded. Changes in the glomeruli and the tubules vary according to the extent of the occlusion of the arterioles. Some of the glomeruli show various stages of atrophy to complete hyalinization. The tubules show proliferation, degeneration, atrophy, and complete disappearance. There is a great increase of fibrous tissue.

Case no. 5.—J. C. H., supernumerary, age 23 years, admitted December 8, 1932. Chief complaint: Convulsions, headache, and weakness; poor vision. Past history: Irrelevant. Family history: Negative. Present illness: Yesterday following a nap he noticed that his vision was poor and suffered from a severe headache. Physical examination: Heart slightly enlarged, blood pressure 200 over 130, pulse full and regular, marked neuroretinitis. Urine; Reaction acid, specific gravity 1.005, albumin 3 plus, hyaline and granular casts. Blood urea nitrogen 150 mg per 100 cc of urine, creatinin 10 mg per 100 cc of blood. R. B. C. 3,370,000, hgb. 50 percent. Kahn negative. During stay in

the hospital patient became totally blind, blood pressure 230 over 160, the heart became markedly enlarged and began to fail. The specific gravity of the urine was fixed between 1.012 and 1.005. Traces of albumin and granular casts were constantly present in the urine. April 26, 1933, patient died a cardiac death. At autopsy a granular contracted kidney was found. Microscopic diagnosis: Arteriolosclerosis.

Case no. 6.—C. W., Veterans' Administration patient, admitted February 6, 1933. Diagnosis: Nephritis chronic. Chief complaint: Dizziness, swelling of feet and legs, periodic difficulty in breathing. Family history: Aunt suffering from kidney trouble. Past history: Negative. Present illness: Has had asthmatic attacks all his life, recently he has had 6 to 8 attacks daily. For past 8 months he has suffered from headache and fatigue. His feet became swollen at times. Physical examination: Well developed, cyanotic and dyspnoeic. Râles in dependent portions of the lungs. Heart enlarged, apex beat diffuse, systolic blow present, pulse 100, full and regular. Blood pressure 230 over 154. Urine: Acid reaction, straw colored, specific gravity 1.012, albumin 2 plus, coarse granular casts present. P. S. P. first hour 15 percent, second hour 10 percent. R. B. C. 3,250,000, hgb. 79 percent. Urea nitrogen 27 mg per 100 cc of fluid. Creatinin 2.5 mg per 100 cc of blood. Electrocardiograph showed marked cardiac damage. Mild neuroretinitis present. The specific gravity was fixed between 1.007 and 1.014, with 1 plus to 2 plus albumin. Hyaline and granular casts were present. The blood pressure remained around 224 over 156. The heart gradually failed and on May 22, 1933, patient died a cardiac death. At autopsy a granular contracted kidney was found. Microscopic diagnosis: Arteriolosclerosis.

The differential diagnosis of nephritis depends on a careful history, physical and laboratory examination.

Glomerulonephritis acute is characterized by slight edema usually of the eyelids, fever, and slight rise in blood pressure. The urinary findings are oliguria, high specific gravity, highly colored, albumin, casts, and red blood cells.

Glomerulonephritis subacute is characterized by edema which is usually marked and generalized. Edema may be present without being observed clinically; the way to determine the presence of edema is by weighing the patient from time to time. The urinary findings are marked albuminuria, casts, red blood cells, which may be absent over long periods of time. The blood shows hypercholesteremia; nitrogen retention is not present. The concentrating power is only slightly impaired.

Glomerulonephritis chronic is characterized by renal insufficiency. The concentrating power of the kidney is greatly impaired. Albumin and casts are present in small amounts, anemia of secondary type is present. The blood pressure is elevated. Increased nitrogen retention develops, neuroretinitis is of grave import.

Nephrosis is characterized by edema usually marked and generalized, marked albuminuria, urine of high specific gravity, and large numbers of casts of all kinds. The blood cholesterol is high. There are no red blood cells present in the urine, which distinguishes this type of kidney condition from subacute glomerulonephritis.

KIDNEY TUBULE CONTAINING A CAST.

ARTERIOSCLEROSIS IN A BLOOD VESSEL OF THE KIDNEY.

Differential diagnosis of nephritis

Type	Glomerulonephritis			Nephrosis	Arteriolosclerosis
	Acute	Subacute	Chronic		
Age	Under 40 years	Under 40 years	Under 40 years	Under 35 years	30 to 65 years.
Course	Acute	Subacute	Chronic	Acute or chronic	Chronic.
Edema	Slight; face and ankles	Usually generalized, marked	Absent	Usually generalized, marked	Absent.
Blood pressure	140 systolic	140 systolic	High, 160 systolic plus	Normal	High, 160 to 200 systolic plus.
Retinitis	Absent	Absent or slight	Present	Absent	Present.
Heart	Normal	Normal	Enlarged	Normal	Enlarged.
Anemia	Absent	Absent	Present	Absent	Absent or slight.
Urine volume	Oliguria	Oliguria or polyuria	Polyuria	Decreased	Polyuria.
Specific gravity	High	Variable	Low and fixed	High	Low and fixed.
Albumin	2 to 4 plus	2 to 4 plus	1 to 2 plus	2 to 4 plus	1 plus to minus.
Casts	All types	All types	Present	Occasional	Occasional.
Red blood cells	1 to 3 plus	Showers	Showers	Absent	Absent.
Lipoid bodies	Absent	Present	Absent	Present	Do.
Nitrogen retention	Absent or slight	Absent or slight	Present	Absent	Present.
Blood cholesterol	Normal	Increased	Normal	Increased	Normal.
Albumin-globulin ratio	Normal, 3 to 1	Reversed	do	Reversed	Do.
Serum proteins	Normal	Low	do	Low	Do.
Indican	1 to minus	2 plus to minus	Minus to 4 plus	Minus	Minus to 4 plus.
P. S. P. excretion	Low	Variable	Low	Normal	Decreased.
Urea clearance	Variable	do	Falls gradually	Variable	Falls gradually.
Dilution concentration test.	Variable, usually high and fixed.	Variable, depending on edema.	Specific gravity, 1.002 to 1.020; unable to concentrate.	Normal	Specific gravity, 1.002 to 1.020; unable to concentrate.
Uremia	May occur	May occur	Most cases	Never	10 percent of cases.
Prognosis	95 percent recover	90 percent become chronic	Poor	Poor	Poor.

Arteriosclerosis is characterized by high blood pressure, enlarged heart, polyuria, traces of albumin with occasional casts. The concentrating power is good until the late stages. Nitrogen retention is present when the kidney begins to fail. Neuroretinitis is usually present.

In interpreting the various kidney function tests the large amount of reserve tissue present in the kidney must be kept in mind. It has been estimated that from 60 to 70 percent of the kidney parenchyma must be destroyed before we are able to determine any positive findings from the various kidney function tests. Cases with marked kidney destruction may be able to carry on a useful occupation, the only findings being an elevation of the blood pressure, enlarged heart and polyuria, the specific gravity being low and fixed, the kidney being referred to as a compensated kidney.

In conclusion it might be stated that during the past 100 years little has been added to the classical description of nephritis as presented by Richard Bright, although a great deal of work has been done. The etiology remains an unsolved problem. The clinical picture takes in many factors foreign to the kidney so that a clinical classification is impossible. A pathological classification appears to be the most satisfactory.

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A METHOD OF LOCAL ANESTHESIA FOR INTRANASAL OPERATIONS

By Franklin F. Lane, Lieutenant Commander, Medical Corps, U. S. Navy

The following method of inducing local anesthesia for intranasal work is not original and may not be new to many surgeons. It is reviewed for the purpose of inducing those who have never tried it to do so.

The rationale of the procedure is best set forth by a review of the nerve supply of the mucosa and the underlying cartilagenous and bony structures of the nose. The technic of the procedure can then be described in detail with a better understanding of the reasons for its effectiveness.

Nerve supply: The fifth cranial nerve is the sensory nerve to the nose.

The first or ophthalmic division of the trifacial nerve divides into three branches, one of which—the nasociliary nerve, runs forward on the superior surface of the lamina cribrosa of the ethmoid bone and enters the nasal cavity from above through the nasal fissure as the *anterior ethmoidal* nerve. The nasal fissure lies at the most anterior superior portion of the nasal cavity, i. e., anterior to the cribiform plate of the ethmoid. As the *anterior ethmoidal* nerve emerges from its canal, it divides into two terminal branches. One to the lateral nasal wall called the lateral branch and a medial branch to the septum. The first supplies the anterior one-third of the lateral intranasal wall including one-third of the middle turbinate but very little, if any, of the inferior turbinate bone. The medial branch supplies the anterior one-half of the septum.

The second or maxillary division of the trigeminus has numerous subdivisions, one of which is the infraorbital nerve supplying the mucosa lining the maxillary antrum. However, the more important branches of the second division from the standpoint of the present discussion are the two sphenopalatine nerves from which hangs suspended the sphenopalatine ganglion or Meckel's ganglion in the pterygo-palatine fossa which is situated just posterior and lateral to the posterior attachment of the middle turbinate. Arising from this ganglion are 7 nerves, 6 of which supply all of the naso-pharynx, the hard and soft palate and part of the palatine or faucial tonsil. These are the anterior, middle, and posterior palatine, the posterior superior lateral nasal, the nasopalatine, and the pharyngeal.

In this discussion we are interested in only 3 of the 6.

(1) The anterior palatine gives a terminal branch, the *posterior inferior lateral nasal,* which supplies practically the entire inferior turbinate.

The other 2 nerves of this group of 3 enter the nose through the sphenopalatine foramen.

(2) The *posterior superior lateral nasal* innervates the balance of the lateral nasal wall, i. e., the posterior two-thirds, except the inferior turbinate.

(3) The *nasopalatine* nerve emerging from the sphenopalatine foramen courses medially over the roof of the nasal cavity. When it reaches the septum it turns downward and forward along the vomer to the incisive foramen at the anterior part of the hard palate. Passing through this opening it runs backward on the roof of the mouth to anastomose with the palatine branch of the anterior palatine. This nerve supplies therefore: The roof of the nasal cavity; the posterior one-half of the septum and the floor of the nose.

The blood supply of the nasal cavity is from arteries accompanying, with few exceptions, the nerves of the same name.

In résumé it will be seen that a cotton wound applicator saturated with anesthetic and placed in the vault of the nose as far anteriorly as possible will cover the nasal fissure—the exit of the anterior ethmoidal nerve as it divides into its lateral and medial branches supplying respectively, the upper two-thirds of the anterior one-third of the lateral wall and the anterior one-half of the septum.

Another cotton wound applicator placed just behind the posterior tip of the middle turbinate will be in the region of the sphenopalatine ganglion which controls the balance of the innervation of the nose plus the hard and soft palate and will anesthetize everything but the frontal sinus and the external walls of the antrum.

Technique of anesthesia.—The preparation of the patient plays an important part in the ease and speed with which an operation may be performed because the operator is saved the time consuming interruptions from fainting, dizziness, and vomiting.

Preparation.—Where time permits, the patient is kept under observation for a few days, while the laboratory tests are made and any incipient acute rhinitis or bronchitis has time to develop. If the coagulation time is more than 3 minutes, a course of calcium lactate gr. XX, T. I. D., is instituted and continued until the coagulation time is reduced to 3 minutes. The night before operation, the patient is given a course of calomel, and salts follow the morning of the operation. No breakfast, except occasionally, a cup of black coffee is allowed if the patient complains too bitterly of hunger.

Twenty minutes before the patient is brought to the operating room barbital, grains X, is given. This calms the patient and the effect lasts for several hours. It does not give the nausea sometimes associated with morphine and, most important of all, where cocaine is used, barbital or luminal, acts as a direct antagonist to cocaine poisoning, while morphine and atropine are synergistic in their depressant action on respiration and the heart.

Using barbital, now that the preferable luminal is not on the supply table, there is no vertigo or fainting due to the cocaine itself and no feeling of oppression in the chest and no desire for "open windows". The patient may perspire profusely at some period of the sitting and is often quite talkative and shows other signs of mental stimulation, but unless the sight of blood or the sound of the scraping and breaking of bone causes the patient to faint, the operation will not be disagreeable to either the patient or the operator.

Induction of anesthesia.—With the subject in the sitting posture, anesthesia is started by a series of sprays from a hand atomizer First adrenalin, 1:1,000, immediately followed by cocaine, 5 percent. This is repeated about four times, 2 minutes apart. The entire nasal cavity, above and below, on each side is covered by as thin a film of each as is possible, and the patient cautioned against swallowing any of the solution. Between sprays the cilia are clipped short, the sinus areas watched for discharge and the variations from normal of the different structures observed as the mucosa shrinks.

Four metal applicators, cotton wound with well-protected tips and large enough to remain in place after introduction are prepared and saturated with a solution of equal parts of adrenalin, 1:1,000, and cocaine, 10 percent. Two of these are introduced, one behind the posterior tip of each middle turbinate or as near to that point as any nasal deformity present will permit. Properly placed the handles of these applicators will cross each other about 2 inches in front of the nose.

The two remaining applicators are introduced into the vault of each nares as high and as anteriorly as they will go without injury to the cribiform plate of the ethmoid. The tips of these applicators should be especially well protected by cotton and only enough force used to introduce them, as will wedge them firmly in place. The amount of cotton on each applicator may have to be changed until a good fit is obtained. The applicators remain in place while the operator is scrubbing and being gowned, 10 to 20 minutes, depending upon how close to Meckel's ganglion, with the deformities present, it has been possible to place them. The greater the distance from the ganglion, the longer the time, but 20 minutes is usually about the effective time limit. The applicators are removed and the operation commenced immediately.

Cocaine crystals moistened with adrenalin used the same way gives the same and a quicker result, but increases the probability of poisoning. The same applies to 20 percent or 10 percent cocaine. The longer time with 5 percent cocaine is well spent from all standpoints.

Result.—With the applicators properly placed, a perfect anesthesia and a bloodless field is obtained for submucous work and a perfect

anesthesia and a very minimal bloody field is present for the other procedures on the lateral wall. This anesthesia is not effectual for frontal sinus work, except the nasal portion of the naso-frontal duct and is not necessary for antrum puncture where a cocainized applicator under the inferior turbinate is sufficient.

Discussion.—This method is far superior to the method of cocaine on cotton packs. The latter requires over half an hour for induction. These packs cover a very large surface which increases the absorption of the cocaine, increasing the chance of poisoning and does not reach much of the tissue beneath the surface usually resulting in a very poor anesthesia and a bloody field.

The applicator method described is superior in that: (1) If there is not too much deformity of the middle turbinate or septum to interfere with the accurate placing of the applicators, 10 minutes' time will give a perfect anesthesia, lasting about an hour. When not so closely applied to the ganglion and allowed to remain longer, 50 percent will be painless until the region of the vomer is reached and 50 percent will have more or less discomfort all of the time but there is never the excruciating pain which interferes with the operation and causes the operator to stop before completion of the work.

(2) It is flexible, for example, in submucous resection; when the deformity is so marked as to occlude the nares on one side, an applicator in the vault anteriorly on this side with the two in the opposite side will allow the cartilagenous portion to be removed. This opens the nares and allows secondary anesthesia of Meckels' ganglion on the occluded side. For removal of the anterior tip of the middle turbinate, one applicator in the vault on the same side is sufficient.

(3) The four cotton wound applicators present only a comparatively small area for the absorption of cocaine, thus cutting down the probability of poisoning.

(4) They are applied at the spots which will anesthetize the nerves supplying the deep as well as the superficial structures.

(5) The arteries accompanying the nerves are also constricted as they enter the nasal cavity, so that bleeding is very much less. In submucous work, if the original incision over the anterior portion of the cartilage extends too far downward and laterally, the vessels in Kiesselbach's area will cause some troublesome hemorrhage and occasionally some aberrant vessels around Jacobson's cartilage in the floor of the septum may be quite annoying if the anesthesia is not quite perfect; but otherwise the field is practically bloodless and no time is spent sponging. It will be found that in all other intranasal work the hemorrhage is less than with other types of anesthesia.

(6) The effect on the patient, therefore, is better, not only from the standpoint of reducing the chances of cocaine poisoning but also from the standpoint of freedom from pain, increased cooperativeness,

and increased confidence in the operator. They are particularly surprised not to see any blood and greatly pleased that there is no pain.

(7) The effect on the operator is threefold:

(*a*) Greater ease in working.

(*b*) A better and more complete operation.

(*c*) Shorter operating time.

A complete removal of the entire septum may be accomplished in 25 to 30 minutes, no matter how bad the deformity, taking plenty of time around the angles so as to prevent tears. With the less severe deformities, where there is little likelihood of tearing, 15 to 20 minutes is quite sufficient for a complete removal of the cartilagenous and bony septum.

(8) As a result, the surgeon can perform a greater number of submucous resections or other intranasal operations during any one operating period. This is of importance in a very active service with many daily admissions and a small staff. An assistant can easily be trained to carry out this method of anesthesia. If, when the operator commences work on the first case, the sprays are started on the second patient, the latter will be completely anesthetized by the time the surgeon has finished the first operation and is rescrubbed and regowned.

(9) For those with a known or suspected idiosyncrasy for cocaine, the preparations "Pantocaine 2 percent and suprarenin 1:1000" in equal parts, serves as a good substitute. However, the anesthesia is not quite so complete, as a rule, and the operative field is more bloody when these drugs are used.

THE RUSSELL TREATMENT OF FRACTURES OF THE FEMUR [1]

By HERMAN A. GROSS, Lieutenant (junior grade), Medical Corps, United States Navy

This method of treatment of fractures of the femur has been used by Dr. R. H. Russell (1) and his colleagues for the past 19 years at the Alfred Hospital in Melbourne, Australia, and for the past 17 years at the children's hospital in the same city.

Thomas J. Ryan (2) of Philadelphia in 1927 published a series of eight cases treated by the Russell method. He added nothing to Dr. Russell's original article except that he went into a detailed discussion of the physics of the mechanics involved and dealt with the anatomy of the thigh, the physiology of the muscles, and the physiology of new bone formation.

Blum (3) of New York City has applied the principle of double-pulley traction in a case of fracture of the humerus.

[1] Read before staff meeting, Naval Hospital, New York.

Attention was first called to this form of treatment while Lt. Comdr. F. R. Hook, M. C., U. S. N., and the writer were taking a special course in fracture work given by the American College of Surgeons several years ago.

We were very favorably impressed by the simplicity of the apparatus, the comfort of the patient, the apparent ease of reduction and retention, and most of all by the lack of atrophy of muscles which is so noticeable when plaster-of-paris casts are used.

It was not long before we had an opportunity to use this method of treatment, and the results obtained, in my opinion, are as good as could be obtained by other methods of reduction and retention. I feel that it should be listed as one of our better methods of treatment.

Someone once said that the ideal way to treat a fracture was to wish the fragments into position and hold them in their proper place with moral persuasion while the patient goes on with his regular work. The Russell method does not quite do this but it comes as close to the axiom as could be expected.

The literature on the subject is so meager, the writer having found only the original article and two references, that failure to quote Dr. Russell on the physiology and the mechanics of the reduction apparatus would be an injustice to his masterly monograph. Nothing can be added:

"Let us suppose a patient has just been admitted to the hospital with a fracture in the middle of the femoral shaft. *The thigh is shortened. Why?* The shortening is caused by the tonic contracture of certain long muscles that are attached above to the pelvic bone, and traverse the entire length of the thigh to be inserted into the head of the tibia and fibula. Of these there are two opposing sets, consisting in the main of the hamstrings posteriorly and the rectus femoris anteriorly. The other numerous muscles which are attached to the femur itself play little if any part in the production of the shortening, and for the sake of clearness we will disregard them.

"Muscular tone (which must always be carefully distinguished from muscular action) is a physiological property of living muscle, which, for practical purposes, causes the muscle to behave like rubber bands slightly stretched. Their correct length is maintained by the length of the femur, and as soon as the femur is broken they shorten and produce the overriding of the fragments. Here the analogy ends, for rubber once released from its tension will have no further power to contract, whereas the tonic shortening of the muscles will be progressive; hence the excessive shortening, amounting to 3 or 4 inches, that almost invariably complicates an ununited fracture of the femur.

"Our first aim, then, is to pull out these muscles to their correct length, and when we have accomplished this we may be sure that every other structure in the thigh will be in its correct position in-

cluding the fragments. This does not mean exact anatomical position as this is not always necessary.

"*What must we pull on?* Clearly the tibia and fibula, seeing that the muscles are attached to them. We use adhesive plaster and an 'Ace' bandage to maintain its proximity to the skin—only after the leg has been well cleansed and shaved. We do not carry the plaster above the knee for reasons which are quite obvious. The practice, which is quite in vogue, of carrying the strapping up the thigh indicates some confusion in our mental picture of the object to be obtained. Anxiety as regards the knee seems also to be felt; but this is quite needless, for the ligaments being attached to a fragment cannot be subjected to stretching. The whole of the traction force will fall upon the muscles, none at all on the ligaments; to convince ourselves we have only to reflect that if the muscles were severed at the site of the fracture the limb would drop off.

"*How must we pull on the tibia and fibula?* If we merely attach a weight to the leg to pull the thigh muscles out, it is obvious that the thigh and leg will have to be in a straight line or the thigh muscles cannot be extended. But this would never do because it would be intolerably uncomfortable; and perfect comfort is the first essential requirement in any appliance for the treatment of a fracture. We *must* have the knee slightly bent; but the bending of the knee is incompatible with the necessary pull on the thigh muscles that are attached to the tibia and fibula. This seems a bit inconsistent, but the following manipulation will help make the point clear.

"In the case of a fractured femur how does the surgeon manipulate it in order to draw out the thigh muscles? He will do it in the folfowing manner: Standing by the side of the bed he passes his left hand under the knee, the right hand grasps the leg just above the ankle. Now he gradually exerts a little power, the right hand pulling horizontally toward the foot of the bed, the left hand up toward the ceiling mostly, but with a slight inclination footward also. The limb will not come out to its proper length all at once, but the patient will feel more comfortable and will instinctively know that his limb is being skillfully and properly handled. The surgeon now reasons thus: 'I am sure that this is the right way to get the thigh out to the proper length if only the patient's thigh muscles were quiescent; but they are not, owing to the patient's apprehension and fever. Were I able to stand here doing this for hours or until he sleeps, then there would be no difficulty; but obviously this is impossible. I must then devise some means of doing what I am now doing; something that will not tire, that will make the limb absolutely comfortable and in that way favor the return of mental quietude.'

"*Apparatus.*—The arrangement shown in figure 1 was evolved in the manner just described: A sling beneath the knee corresponding

to the surgeon's left hand, and horizontal traction on the leg corresponding to the surgeon's right hand. The arrangement provides that the pull on the leg shall be nominally double the upward lift at the knee although actually modified by friction between the pulleys and the cord" (*1*).

Special apparatus.—1. Ordinary fracture bed with an overhead longitudinal bar which can be shifted laterally.

2. An arrangement to which may be attached a couple of pulleys beyond the foot of the bed. These pulleys should be in an horizontal line with the foot of the patient when the leg is lying horizontally on a pillow with the heel just clear of the bed.

3. Four block pulleys and flexible cord.

4. Two bed blocks to raise the foot of the bed 12 inches.

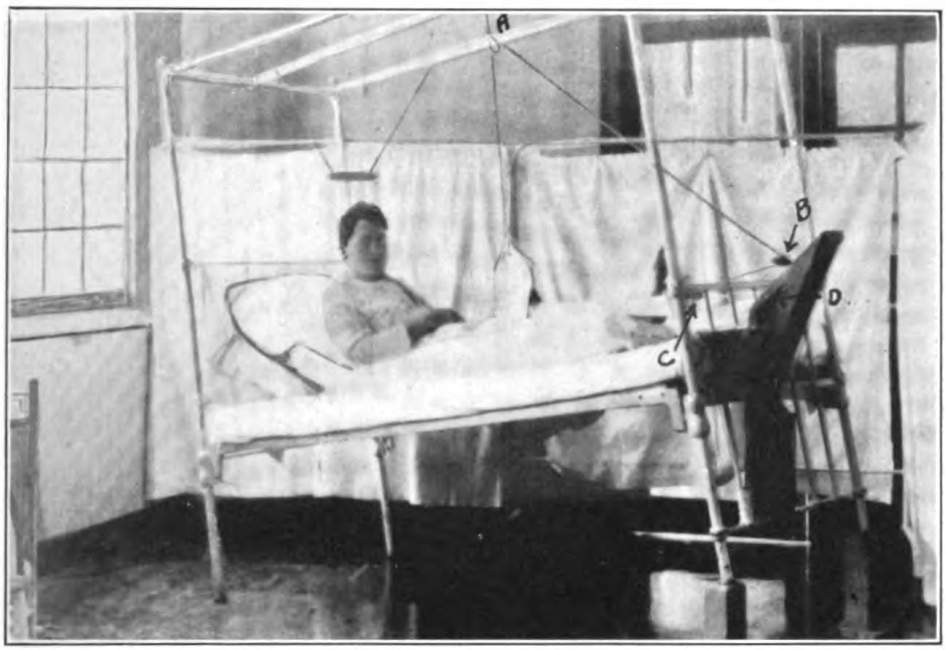
Application of the apparatus.—The leg is entwined with spiral adhesive made by overlapping the anterior and posterior portions of the triple-tailed adhesive plaster at the lower end of which the spreader is placed. The spreader is approximately 5 inches wide so that there will be no pressure on the malleoli. The leg is then snugly encased in an "Ace" bandage. The sling is now passed beneath the knee which all this time has been lying comfortably on a pillow. The sling should be broad and soft and the popliteal space should be well padded. The ends of the sling are now securely tied together with the cord which is then passed through pulley "A" which should be directly above a point about 1 inch distal to the tibial tuberosity, to pulley "B" beyond the bed, back to pulley "C" on the spreader, back to pulley "D" and then the weight is applied. (See fig. 1.) The soft pillow is adjusted under the thigh to prevent gravitational sagging at the site of the fracture. The heel must not be touching the bed and another soft pillow is arranged under the leg and Tendo Achilles to prevent it from doing so.

The foot of the bed is elevated 12 inches so that the weight of the body will give greater counter traction.

The knee sling prevents inversion and eversion. Eight pounds of weight are used in the adult and at the end of the third week are reduced to 5 or 6 pounds.

In common with all other traction and suspension methods this needs constant attention, daily measurements and frequent check up with the X-ray.

The thigh muscles are being extended by a combination of forces. Figure 2a makes evident the mode of action of the two forces employed by construction of a parallelogram of forces. It will be seen that the resultant of the forces lies in the line of the thigh. Figure 2b shows the resultant of the forces if the knee is not flexed and if the pull at the knee is greater than that applied to the leg. The resultant of the forces is not in the line of the thigh.



BED EQUIPPED FOR TREATMENT OF FRACTURE OF THE FEMUR.

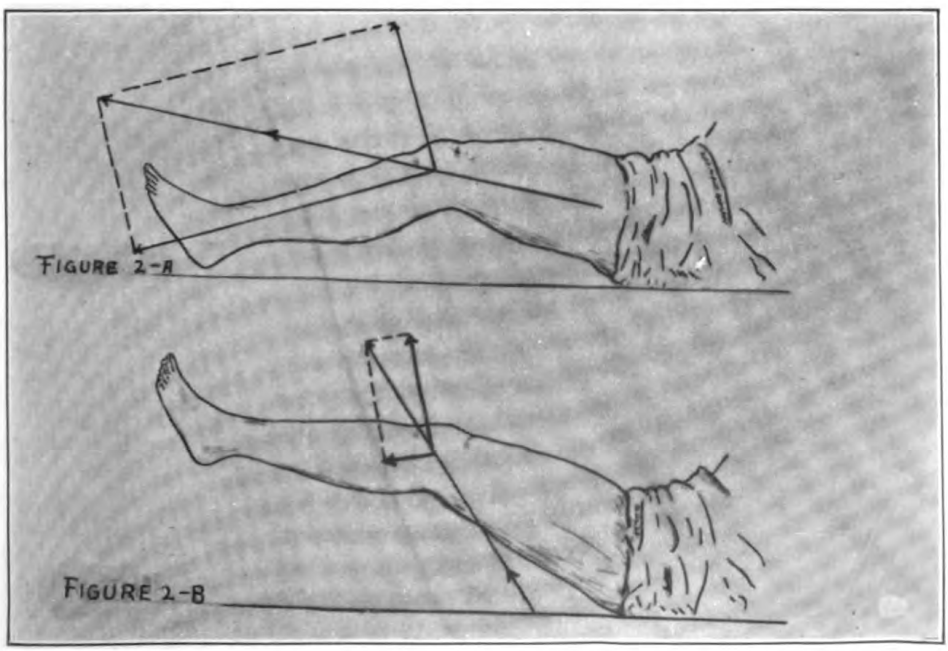


DIAGRAM TO SHOW DIRECTION OF TRACTION IN FRACTURE OF THE FEMUR.

It is evident in the following cases that the general behavior of the fractures treated contrasts favorably with those treated with other retentive measures. The nutrition of the tissues of the limb, notably the muscles and bone, was better preserved when merely laid on a pillow with considerable freedom of movement than when subjected to the compression associated with casts, splints, and bandages.

The patients were happy—their nursing care was facilitated, they were out of bed in the fourth to fifth week, and encouraged to use the limb in active motion, and were gradually worked up to the point of weight-bearing.

The Russell technique and the apparatus required are unique in their simplicity. All of the paraphernalia such as Thomas splints, Pierson attachments, numerous weights and counterweights and lines, and the care to see that they are in proper working order are eliminated.

DISCUSSION

To estimate the end result three factors must be taken into consideration: (After Moorhead).

1. Function—capacity to perform.
2. Union—state of repair.
3. Contour—external appearance.

Function (F)+Union (U)+Contour (C)=End result.

As an arbitrary valuation, we allot as follows:

F perfect=60 percent.
U perfect=20 percent.
C perfect=20 percent.

In case 1 we may make the following equation:

Function	40 percent
Union	15 percent
Contour	15 percent
End result	70 percent

In case 2 we may make the following equation:

Function	60 percent
Union	20 percent
Contour	20 percent
End result	100 percent

SUMMARY

1. A description of the technique of the Russell treatment of fractures of the femur is presented.

2. Two cases treated by this method are reported.

3. In cases of fracture of the lower third of the femur an additional sling under the upper end of the distal fragment with the line of force

of 3 to 5 pounds acting at 90° to the long axis of the femur is advocated.

4. The early use of the Thomas walking caliper to stimulate early callus formation and earlier active use of the limb is advocated.

5. The end result in both cases is computed.

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FOUR INTERESTING SURGICAL CASES

By K. E. Lowman, Lieutenant Commander, Medical Corps, United States Navy

We are presenting in this paper the histories of four interesting surgical cases which have occurred on the Surgical Service at Quantico during the past 10 months. The first case is that of Mrs. L. E. S., age 21 years, wife of a first sergeant, U. S. M. C. During a period of approximately 20 months she underwent two caesarean sections, the first January 6, 1932, the second December 20, 1932, and the third operation, that for tubal pregnancy, performed on our service, was done August 19, 1933. The remaining three cases are all perforated gastric ulcers, all operated, the first on December 31, 1933, the second on February 2, 1934, the third April 3, 1934, and fortunately, all have recovered. It is a bit noteworthy that these three cases presented themselves in a period of shortly over 3 months.

To return to our first case, the first caesarean section on Mrs. L. E. S. was performed January 6, 1932, because of deformed pelvis of mother due to pelvic fracture sustained in 1929. The patient stated that this diagnosis was confirmed by X-ray. At the time just prior to the performance of this operation, X-ray showed distinct deformity of the right pelvis so contracting the parturient canal as to make normal delivery impossible. At operation a classic caesarean section was performed by Capt. Reynolds Hayden (M. C.), United States Navy, and a normal female child weighing over 9 pounds was delivered from the uterus. The mother made an uneventful recovery from the operation.

The second caesarean operation was successfully done, also by Captain Hayden, a little over 11 months later, and another normal child delivered. At this operation rather heavy omental adhesions

to the anterior abdominal wall and uterus were found as a result of the first caesarean. Recovery was uneventful.

On August 18, 1933, almost exactly 8 months after the second caesarean, the patient was again admitted to the Family Hospital at Quantico complaining of intense pain in the right lower abdominal quadrant. About 1 month previous to this admission she had noticed pains low down on the right side of the abdomen. On July 9, 1933, after her menstrual period, she was in bed 2 days as a result of this pain. No nausea or vomiting. Menstruated during most of the latter part of July, until July 25, 1933, when menstruation ceased. She felt that during the past 3 days the pain had become much worse. It was so severe last night that she was unable to sleep.

Bi-manual examination reveals a palpable mass in the right lower quadrant which is about the size and shape of an ordinary sized lemon. Abdominal palpation shows soreness and tenderness over appendiceal area and also lower down in the right pelvis. Muscular rigidity noted but not very marked. Physical examination otherwise essentially negative. Urinalysis negative; W. B. C. 10,400, 64 percent segmented and band forms.

A preoperative diagnosis of probable tubal pregnancy and possible appendicitis acute was made, chiefly on the history and the physical findings. At operation the following morning, an acutely inflamed appendix and a large fairly tense indurated right fallopian tube measuring about 9 by 5 by 2½ cm, were removed and the tubal mass sent to the United States Naval Medical School for pathological examination. Many adhesions and dense ones were noted. Free pus was also found in small quantity and pocketed between the right tubal site and the right ovary. Drainage was therefore instituted. It was impossible on account of the many dense adhesions to tie off the left tube after having removed the right and deemed inadvisable also to remove the left tube through another incision, which would necessitate further operative procedure and possibly unnecessary risk to the patient's welfare.

The drainage tube was entirely removed after 48 hours. Sutures were removed on the seventh day from a clean wound, and she left the hospital on the fourteenth day after an uneventful recovery. This case has been cited because such a history over a period of approximately 20 months is considered fairly noteworthy. The examination of the tubal mass sent to the United States Naval Medical School for examination revealed ectopic pregnancy.

The first ruptured gastric ulcer was admitted to the post hospital as an emergency case on the morning of December 31, 1933. Physical examination disclosed an adult male, Mr. T. C. H., civilian employee, about 60 years of age, and who was evidently quite ill. There was intense pallor of the face. The abdomen was scaphoid in shape,

with all muscles absolutely and characteristicly boardlike in their rigidity. He gave a history of sudden terrific upper abdominal pain about 12 hours previous to admission. The abdomen was tender all over, but particularly so in the right upper quadrant and epigastrium. There was no history of recent dietary indiscretion, nor had the patient indulged in any large amount of alcoholic liquor at any time during his life. A preoperative diagnosis of perforated ulcer, probably gastric, was made.

At operation, which was performed immediately after admission, a perforation was found in the anterior wall of the stomach, the opening being just about the size and shape of a 10-cent piece. Quite a large amount of gastric contents was free in the upper abdomen.

The opening was closed by a purse-string suture, the bites of which were taken deeply and firmly in healthy tissue beyond the characteristic indurated and more or less friable tissue of the ulcer wall surrounding the perforation. This closure was doubly reinforced by two rows of Lembert sutures. As soon as this procedure was finished, the wound was quickly closed, and without drainage. The operation began at 11:20 a. m. and was completed at 12 noon.

The usual post-operative care for this type of case was given. Especially strict were the post-operative measures as to the diet when feeding was again commenced. Sutures were removed from a clean wound on the seventh post-operative day and the patient allowed to go home on January 27, 1934, 28 days after operation. About 1 month later than the date of discharge he resumed his ordinary work in the power-house at this station. When last seen after a lapse of 4 months subsequent to operation, he appeared well, reported about 25 pounds gain in body weight and no untoward gastric symptoms.

The second case of perforated stomach ulcer was that of H. C., age 19 years, male civilian, who was admitted in the afternoon of February 2, 1934, complaining of intense abdominal pain. States that it began about 3 hours ago while en route by rail from Washington to Quantico. This pain was terrible in its intensity and came on as suddenly as though he had been struck. It has continued unabated since onset. A history is given of gastric ulcer for the past 2 years which has necessitated his observing a bland, soft diet. However, at lunch today he ate a goodly amount of veal chow mein. Has not eaten since noon. Does not indulge in alcoholic liquors.

The patient is a well developed, but poorly nourished young adult male of 19 years. He was in a state of profound shock. The abdomen was scaphoid in shape, with marked and boardlike rigidity. There was extreme tenderness and soreness in midepigastrium. Prepared at once for emergency operation. Diagnosis of probable ruptured gastric ulcer made preoperatively.

Usual incision for exploratory upper abdomen; upon locating a large perforated ulcer, it was carefully closed with purse-string suture, and this closure was reinforced by two rows of Lembert sutures. A remarkably large perforated ulcer was found in the anterior wall of the stomach about 2½ inches from the pylorus and just approximately midway between the greater and lesser curvatures. The opening itself measured about 1¼ inches long and three-fourths inch in width. Gastric contents in an alarming amount previous to closure were noted leaking into the abdominal cavity. The ulcer was surrounded by the characteristic layer of fibrotic more or less friable tissue. The wound was closed without drainage.

The immediate post-operative condition was not good, the patient being still in a condition of considerable shock. In addition he took the anesthetic badly; the respirations being shallow but the pulse good, running from 80 to 118. He was cyanosed most of the time. The operation was started at 10:20 p. m., ended at 11:20 p. m., considerable difficulty being encountered in closing the peritoneum on account of the excursions of the intestines while this part of the surgical procedure was being done.

Whereas in the first ulcer case recovery was uneventful, the second proved to be very eventful indeed. Temperature and pulse returned to normal in about 7 days, when sutures were removed from a clean wound. Respirations remained practically normal throughout convalescence. However, temperature and pulse went up about the eighth post-operative day and for 2 weeks remained elevated before finally returning to normal. The patient developed muscle spasm in the lower half of the abdomen about the eighth post-operative day accompanied by distension and constipation. We were at a loss to account for these symptoms but ascribed them as due to absorption from a collection of some of the escaped gastric contents which had remained in the lower abdomen post-operatively and which stomach contents therefore had not been removed in their entirety at operation. Conservative treatment was carried out owing to the patient's weakened condition, no further surgical intervention was made, and all symptoms fortunately disappeared by the twenty-second post-operative day.

The patient was discharged to his home March 3, 1934, 30 days after admission. Since this time, about 3 months after operation, under strict dietary and general surveillance he has gained 27 pounds body weight, feels well, incidentally weighing more than during any period prior to admission here as a patient. He reports very occasional gastric disturbances accompanied by pain at rather rare intervals.

The third ulcer case, that of First Sergeant J. A. D., U. S. M. C., 42 years of age, was admitted April 3, 1934, with chief complaint

intense pain in the abdomen, which pain came on with characteristic and terribly sudden intensity just ½ hour prior to admission, while patient was standing leisurely in front of the post fire department building. It felt as though something had grabbed him in the epigastrium and then had begun to twist. The pain was excruciating and caused him to break out in a cold sweat.

He gives a history of first having had stomach trouble in Guam in 1928 when he had severe abdominal cramps followed by vomiting of a considerable amount of red blood. He was at the time on the sick list for 7 days and experienced no further trouble until 1932 when he underwent the torture of severe upper abdominal cramps for 3 days. He was on the sick list this time for 6 days. He has had no trouble since then until about 1 month ago he began having occasional cramping pain in his abdomen. He had taken no treatment and had not followed any particular diet. Uses alcohol moderately and has done so for many years.

Prepared for immediate operation with a preoperative diagnosis of perforated gastric ulcer. One hour from onset of attack the abdomen was opened with a high right rectus incision, and a perforated ulcer located near the pylorus close to the greater curvature with an opening about the size of a nickel (5-cent piece), with free gastric contents escaping through the aperture. It was closed with a purse-string suture, this closure being reinforced by a double layer of Lembert sutures. The wound was closed without drainage.

The patient stood the operation well. The pulse was of good quality throughout; his color was good. He was, however, nauseated at intervals while on the table. The operation was commenced at 10:45 a. m.; completed 11:35 a. m. Condition was good immediately following operation.

His convalescence proved to be uneventful and he has gained weight in the past 2 weeks. The patient has been in the hospital now for a little over 1 month and will return to duty at an early date.

GOITER [1]

By Ogden D. King, Commander, Medical Corps, United States Navy

The term toxic adenoma is misleading. The clinical picture which it implies is common, but histologically it does not exist. Toxicity is always associated with hypertrophy and hyperplasia and true adenoma is an outright nontoxic lesion. If the two conditions occur together, hypertrophy and hyperplasia are found in the parenchyma of the gland outside the adenoma, and are in no way connected with it.

[1] From the Surgical Service, United States Naval Hospital, Brooklyn, N. Y. Read before the Brooklyn Surgical Society, December 1, 1932.

Much of the confusion which existed in regard to this subject was due to the fact that toxic adenoma was wrongly considered to be a distinct entity, in which iodine therapy and type of operation differed from that indicated in exophthalmic goiter. The surgeon found so much disparity to exist between the clinical and histological findings of such a classification that he was often uncertain as to the type of case with which he had to deal. Cases clinically typical of exophthalmic goiter were found frequently to have a nodular type gland instead of the smooth, symmetrical gland of Graves' disease. Also, cases both clinically and histologically typical of exophthalmic goiter often had associated other areas entirely different in appearance from those of hypertrophy and hyperplasia, which areas resembled the so-called colloid adenoma, fetal adenoma, colloid cyst or areas of marked degenerative change. In other words, this varying histological picture is found so frequently that one is at a loss, considering the old classification, to determine from the clinical aspect of the case what the underlying pathology is. Pathologically the gland can be either diffuse or nodular, hard or soft, highly vascular or not, friable or tough, or a part of or all the gland may be involved and yet present a similar clinical picture.

There has been a decided change of opinion of many of the leading pathologists (1) in respect to the interpretation of the histological findings in goiter, and research has accomplished much for a better coordination of the clinical and histological findings in this disease. Clinically, the case has symptoms of hyperthyroidism, or it has not. Boyd makes the following pathological division; (*a*) diffuse goiter with hyperplasia, (*b*) diffuse goiter with involution, (*c*) nodular goiter, in which either hyperplasia or involution may be predominant.

Rienhoff (2) states that toxic adenoma is a misnomer and that 90 percent of the nodular type glands are not adenoma at all but are nodules which are the result of involutionary changes following previous hypertrophy and hyperplasia and are in no sense neoplasms. True adenoma does exist but is responsible for only 10 percent of nodular goiters. By the use of Lugol's solution, which is well known to produce a marked remission of symptoms in hyperthyroidism, he (3) was able to construct the picture of the change from hyperplasia to involution, something that had been possible before only by inference and indirect observation. He points out that the remission caused by iodine is identical histologically with spontaneous remission. Also, that all cases of thyro-toxicosis, except the most fulminating forms, undergo remissions and exacerbations. In seven cases which presented the usual diffuse enlargements characteristic of exophthalmic goiter, he removed a portion of the thyroid, administered Lugol's solution and 2 or 3 weeks later removed another section of the gland. In this way he was able to compare the change caused by involution

with those of hyperactivity in the same gland. In all seven cases after the use of Lugol's solution, the gland was no longer smooth but presented nodules varying from 0.5 to 3 centimeters in diameter. Cyst-like areas containing fluid and more opaque and granular areas could be recognized. Microscopically, the change in the picture was extreme; certain areas, however, showed greater changes than others.

According to him there are two opposing factors, one responsible for hypertrophy and hyperplasia and the other for involution. These account for the histological picture of every type of goiter, whether it be the simple colloid, simple physiological hypertrophy of puberty, or the most toxic type of Graves' disease. The only differences are of the degree of these changes. No qualitative difference has yet been demonstrated. The toxic and nontoxic or the diffuse or nodular enlargements and the other confusing divisions and variations merely represent different stages or degrees of change in this process. Just why the hypertrophy and hyperplasia and subsequent involution takes place and why the various degrees of each is not definitely known. This hypothesis, however, does seem to correlate the diseases clinically and histologically and affords a better understanding of the underlying pathology. Which all resolves itself into the fact that we are not, in the various types of goiter, dealing with a multiplicity of diseases but with different syndromes depending on the stage or degree of activity of hypertrophy and hyperplasia, or of involution, and upon the proportion of the gland affected.

There appears to be two types of hypertrophy and hyperplasia. In one the acini contain large papillomatous projections and a small lumen with colloid, while in the other type the acini are small, non-papillomatous with small or no lumen or colloid. If the proliferation in a small area is rapid, it will simulate the formation of benign tumors. Also, the larger type of acini if limited to confined areas will simulate tumors. These nodules are, however, the product of hypertrophy and hyperplasia and are in no sense neoplasms.

In involution the opposite kind of changes from those of hypertrophy and hyperplasia takes place. The acini become larger, more regular in size and shape, and are ballooned out with colloid approximating the appearance of the normal gland. The cells lining the acini lose their columnar shape and become more cuboidal, the colloid becomes less fluid and better staining. The tendency is toward complete involution, but always some cells are found to undergo greater involution than others and the histology of the normal gland is only approximated. Involution of the above degree, which approximates the normal, is called normal involution. Hyperinvolution is of a greater degree and hypoinvolution is of a lesser degree than normal. In the hyper-degree the acini become more distended with colloid than normal and the lining cells flatter and smaller, perhaps

forming only a slight framework about the enclosed colloid. This process may continue on to that of degenerative changes. Histologically, hyperinvolution is indentical with so-called "colloid adenoma", colloid cyst, and degenerative states depending upon the degree of involution beyond normal which has taken place. Hypoinvolution, which is involution of less than normal degree, is shown as small round acini without lumen or colloid, crowded together into nodules, really areas of persistent hypertrophy and hyperplasia, which have failed to undergo involution. These areas are identical with so-called "fetal adenomata." The picture may vary from this degree of involution up to the normal degree of involution. Thus nodular formations in the thyroid gland are caused by hypertrophy and hyperplasia and nodular formations are caused by hyper- and hypo-involution. These are not neoplasms but are the product of this process, the same process which is responsible for all thyroid enlargements, excepting thyroiditis and neoplasm, different only in degree in the diffuse or nodular and toxic or nontoxic types.

In hyperthyroidism, the histological picture of hypertrophy and hyperplasia is always present and the degree is proportional to these changes. In the normal cycle of the disease there are periods of exacerbation and periods of remission with a tendency toward greater exacerbation and less complete involution each time. In other words, it is a continuous process with changing phases, and areas representative of these phases are always present, perhaps may all be found in a single gland at the same time. The correct diagnosis is therefore to be made from the histological picture of the entire gland and not from a single area. Since these changes are truly representative of the clinical manifestations, one can, by careful history as to duration, number and degree of exacerbations and remissions, basal metabolic rate, and palpation of the thyroid, get a very accurate idea of what is to be dealt with pathologically.

Diffuse goiter is usually the more acute and more toxic. The nodular type means chronicity and is generally less toxic. It is, however, the form in which considerable damage has been done due to persistent low grade of hyperthyroidism which has existed a long time. It is the type in which auricular fibrillation most often occurs.

In the simple goiter there are also both diffuse and nodular glands. This condition is seen mostly in goiter districts. It is almost certainly due to iodine deficiency. That is, iodine deficiency causes some chronic enlargement of the gland, but the actual goiter is caused by this same process of hypertrophy and hyperplasia and involution which occurs here as a normal physiological process associated with changes of puberty, pregnancy, etc. These changes occur in goiter and nongoiter districts alike, but it is most often in the goiter districts where the thyroid is already enlarged that the

process results in enlargement sufficiently great to be called a goiter.

Benign adenoma, or true neoplasm, as stated above, accounts for about 10 percent of nodular goiters. It is an out-and-out nontoxic change and behaves like adenoma elsewhere in the body. It is encapsulated, may be large or small, firm or soft, cystic or not, depending on how it has developed and what has happened to it. If associated with toxicity, the adenoma is in no way connected with the surrounding hypertrophy and hyperplasia, which alone is responsible for this toxicity. Adenoma does not occur in goiter districts any oftener than elsewhere. It is surprising to note also that hyperthyroidism does not seem to occur in goiter districts oftener than elsewhere.

From the standpoint of diagnosis, the typical case of hyperthyroidism with the cardinal signs and symptoms of tachycardia, tremor, enlargement of the gland, exophthalmos, and increased basal metabolic rate presents little difficulty and is easily recognized. In the atypical case, however, any or all of the cardinal signs and symptoms, excepting increased basal metabolic rate may be absent. Difficulty comes in diagnosing the atypical, incipient, low-grade case and ruling out the functional disorders of psychoneurosis, neurasthenia, and neurocirculatory asthenia which often simulate hyperthyroidism but in which the thyroid gland plays no part. It is important to remember that hyperthyroidism may be superimposed upon any and all of these functional disorders. The basal metabolic estimation on which one relies for diagnosis may be upset in both types of cases and be misleading by giving an increase in the neurotic type, caused by irregular breathing, and a decrease in the low-grade type of hyperthyroidism, due to lack of cooperation. In these cases, the Goetsch adrenalin test is most valuable. This test is always positive in hyperthyroidism and, if carried out carefully, is very reliable. Occasionally, cases are seen presenting marked symptoms of hyperthyroidism without any apparent change in the thyroid gland. Most often, however, careful palpation will reveal some slight change in the gland.

Iodine in Goiter

Iodine has been used in the treatment of goiter for a long time, about as far back as medical history goes and long before the benefit obtained by chewing seaweed was known to be due to its iodine content. Kocher, however, discovered ill effects from the use of iodine. Due to his writings, and later to the writings of others who condemned the use of iodine, claiming that it not only increased hyperthyroidism but actually caused it to develop in simple goiter, the use of iodine was all but discontinued. About 10 years ago Plummer of the Mayo Clinic reported marked temporary beneficial effects from the use of iodine, in the form of Lugols' solution, in practically all of 600 cases

of hyperthyroidism and in none of these did he observe any bad effects. He, however, warned against the prolonged use of iodine in exophthalmic goiter, stating that its beneficial effects were temporary and that actual harm was done if too prolonged. He also rather emphasized the difference between the exophthalmic and the toxic adenomatous types of cases and stated that iodine did no good but actual harm in cases of the latter type. Following this report, iodine was reinstated in the form of Lugols' solution and it became generally used as a preoperative measure in exophthalmic goiter with excellent results. Soon, however, many surgeons began using iodine in all toxic cases, in both diffuse and adenomatous type glands, and good results were reported alike in both types.

Iodine serves goiter in two ways: First, as a prophylactic in simple goiter, in which, due to an iodine deficiency, iodine in 10-milligram doses once a week is an effective prophylactic. Second, its use in hyperthyroidism causes an artificial involution. This involution is identical histologically with spontaneous involution. Areas of persistent hypertrophy and hyperplasia (hypoinvolution) remain in both, giving rise to a low degree of persisting hyperthyroidism which later, in the normal cycle of the disease, gives rise to exacerbations. This is why iodine brings about remission only to a certain point and also why it does not prevent exacerbations. Iodine will cause an involution, that is in all except a small percentage of cases, about 5 percent, in which for some unknown reason iodine does not cause a remission of symptoms nor are involutionary bodies found in the gland. If the administration of iodine is stopped during an artificial involution, an exacerbation will occur immediately. If its use is prolonged, a gradual tolerance to iodine is acquired after which an exacerbation will take place whether iodine is continued or not.

So long as areas of hypoinvolution persist, there will be a persisting low grade hyperthyroidism and recurrent cycles of exacerbation. This is why it was formerly thought that iodine actually caused an increase in hyperthyroidism. Each subsequent involution is brought about with more difficulty and to a lesser degree with iodine. So it is due to this tolerance, the difficulty in producing subsequent involutions and the fact that the persistent low grade hyperthyroidism remains, that iodine should not be prolonged unnecessarily and should not be given in the first place to a fresh case of hyperthyroidism until definite arrangements have been made for operation. Iodine in therapeutic doses does not harm goiter cases, except as stated above. All the old claims of its ill effects have been largely exploded and today it is pretty generally used in the above capacity. Its use has cut down the mortality of operations tremendously and has mostly done away with the dreaded acute reactions of thyrotoxicosis formerly so frequent after operation. The average dose, in the form of Lugols'

solution, to prepare for operation, is 10 minims 3 times a day. The point of maximum remission is reached in 10 days to 3 weeks. Larger doses of about 30 minims are given immediately before and immediately after operation and have proven of further benefit in preventing thyrotoxicosis following operation. It is also used post-operatively in smaller amounts for several months in hyperthyroid cases. Certainly the reinstatement of iodine has done more than has any recent improvement in technique of operation to benefit this field of surgery.

Treatment

As for treatment of hyperthyroidism, the consensus of opinion is that a combination of medical and surgical treatment, iodine to bring about an artificial remission followed by surgery to remove the excess amount of gland, is the most satisfactory procedure. Iodine alone, X-ray, thyroid extract, hot-water injection, and other measures which have been used, have proven unsatisfactory. It is necessary, in the light of our present knowledge, that sufficient gland be excised to correct its increased activity in order to permanently cure the condition.

Each thyroid case requires considerable individual study both by the internist and the surgeon. These cases cannot be treated successfully as a group in a routine way. There should be the closest cooperation between the internist and the surgeon at all times in order that both may follow the case, the internist chiefly to determine the treatment necessary to obtain maximum benefit in preparation for operation, the surgeon to get an accurate idea of the clinical aspects of the case and its progress that he may know the underlying pathology which must be dealt with at operation. In addition to a careful general working-up, each case should have routinely an electrocardiogram, basal metabolic estimations, examination of teeth, tonsils, sinuses, vocal cords, and an X-ray of the chest, for record. The most favorable time to operate is during the first artificial involution. Once this opportunity passes, the conditions will never again be quite so favorable.

As stated above, it is necessary to remove surgically a part of the gland in order to permanently relieve the hyperthyroid state. Failure of operation to accomplish this result is most often due to the fact that too much gland was left behind. Excepting in that class of cases in which the disease has existed so long and to such a degree of intoxication that resulting organic changes and the residuals of the disease are too great, we cannot hope for a satisfactory cure. However, it happens entirely too often that the operation was not as complete as was indicated. The wedge-shape resection which did so much to simplify and popularize subtotal thyroidectomy is the one factor more than any other that is responsible for too often leaving too much gland behind. It is the simplest technic, looks well when

completed, but unfortunately is not adaptable to radical resection. All the gland necessary to leave in the usual case of hyperthyroidism is the posterior capsule which preserves the recurrent laryngeal and parathyroids, and a small amount of adjacent gland tissue which lies in the tracheo-esophageal angle.

A technic well adapted to this type of case begins, after the usual collar incision and retraction of the ribbon muscles and thyroid capsule to expose the gland, by a division of the superior thyroid vessels on the right side and a complete enucleation of that upper pole. After this the inferior thyroid artery is divided and the lower pole is delivered into the operative field. At this point it is sometimes advisable to divide the isthmus of the thyroid and free it from the trachea in order that there may be a satisfactory exposure. Good exposure, so that an accurate estimate of the exact amount of gland to leave behind can be made, is the most important part of the operation. The right lobe is delivered into view in this manner and, after an estimate is made of the pathology of the gland and a determination of the amount of gland to remove is decided upon, the lobe is resected. The isthmus and the left lobe are then dealt with in a similar manner. Catgut is used throughout and all cases are closed with rubber tissue drainage.

The pathology of the gland determines the amount of gland tissue necessary to remove at operation (4). Naturally, every case presents certain variations and no one prescribed technique is applicable to all cases. Suffice it to say that any clean dissecting type of technique which keeps the situation well in hand at all times and insures a satisfactory exposure and which avoids danger of injuring other structures, is satisfactory. The type of operation varies from a simple enucleation of an encapsulated neoplasm to subtotal resection of both lobes and isthmus for hyperthyroidism or a lobectomy for malignancy.

It is important to clean up foci of infection since it has been shown that bad teeth, tonsils, sinuses, and certain general infections have been too frequently associated with attacks of hyperthyroidism to be merely incidental but must be considered as predisposing factors. These should be cleaned up after operation, not before.

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FRACTURES OF THE MAXILLAE

By E. B. Howell, Lieutenant Commander, Dental Corps, United States Navy

The following conclusions drawn from the observation of 131 cases of fractures of the maxillae treated at the United States Naval Hospital, San Diego, Calif., during 1932 and 1933, are not to be considered as proven statistical data. Obviously, the number observed is too small to serve such a purpose. It seems pertinent, however, to present some of the most salient features encountered during the treatment of these cases.

Of the above number of cases treated, 60 percent of them were the direct result of traffic accidents. This fact is of no particular importance except, that from a standpoint of clean and dirty surgery, it is significant to note that this 60 percent for the most part, came under the former classification. Although fractures of this type are usually accompanied by lacerations and other body injuries, infection was less prevalent than in those which were the result of drunken brawls.

In spite of popular theories relative to the reaction of fractures in the presence of systemic disease, I believe that the effect of such diseases has very little to do with bony union; syphilis and tuberculosis to the contrary notwithstanding. Many cases of syphilis were encountered but in no instance was there any evidence of malunion, nonunion, or other complication. As a matter of fact, luetic treatment was always postponed in these cases because of the possibility of nausea which sometimes results from intravenous injections, and subsequent danger to patient with immobilized jaws.

Rather, I believe, that the patient's general physical condition at the time of injury has a more direct bearing on the ultimate outcome. If his condition is good and attention to diet is given proper consideration, the outcome is invariably good. But if the injury is the result of drunkenness and his resistence is at a low ebb, the case is usually fraught with complications.

In this connection I believe that diet plays an important role. Ivy's balanced diet was used at this hospital and adhered to in every detail. Because a patient is ambulatory it is not enough simply to inform him that fruit juices and other liquids of high vitamin and caloric content are available for him in the diet kitchen at all times. A separate mess should be maintained where food is accurately measured and the amount consumed recorded. Nourishment should be administered by direction, not by option.

Nothing new or original was undertaken in the treatment of these cases. Fracture appliances and splints of popular design, together with various forms of intermaxillary wiring were used as the individual needs of each case arose. Special attention was given to diet and to several other phases of this work which seem worthy of mention.

From the amount of pain and displacement suffered by patients whom I observed where the Barton bandage had been used as first-aid treatment, I heartily condemn the use of this bandage in fractured mandibles. The object of a bandage is to temporarily immobilize the part and to give some degree of comfort to the patient. A plain vertical bandage secured with adhesive tape will keep the jaw from sagging and accomplish this purpose with no danger of comminution or backward and downward displacement.

Early application of ice rather than heat gave more comfort in these cases, and appeared to do more toward warding off infection than any other thermal expedient.

The Jelenko fracture appliance proved to be much more adaptable than the Baker anchorage. The smooth rounded lugs of the Jelenko were more easily engaged with wire, less irritating to the patient, and its eyelets facilitated the placing of traction in many different directions. Where short sections were used, the flat inner surface worked perfectly because of its resistance to rotation.

I believe that much harm can be done by the injudicious removal of teeth in the line of fracture. Every tooth so located, should not be considered a foreign body and arbitrarily extracted. Such procedure sometimes changes a simple into a compound fracture and infection results. Where the tooth involved is fractured, dislodged, or so placed as to prevent the fragments from coming into apposition, naturally extraction is indicated. In numerous cases of mandibular fractures where the line of fracture extended from the angle to a point on the alveolar border between the last two molars, invariably the last molar was retained. Particularly valuable was this molar when an apposing tooth was present in the maxilla. Because of the tremendous traction placed on these posterior fragments by the upward pull of the masseters, only the presence of this important tooth prevented marked displacement.

An appliance for the exercitation of the mandible as described by Ivy, in his book on Fractures of the Maxillae, was found to be invaluable in overcoming trismus and partial ankylosis. It acts as an elastic articulator and is easily constructed. In two cases of bilateral fracture of the necks of the condyles, partial ankylosis resulted from prolonged immobilization. One case was operated and the other was freed by the use of this appliance. Unlimited motion was obtained in 3 months.

Normal occlusion and restored function is of course the objective in all these cases. However, only as an extreme measure should we resort to open reduction as a means to this end. Variations in relationship of fractured fragments to each other mean nothing so long as normal occlusion has been achieved. Surgical interference for the purpose of obtaining true bony alignment is a dangerous expedient, and the benefit derived questionable.

APPENDICITIS

BY RUDOLPH D. JOLDERSMA, Lieutenant Commander, Medical Corps, United States Navy

The fact that the annual loss of life in the United States from appendicitis is 20,000 is appalling. Then, we study statistics and find that the death rate has increased from 11.4 per 100,000 in 1910 to 13.4 in 1920 and to 18.1 in 1930 and also find that we have the highest mortality rate per population of any of the civilized nations, it is high time that a careful study of cause of this mortality increase should be made and an alarm sounded.

We have, in this time, had a marked increase in hospital facilities, also a tremendous increase in transportation facilities; and we know a marked step forward in surgical technic. So these excuses are ruled out.

Kalodny, in the Journal of the Iowa State Medical Society, states:

> The factors influencing the mortality from appendicitis may be reduced to two, the delay of operation and the incompetency of the operating physician. If one were to consider delay as the only cause of the increasing mortality from appendicitis, it would be impossible to explain the contrast between the mortality in this country and in Europe. Human nature is about the same everywhere, and the average person is nowhere enthusiastic over undergoing a surgical operation. One must admit that there is another factor in the mortality. This factor is the incompetency of the casual operator in America in dealing with the difficult problems that arise in advanced appendicitis. This is a factor of foremost importance in this country, but not in Europe, where most of the surgical work is concentrated in the hands of master surgeons. He also states when patient is not at fault, delay by the attending physician is almost always due to inability to make a diagnosis.

Acute appendicitis is the most common acute abdominal condition that the general practitioner either in a large or small town has to deal with. It is the most insidious and at times baffling of acute conditions and like a kaleidoscope in the twinkling of an eye changes from an apparently subsiding condition to a case of general peritonitis. In 1 hour it can change from an immediate operative case to a case which would better be operated at a later time. Yet the teaching and training of internes as regards accurate diagnosis and stressing of when to and when not to operate is minimized.

We seem to have two wide-spread schools. One, if the patient has typical symptoms, operate at once and this is taken to mean by many surgeons, no matter in what stage. The other school, of whom I hope there are few, if given expectant treatment, most of them will get well. The radicals of these two schools, I think are the cause of our high mortality.

I desire in this paper to stress two points. After the diagnosis of the case, that involvement of the appendix exists, we must determine accurately, first, what pathology of the appendix is present, then, secondly, with the pathology present and condition of patient con-

sidered, is immediate operation imperative or will the patient's chance of recovery be improved by delay of operation?

If we will take the history, the symptoms, the findings by examination and the laboratory findings and then try and correlate them all and decide what pathological condition would give the symptoms, the disturbed physiological reactions and the changed blood picture findings, we could then better decide what the exact pathology is and what is the best immediate treatment for the pathological condition present, considering the patient's condition, not just what treatment should be given for the symptoms. Symptomatic treatment is not in the present scientific modern age considered up-to-date medicine or surgery.

Let us consider the pathological changes that can take place in appendicitis. In all cases, to start with, we have acute inflammation, this terminates in one of three ways:

(A) Subsiding or return to normal.

(B) Tissue destruction with (1) local ulceration and perforation (2) gangrene or suppuration. These both may terminate with perforation or rupture and result in (*a*) general peritonitis (*b*) walled off abscess.

(C) Healing with fibrosis.

In all cases at the start the appendix becomes swollen, tender, the vessels dilated and the mucosa swollen and congested. This gives rise to the typical syndrome colic nausea or vomiting and final localization of pain on right lower quadrant.

On examination we find:

1. Pain on pressure over region of the appendix is a never failing sign.

2. Muscle spasm may or may not be present. Muscle spasm is due not to the inflamed appendix but to the extension of the inflammation to the parietal peritoneum. An appendix hanging free over brim of pelvis or one bound down retrocaecally may give very little if any muscle spasm.

3. The W. B. C. will be 15,000 to 18,000 and most important early in attack the poly percent will be slightly below normal, about 65 percent. Pulse will be accelerated.

(A) If this inflammation is going to subside and the appendix return to normal, in 4 to 6 hours the pain will subside, the muscle spasm, if present, will lessen, pain on pressure will markedly decrease, the pulse rate will be very little above normal and the total W. B. C. will be less, and of most vital diagnostic significance there will be no increase in poly percent—it will remain below 70 percent. True, if a case of this type, even with a low W. B. C., count and below 70 poly count, is operated early, the surgeon can always demonstrate a red, inflamed appendix and claim he is correct, but we all know in in-

flammation the tissue involved does not return to normal in 1 or 2 hours nor does it always terminate in tissue destruction and formation of pus. These cases can very safely be given medical treatment and will subside.

(B) (1) Under this pathology if after the typical onset syndrome the inflammation progresses and involves the muscularis and subserous coats, we may get thrombosis of some small vessel and local death and ulceration which rapidly extends through the muscularis and serous coats and perforates. What is our warning signal here? After 6 to 8 hours the pain will have become duller and subsided somewhat, muscle spasm if present persists, finger-point tenderness on pressure becomes markedly more and more acute, "exquisitely tender on pressure", and as there is tissue destruction going on the pulse will be more rapid. There will be slight temperature rise and the one unfailing sign of tissue destruction is a marked increase of poly percent though the total W. B. C. has fallen. Falling W. B. C. with polys rising over 72 percent should be viewed with apprehension and should be operated on at once.

(B) (2) If the inflammation progresses, and instead of as above, thrombosis of small vessel taking place, the appendix becomes generally involved and gets larger and more swollen. Stricture of the vessels due to marked swelling and oedema of the appendix may take place and due to lack of blood supply the appendix becomes hard and white, the lumen is dilated and becomes filled with mucopurulent material, (1) gangrene or sloughing may occur or (2) liquefaction necrosis may take place, and we finally have a peritoneal sac filled with pus which may rupture at any time. In these cases, due to oedema and shutting off of blood supply, the pain subsides and the patient feels much better, but the pulse and temperature is up, tenderness on pressure is still present and is exquisite on deep pressure. The most important diagnostic point is an increase of over 80 percent in polymorphonuclears with falling white blood count, as there is tissue destruction. Many times a case about 6 to 10 hours old with these symptoms is operated and a hard white appendix is removed and the operator remarks this is not so bad. To the naked eye no inflammation is apparent but pathologically it is a bad appendix, as its blood supply is shut off and we have white oedema and death tissue with necrosis due to take place. If these cases are 4 to 6 hours older and more progressed and tissue destruction is taking place, the patient complains of very little pain and says he feels better but the most diagnostic point is finger-point exquisite tenderness over locale of appendix with increase of pulse and temperature. Muscle spasm may not be increased. But at this stage the polymorphonuclear percent will be well over 80 and this is the red flag danger signal even if the white blood count has fallen to 12,000. I

remember one case which had had a typical attack, 14 hours previous, and when brought in, stated he felt fine and wanted food. He had no muscle spasm at all but finger-point exquisite tenderness on very deep pressure below the McBurneys point. Pulse was 80 and temperature 99. His white blood count was 10,400 but his polymorphonuclear lencocytes were 91 percent. I operated at once and found an appendix the size of a frankfurter, which was nothing but a peritoneal sac full of pus, and it ruptured just as it presented right through the incision.

Now, we will consider a case in a later stage, where rupture or perforation has taken place before we see the patient and we have (*a*) general peritonitis or (*b*) a walled-off condition. When the patient is seen the general appearance is of extreme toxicity. The history is of a typical acute attack 18 to 24 hours or longer in duration and the patient states that suddenly his localized pain disappeared.

In the first of these conditions, perforation or rupture with general diffuse peritonitis, the abdomen will be acutely distended and tender, there will be a general hard boardlike rigidity of the whole abdomen, high white blood count, and polymorphonuclear increase. The condition is caused by a ruptured appendix, there is no walling off and immediate operation with just eliminating the source of infection, ligating the ruptured appendix should be done. Here and here only does Murphy's dictum hold "open the abdomen as quickly as possible and close it still quicker." Drainage does not drain out the pus, this is physiologically impossible. Drainage only inhibits the defense reaction of an already severely overtaxed peritoneum, and postoperative obstruction is much more frequent in the drained cases than the undrained ones. Reference (*b*) authors state:

> In analyzing 2,126 cases of acute appendicitis, 231 cases admitted with diffuse peritonitis. In cases with diffuse peritonitis operated on in first 4 days, death rate was 20 percent and complication 20.6 percent. In cases of appendicitis with diffuse peritonitis immediate operation would appear to be the only rational form of treatment to adopt. If appendectomy is done, drainage of the peritoneum seems of little importance.

In cases of perforation or rupture with walled-off mass, the abdomen will be acutely tender, most marked on the right side, there will be a hard boardlike rigidity on the right side only, left side fairly soft. Patient will be very toxic. Temperature will be high, pulse very high, the white blood count high.

The sloughed or perforated appendix is walled off in these cases. The patient is extremely toxic, as the extremely high pulse and temperature and white blood count show. The system is carrying an extreme peak load and to add the shock of an operation may be the fatal turning point. Another point against immediate operation is that the peritoneum is carrying its maximum load and has laid down numer-

ous fresh and fine adhesions, and to operate and to certainly spread the infection to the rest of the peritoneum will certainly increase our mortality rate. These cases should be treated expectantly until the body resistance is built up as shown by pulse nearly normal and morning normal temperature. Then, if mass is palpable small incision and drainage of abscess formed is almost without danger. It should be 5 or 6 days before the abscess should be drained. These are the cases where Oschners treatment is of so great a value.

Sworn and Fitzgibbon, in the British Journal of Surgery, state that of 487 cases admitted with palpable mass:

	Died	Percent
65 appendectomy and drainage	10	15.4
124 drainage only	5	4.03
298 no operation or drained 5 to 7 days later	2	.65
Mortality in subsequent appendectomy	0	

Their conclusion.—It has been the practice in this hospital to follow a conservative course where possible; if urgency of symptoms make operation essential, drainage alone is the method. In some instances of spread of infection of the peritoneal cavity appendectomy and drainage is done. Although this conservative method of treatment necessitates readmission for appendectomy after a suitable time, of such cases 299 were operated on with no deaths.

Personally, in the past 2 years I have had 11 cases of palpable mass; 1 was opened and drained on admission and died. Ten were given conservative treatment and walled off abscess opened and drained 4 to 7 days later with no deaths.

Kolodny (reference a), states:

> There are cases of appendicitis in which immediate intervention is meddlesome. A widely spread erroneous idea is that the appendix must be removed in whatever stage appendicitis is tackled by the surgeon. The casual operator does not realize it is poor and reckless surgery to go hunting after an appendix that is buried between inflamed, sticking-together, intestinal loops. He is not familiar with the fact that breaking up protective adhesions in presence of a palpable lump is ill-advised surgery.

In a discussion at the Royal Society of Medicine in London, the recommendations of Dr. J. A. Ryle, of Guys hospital were: (1) Inflammatory cases, interval operation, or expectant treatment. (2) Abscess, drainage at most favorable moment. (3) Obstructive or gangrenous type, immediate operation. (4) Late case with distention and signs of obstruction, consultation.

Mr. H. H. Rayner, of the Royal Infirmary, Manchester, advised early operation on diagnosis and said in patients seen late second or third day he recommended expectant treatment for (1) cases of localized muscle rigidity. (2) Those presenting a palpable mass. (3) In general peritonitis operate at once.

Mr. Herbert Brown stressed the risk of operating on an apparently simple abscess in the middle of the first week. Until the end of the week there was no general immunity, and swabbing a cavity might disturb the layer of protecting lymph and cause septicaemia.

Mr. Zachary Cope was certain that there were cases in which it was better to wait. If the appendix had perforated there was a lump and the resistance of patient was rising after few days expectant treatment.

A typical acute case admitted 6 to 8 hours after onset with original white blood count of 17,000 who on admission showed 12,000 white blood count and polymorphonuclear leucocytes below 70 percent, I have always treated expectantly. On the other hand if the white blood count decreased, had but the polymorphonuclear count increased, I considered this suspicious, and a check 4 hours later nearly always showed above 80 percent polymorphonuclears, and immediate operation was performed.

All cases with 75 percent polymorphonuclears or over were operated on at once. Did you ever consider that with 17,000 white blood count and 65 percent polymorphonuclears there are actually 11,050 of these cells present? With the count 14,000 and the polymorphonuclear percent 80 there are now 11,200 polymorphonuclears actually present. The riot squad is being called out even though the general police force is reduced in numbers; 3,000 less white blood cells, but 150 more polymorphonuclears actually present.

DANGER SIGNALS

(1) Increased exquisite tenderness on finger point pressure.

(2) Increased pulse and temperature.

(3) Decrease of muscle spasm or absence not significant.

(4) Falling white blood count and rising polymorphonuclear count a warning signal.

(5) The red flag of danger polymorphonuclear percent over 80 percent.

CONCLUSION

(1) If subsiding wait and advise interval operation.

(2) If evidence of tissue destruction operate at once.

(3) Ruptured appendix and general spreading peritonitis, appendectomy if patient's condition warrants.

(4) Ruptured or gangrenous appendix and palpable mass, wait till patient's general condition warrants drainage at least from 5 to 7 days later.

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INDUSTRIAL MEDICINE

By H. L. SHINN, Lieutenant Commander, Medical Corps, United States Navy

Little has been written in recent years for the NAVAL BULLETIN on the subject of Industrial Medicine, with reference to its application in the Navy. This topic having come into such prominence in recent years, especially with reference to accident prevention, it is felt that it would be well, at this time, to briefly review the work being done along this line.

It is believed that the average naval medical officer has given little thought to the subject. The NAVAL MEDICAL BULLETIN, in each edition, under the Division of Preventive Medicine, indirectly refers to the subject by listing accidents which have occurred in the Navy. Accidents are classified under the headings of those connected with work or those within command, and those occurring while on liberty or authorized leave of absence, in each case denoting whether or not any apparent negligence or misconduct was a contributing factor leading to the accident or accidents. The Surgeon General's report for the calendar year 1932, page 124, lists a total of 6,647 original admissions to the sick list; 110 A. C. D. and 226 R. A. for injuries to personnel during the calendar year of 1932. Of this number, 6,280 were returned to duty, 178 died, and 106 were invalided from the service. The total number of sick days for the year for accidents was 154,408, or approximately 22 sick days for each injury. These figures of course do not include the number of minor accidents which occurred and which did not require admission to the sick list.

Now, these figures and accounts of injuries make interesting reading. An injury of any nature and at any time is given far more publicity than an illness. Medical officers, as a rule, read these figures and accounts of injuries and promptly forget them, considering them as every-day occurrences, something to be expected, and that nothing can be or is to be done about them. Right here it seems appropriate to quote a paragraph set forth in a book on Industrial Medicine, to emphasize the chief purpose of this paper. "A surgeon in a small town once said to a friend, 'That factory over there is a gold mine to to me. I get at least 2 fractures and 6 hand injuries each week from among the employees there.' Now, this same surgeon had been very active in cleaning up his home town; providing proper sewerage, etc., in an effort to prevent the spread of typhoid fever. He was not mercenary. He further had a high standing in the community, and in his profession." The lesson to be learned from this, and the point that it is desired to emphasize, is that this surgeon's views on prevention had not broadened beyond that of disease prevention. He had not given a thought to the fact that the prevention of injuries is in the same category with disease prevention and had not kept abreast of the times. And so it is in the case of many of our naval medical

officers. We preach and practice preventive medicine but we fail to do the same to prevent accidents. We read that there were nearly 7,000 accidents in the Navy during a certain period, causing hundreds of thousands of days of sickness and hundreds of deaths, but we have not grasped the fact that it is as much our duty to prevent these accidents as it is to vaccinate to prevent smallpox and inoculate to prevent typhoid fever, diphtheria, scarlet fever, and the like. It has just never seemed so important to us and we have never seen it in this light before. I feel that today the practice of preventive medicine, hygiene, and sanitation in the Navy is on a very high plane. The practice of prevention of accidents is not equal to that of preventive medicine, and another purpose of this paper is to stimulate interest in this subject and to put preventive accidents on a par with preventive illnesses.

It is well for every doctor in the service to practice industrial medicine with special interest being given to accident prevention. It is a wide field of medicine and one which needs development. It would be interesting to note just what the attitude of the average naval surgeon is, regarding accident prevention. To be frank, the writer, before having been assigned to this duty, was about in the same class with the small-town surgeon herein referred to. Little thought was given to accidents, other than that they did occur and that they necessitated treatment. Whether the accidents could have been prevented or not, never was given much thought, unless a board of investigation or inquest brought out the fact, and then it was too late. Since the writer had been doing this work many medical officers have jokingly asked, when the subject of industrial medicine was brought up, "Just what is industrial medicine, and what are your duties?" These are honest questions for the reasons previously set forth, foremost of which is the fact that accident prevention has not been given serious consideration in the Navy. Now, many are familiar with the term "industrial medicine" but I venture to say that few are cognizant of the vast amount of territory covered by this subject and its importance to mankind today. In a general way it is the purpose to discuss in this paper, industrial medicine as a whole, but more especially the application of its principles in the Navy and its importance to naval medical officers.

Industrial medicine covers such an extensive field that it is difficult to propound a definition that is suitable. However, the following definition is fairly brief and comprehensive:

Industrial medicine is that branch of medicine which deals with the preservation of the general health of the people as a whole but more especially those of the working classes, against disease or injury. It embraces the principles of preventive medicine, hygiene and sanitation, and accident prevention. Its purpose is to insure good health and prevent injuries, thereby promoting contentment, alleviating

suffering, and increasing the life span of man. It further deals with the rehabilitation of diseased and injured persons in an effort to insure them a livelihood. The efforts of those who are interested in this work are directed not so much to the cure of diseases and injuries but to the prevention of them by placing working conditions of employees on a higher plane with regard to hygiene and sanitation and the prevention of avoidable accidents by means of education, safety devices, periodic physical examination, and the like.

Industrial medicine has been practiced for many centuries and was made necessary by the advent of the factories as early as the fifteenth century. With the opening of the factories the people migrated to a large extent into the towns and manufacturing districts. Working conditions were of the poorest type and the employees were driven like slaves. Death took a heavy toll. The factories were referred to as slaughter houses. In the sixteenth century, it is said that 50 percent of the working population of England died before the age of 20. The average age of the working classes was 22 years as compared to 44 years for the upper classes. Right here then began the study and practice of preventive medicine and surgery in the interest of humanity and as a matter of social and political economy. An interesting study of the progress of industrial medicine is outlined in the book on industrial health by Kober and Hayburst. In passing however, it is interesting to note that one of the first laws enacted for the protection of the working man, limited the working hours to 15 hours a day and the legal day's work was to be 12 hours. How different it is today! Eight hours work a day is the legal day's work in most places. The week has been cut to 5½ days with a possible 30-hour week in view. Now some of this has been occasioned by economic conditions but a great deal of it has been the result of the campaign which has been fought through the years for betterment of working conditions, and the desire to improve the health.

As stated before, aside from increasing the life span of man, industrial medicine deals with the prevention of disease and injury in an effort to alleviate suffering and further endeavors to reduce the cost of disabilities to both employee and employer. A man who is sick or injured cannot work. His pay stops in many instances. His family suffers from lack of money. The employer loses the services of a trained man. His work must be carried on by another at added expense. If there is indication that the sick or injured man was incapacitated in the line of his duty, then the employer must pay the employee compensation. You can readily understand then the value of the practice of industrial medicine from an economic standpoint. It is understood that in the year of 1933 that the cost to the United States Government for compensation due to hernias alone was over $100,000. This was among the civilian employees of the Government, and not in the service.

To my mind the economic consideration is of secondary importance to the prevention of unnecessary suffering and to the betterment of the general health of the people. To maintain good health then, is the primary object of industrial medicine. This is, or should be, the object of all naval medical officers.

Industrial medicine today covers practically the entire field of medicine and surgery. A doctor who is thoroughly conversant with the subject of industrial medicine is indeed well informed in his profession. The Department of Labor issued a pamphlet relative to occupational hazards and the diagnosis of industrial diseases. The number of this bulletin is 306, of April 1922. If space permited I should like to incorporate this entire bulletin in this article, for it is one of the most complete and comprehensive works of its kind. This bulletin may be obtained by letter to the Department of Labor and is well worth having for your use either in or out of the service. It is desired to quote from the introduction to this bulletin to emphasize the importance of industrial medicine today, and to bear out the statement made regarding the usefulness of this subject to the physician of today. "Many occupations have injurious effects on the physical condition of those engaged in them. The health of those who work with the poisons, such as lead, arsenic, mercury, picric acid, etc., or those who are exposed for long periods to dust, heat, humidity, or to the infectious materials, etc., may be impaired seriously as the result of their work. The occupation is now recognized as of the very first importance as a factor in the causation of disability and even of death. Dr. Edsall has shown that in his clinic at the Massachusetts General Hospital many of the conditions for which treatment is sought by men of working ages are the effects of occupation. Other industrial clinics are reporting similar results. With their attention directed to occupation as a possible factor, industrial physicians are able to diagnose a great many obscure cases which previously had puzzled even the most competent clinicians. In this way they discover a great many more cases of disease of occupational origin than had before been thought possible. Thus, in 1917 about 150 cases of lead poisoning were recorded by this clinic during the 5-year period prior to the adoption of the more intensive methods of study. It is generally recognized that patients come to physicians with pains and complaints of an indefinite character, and it is only when consideration is given to the occupation and its possible effects that many of these cases are cleared up.

The medical examiner should, therefore, be very careful to see if any of the usual diagnostic signs of poisoning, dust, heat, or other hazards which are known to be inherent in occupations are in evidence among their patients where no other explanation of the case is readily available. In the case of those exposed to lead, such as employees of

storage-battery plants, white-lead workers, paint mixers, painters, etc., the blue line on the gum, the pale, sallow appearance, and the trembling fingers are significant as indications of chronic lead poisoning, and the physician should look for these signs. Physical symptoms and conditions which ordinarily might be passed by, in this way become very important if they point to the possible effect of the occupation.

This handbook has been prepared to aid physicians in general practice, industrial hygienists, safety engineers, and others who come into close professional contact with those who are engaged in industrial processes. Nine major hazards of employment are listed; namely, abnormalities of temperature; compressed air; dampness; dust; extreme light; infections; poor illumination; repeated motion, pressure, or shock; and the poisons. A separate section of the bulletin is devoted to a discussion or skin irritants.

Medical examiners should remember that it is often necessary to keep in mind not only the present occupation, but the former one as well. Persons suffering from certain ailments may no longer be engaged in the industry which was originally responsible for their condition. But careful inquiry into their occupational history will sometimes result in the recording of an occupation the effects of which are clearly those from which the patient is suffering. The medical profession must give occupational findings greater weight in forming their judgments regarding physical conditions and in diagnosing and treating disease.

It is hoped in this way that the medical profession will become more and more acquainted with occupational diseases and help in the movement to discover and eliminate cases thereof. In our country it is still true that very large numbers of working people are constantly exposed to serious occupational hazards and suffer, often unnecessarily, very seriously from the effects of such exposure. The greater interest of medical practitioners will help materially in bringing about this result by giving due weight to the subject in their courses of study. Already the form and content of the pamphlet have recommended it to several schools, which report its value. In the same way plant executives and safety engineers must look carefully into their own establishments to see to what degree the processes in their shops are devoid of the dangers which are usually associated with industrial operations. Factory inspectors, labor officials, and workman's compensation boards will find it helpful in inspection and rating numerous industries. Many hazards should be revealed which they have not known were associated with the processes of manufacture and of which the employers themselves have been ignorant. The comparatively new but rapidly expanding field of industrial rehabilitation should find this bulletin an aid in selecting occupations

for those with arrested cases of tuberculosis and for others weakened by disease.

The introduction to this pamphlet states clearly and truly the importance of industrial medicine. One of the first things taught to students of medicine and internes is the importance of getting a thorough history of the case before trying to arrive at a diagnosis. Stress is laid on the family history, the past personal history, and the history of the present illness. Little attention was paid to the occupation of the patient. Now, with our advance in the knowledge that many diseases are caused directly or indirectly by industry, it is essential that we should get a careful history regarding the past and present occupations of the patients. This history will in many instances lead to the establishment of a diagnosis.

Now, you probably wonder just what all of this has to do with the Navy. The Navy, whether you realize it or not, is a vast industry, and the medical officers are the ones on whom, to a large extent, depends the efficient maintenance of this industry. I have explained the object of industrial medicine. In civil life, the sick or injured man is unable to work. The economic loss to him and his employer is great, to say nothing of the suffering and distress. In the Navy, the same applies, with the addition that this industry may be materially weakened in its defensive powers through the sick and injured. Any military organization is dependent upon its man power for its efficiency, both on defense and offense. A military organization whose men are sick or injured cannot hope to combat a force of men who are physically fit. It is therefore the duty of the medical officers of the Navy to keep the men in the highest form of physical fitness, in order that our Navy will be ready at any and all times to protect this country against attack. A thorough knowledge of industrial medicine is essential in keeping the men fit.

The industry, as I choose to call the Navy, in this paper, is composed of approximately 80,000 or 90,000 men, exclusive of civilians. These men join the industry from every part of the United States and its possessions. Before being accepted by the industry they must be examined by a naval medical officer. I wish right here to impress upon you the importance of this examination. Right here is the foundation of any industry. In civilian industries more stress is laid on the physical examination of prospective employees than any other thing. A man may be a first-class machinist, a blacksmith or other rating but if he is not physically fit he will not be accepted. Why? Every employer is required to insure his employees against disease or injury contracted by them while under his employ. It matters not whether an employee is injured through carelessness or from obvious physical weakness. The employer is responsible. If a man is injured because of poor vision the employer is responsible.

If this man be totally disabled, the employer must pay him compensation. This being the case, it is readily understood why so much attention is paid to the physical examinations by civilian employers. Why then, should the Navy fail in this particular.

While on duty at one of our training stations it was the duty of the writer to examine physically, all incoming recruits, i. e., the men who had previously been examined and accepted by medical officers at the various recruiting stations. It is not my purpose to criticize, but had any of my readers seen some of the physical specimens who were accepted at the recruiting stations, you, too, would know that some medical officers were not doing their duty. One case is recalled in particular. The man accepted was 73 inches tall; weight 118 pounds; undernourished; every vertebra protruded so that ulcers dotted the spinal column; pressure ulcers over the crests of the ilium, and had marked spasticity of the spinal column which prevented the man from bending over more than 10°. I cite this one example, among many, to indicate just what medical officers should be careful of. This man could have been of no value to the industry. Again, I say that the preliminary physical examination is important and that all men recruited should be as near physical fitness as is possible. I have heard medical officers state that they hardly knew just what to do with regard to rejecting men on account of physical unfitness for the reason that the recruiting station to which they were assigned had a certain quota to fill in a certain period of time. If they rejected many men the quota would not be filled and the recruiting officer would blame them. On the other hand if they tried to please the recruiting officer and allowed several men to pass, who did not meet the physical requirements and who later had to be invalided or surveyed from the service, that it became necessary for them to explain in writing to the Bureau. Under no circumstances should a physically unfit man be recruited. This Navy is one of the most important industries in the world and we cannot afford to weaken it with physically unfit men from the start. Quotas can always be filled later. If 100 men are recruited and half of this number are physically unfit, then the 50 physically fit men are of no value either, for they are required to do the work of 2 men in the place of 1.

The employment of a healthy working force and the elimination of the unfit, to my mind, is the best rule to follow in all organizations and especially the Navy. Aviation might be classed under industrial medicine for it is an industry. Today we have flight surgeons, whose duty it is to see that no one is allowed to fly a plane who is not physically and mentally sound. One flight surgeon stated that he rejected from 60 to 70 percent of applicants for pilot of planes. If we should allow every man to fly who wanted to, the mortality rate would be high indeed. And so it is in all trades. A man who is not

physically fit has no place in a hazardous trade. He is endangering his own life and the lives of others.

I have spoken of the Navy as an industry, for in the Navy are men of all trades and professions. The men come into the industry from all over the country. They are not all located in one town or factory but are scattered all over the world, ashore and afloat. They are subjected to every disease known to man because of this wide distribution. They are subjected to many hazards which lead to injuries, to which men in smaller industries are not. Many of these injuries are preventable and this is just what we must be on the alert for. I have told you of the necessity for recruiting physically fit men. If we do this, then we must keep them fit. You may ask, how am I, as a medical officer, interested or responsible for accidents, especially when such a large percentage are due to carelessness? This is one thing which I hope this paper will accomplish. It is too much to say, that we are responsible for all accidents but when we know that a big percentage of accidents can be avoided and that nothing has been done to prevent them, we are, in my opinion, just as responsible as if we had neglected to vaccinate our crew and smallpox developed among them. It is not enough to simply sit back and have your hospital corpsman come to you and tell you that quite a few men are waiting at sick call and he believes that they have food poisoning; that a man has just fallen down a defective ladder and injured himself or that there are several new cases of venereal disease aboard.

It is our duty as medical officers to inspect the food, the living quarters, to examine the men physically at various intervals; to talk with them regarding their troubles, both physical and mental and to see that galleys, mess halls, toilets, living quarters, and working stations are kept on a high scale regarding hygiene and sanitation. We must inspect all such things as clothing, ventilation, lighting, heating, etc. Medical officers must not only make such inspections but must make, and know how to make appropriate recommendations for improvements which will benefit the men and insure them good health.

Next, we are more or less responsible for the accidents at our ship or station. Many may not agree with this, but it is the opinion of the writer that it is true. It is the duty of the medical officer to cooperate with the commanding officer in an effort to do away with all hazards which will cause accidents. It is necessary that the medical officer be familiar with the ship or station on which he is serving, to know just what class of work is being done by the men and to be able to make intelligent recommendations regarding health and safety of the men under all working conditions. If a medical officer is with aviation he must know the physical fitness of all of the aviators and

must know the hazards of flying. If with submarines one must know the hazards of under-sea work including the rescue and deep-sea diving. If at a navy yard he must know the numerous hazards of the occupations of the civilians as well as the Navy personnel attached thereto. Again, it is not enough to know these hazards and just sit back and treat the injured as they are brought in. You must anticipate the accidents and make recommendations to prevent them. It is understood that all accidents cannot be prevented but certainly they can be reduced to a minimum. This can be done by educational talks, posters, and pamphlets and by inspiring a spirit of competition in the men. On many ships and stations the medical officers give frequent talks to the men on the subjects of personal hygiene and the dangers and prevention of venereal diseases. In the course of these talks an excellent opportunity is afforded to present the subject of accident prevention and its importance. When education and cooperation fail then it is the duty of the medical officer to see that careless practices which cause accidents are punishable, for it is just as reasonable to punish a man who has injured himself or others through neglect or carelessness, which causes him or others to lose time from their duties, as it is to punish a man who loses time through his failure to take a venereal prophylaxis and who develops a venereal disease.

Industrial medicine has now become a specialty. In the Navy today we have many other specialties. This is all well, but any specialist must know the conditions arising from industry, especially those encountered during the time of war. He must further have a knowledge of that branch of industrial medicine which deals with the rehabilitation of men wounded in battle. This was brought out during the last war, more than ever before.

The internist must be familiar with the diseases caused by the industry to which he is attached.

The eye, ear, nose, and throat specialist must likewise be familiar with the diseases caused to these organs, by dust, chemicals, lights, etc., to treat them intelligently.

The skin specialist also must know the effect of numerous trades on the skin and mucous membranes. The various trade eczemas are a specialty in themselves.

The pathologist and laboratory technician must assist all the former in arriving at a diagnosis. Unless he is familiar with the various pathological conditions arising in the trades, he is of little help. An example of this occurred recently at the Washington Navy Yard. A man reported with a severe dermatitis on both arms which he claimed was caused by the oils used on his lathe and with which his arms were in constant contact. A specimen of the oil was obtained and sent to the medical school for analysis. The analysis proved

that his condition was caused by a certain bacteria which had contaminated the oil. Accordingly corrective measures were taken to prevent the spread of the condition to other employees.

The genito-urinary specialist likewise comes in for his share. Recently an employee returned to work after having been out 2 months. His physician, a venereal specialist, had pronounced him fit for duty. When examined he was found to be suffering from cerebrospinal syphilis. His coordination was poor; he had Argyll Robinson pupils and was extremely nervous. This man was a lathe worker, which work requires the best of coordination. To have allowed this man to work at this trade would have been reprehensible, inasmuch as he would surely have injured himself or perhaps other workmen. His physician either did not understand the character of the work done by the man or else he saw little chance to obtain his fee unless the man returned to work and he was willing to take a chance. It does not pay to take a chance in the trades. This also serves to illustrate another point. The industrial surgeon would classify this workman and endeavor to give him a class of work with the least hazard, in order to make him self-supporting and not a public charge.

And so it is with all specialties. Industrial diseases and injuries must be understood by the specialist.

The writer will not attempt in this article to classify occupational diseases, for as yet there is no real definite means for classifying them. Kober, in his book states:

> An exact classification of occupational diseases is difficult as we yet have no accepted standard nomenclature for this class of diseases, nor do they differ essentially in the majority of instances from the pathology and clinical picture of diseased conditions in general.

It might be well to classify these conditions by cause rather than name. In the industry there are so many forms of eczema that some authors prefer to speak of the condition as trade dermatitis. Skin lesions and diseases in the industry are common, and idiosyncracy plays an important part.

By far the most important cause of occupational disease is industrial poisons. The Department of Labor lists from 50 to 60 poisons which are associated with diseases or injuries. It has been impossible to accurately determine the extent to which industrial poisons cause disability for in the majority of places it is not necessary to report such cases. It is sufficient to say, however, that these poisons are known to cause illness and injury to the body tissues and great progress has been made along lines of prevention. Other poisons will be found in later years by experience and these too will be cared for. Several years ago it was learned that the workers at the Washington Navy Yard who were engaged in the process of chrome-plating were

developing serious nose ulcers and perforation of the nasal septums. This has been corrected by an extensive exhaust ventilation system in this shop. These men can now do the work with little damage to their health.

Here again it is well for us to learn the various industrial poisons which our men are subjected to aboard ship and at stations and prevent illnesses and injuries from them, insofar as possible. Now, injuries of occupation are somewhat easier to classify. Entire volumes have been written recently on accidents and their prevention. The classification which I will submit here is one given by Dr. Kober in his book on industrial health and is considered especially clear and concise. It is practically impossible to list all hazards which will cause injuries and this applies to the Navy particularly, because of the wide distribution of its various activities and the numerous and varied duties of the men. The classification below is a general one but will aid medical officers, no matter what their duty is, if they will but make a thorough survey of their station and first become familiar with hazards to be reckoned with.

Industrial accidents may be classified into three groups, as follows:

(1) Those due to the physical conditions found in the working places.

(2) Those due to physical and mental conditions found in the working force.

(3) Those due to disaster, such as fires, lightning, explosions, cyclone, earthquake, etc.

The prevention of accidents must be done by—

(1) Protection against potential accidents by safety appliances placed about the working places or worn by the employees.

(2) A study of the cause of accidents and protection against recurrence.

(3) Supervision of the physical and mental conditions of all employees and correction of any cause for accident found in them. The removal of susceptible employees to work where no hazard exists. The safeguarding of fellow employees from accidents liable to result from defective workmen.

(4) Protection against disaster as far as possible and providing a means of escape in case of disaster.

To accomplish the aforementioned accident prevention there must be—

(1) Constant study and inspection of the physical conditions in the working places by the safety engineer, the medical staff, the management, and the employees.

(2) Careful study of each accident by the surgeon and the safety engineer to ascertain whether mechanical conditions or conditions in the employees or both were responsible and how a like accident may be prevented.

(3) Educational campaigns an accident prevention by lectures, bulletins, motion pictures, by safety committees among the employees, and by developing an atmosphere of prevention throughout the entire working force.

I cannot go into detail on this branch of the subject but I will point out a few of the physical conditions in working places that constitute accident hazards.

(1) Improper construction of buildings.

(2) Darkness or improper lighting.

(3) Unprotected or unguarded elevations, pits, elevator shafts, open hatches, etc.

(4) Temporary structures such as scaffolds, etc.

(5) Failure to keep buildings in repair (broken windowpanes, poor flooring, etc.).

(6) Loose articles left on floor such as crowbar, acids, nails, etc.

(7) Carelessness in stacking or stowing articles.

(8) The use of old or broken machinery.

(9) Lack of safety appliances on machinery and employees.

(10) Lack of proper clothing.

Disabilities due to physical or mental conditions of the employees as stated cause many accidents. Some of these defects are poor vision, loss of an eye, ear, leg, high blood pressure, uremia, syphilis, any nervous disorder which will cause a lack of concentration, epilepsy, and others too numerous to mention.

Under the classification of accidents due to disaster the most important is that of fire prevention, for this is one thing which causes more injuries that any other catastrophy.

A word might be said right here about the prevention of minor accidents. The same preventive measures outlined for the major injuries are applicable to the minor ones as well. However, every surgeon finds that it is almost humanly impossible to prevent this class of accidents. The next best thing to do therefore is to indoctrinate the men into the practice of reporting all injuries no matter how trivial, and having them attended to at the earliest possible moment. At the navy yard there are no first-aid boxes in any of the shops and none are allowed. Every man is instructed to report to the dispensary as soon as he is injured even though the injury be only a scratch or a minor bruise. If he does not do so and the wound becomes infected or aggravated in any manner he is not allowed compensation. In this manner we prevent many infections and keep the men from losing time on the job.

Under the heading of the spirit of prevention comes much well-used propaganda. Men must be educated to prevent accidents to themselves and to others. In every shop in the country today you will see posters published. One of the first attempts at using this means of

education was the stop, look, and listen signs posted all over the country some years ago to prevent railroad accidents. I venture to say that practically everyone in the country is familiar with this sign. It has educated people to be careful. There are many of such signs now in use today. This means of education has had a tremendous effect in helping to reduce accidents.

Thus far we have dealt chiefly with the principles and purposes of industrial medicine. To go on, we will try to go deeper into the subject and gain a working knowledge of this subject with especial reference to the United States Navy.

(To be continued)

CLINICAL NOTES

A REPORT OF FOUR CASES OF TUMORS OF THE GASTROINTESTINAL TRACT

By Commander R. H. LANING, Medical Corps, United States Navy, and Lieut. A. W. LOY, Medical Corps, United States Navy

It has been thought advisable to report for record four cases of tumors of the gastrointestinal tract because of their comparatively rare occurrence, and because of the special interest shown in these types of cases, in recent reports in the literature.

Case no. 1. Primary Sarcoma of the Colon.—E. E. F. (G. C. M. P.), age 21. Admitted June 22, 1931.

Chief complaint.—Cramping pain, left side of abdomen, nausea and vomiting, diarrhea.

Family history.—Acute rheumatic fever 1926. Tonsillectomy 1923. Denies venereal disease. No history of injury or severe illness.

Present illness.—About 3 weeks before admission, he began to have pain in left abdomen with loss of appetite. He was nauseated, and he vomited frequently, about 4 hours after meals. The pain in the abdomen has become more severe, and vomiting now occurs soon after meals.

Physical examination.—Well developed and well nourished young adult male. Height 67 inches, weight 145 pounds.

Eyes, ears, nose, and throat.—No disease.

Neck.—Normal.

Chest.—Heart and lungs are normal. Blood vessels are normal.

Abdomen.—Scar of left inguinal herniorraphy. A small umbilical hernia is present. No areas of tenderness. No palpable abnormal masses.

Genitalia.—Normal.

Extremities.—Normal.

Urinalysis shows one plus albumin. Blood Kahn test is negative. R. B. C., 5,760,000; H. B., 100 percent; W. B. C., 14,100. P. M. N., 58; band forms, 5; young forms, 7; monocytes, 2; lymphocytes, 28.

Coagulation time, 2¼ minutes.

Feces are negative for ova and parasites, and occult blood.

On July 29, 1931, repair of umbilical hernia was done, without relief of symptoms.

On August 8, 1931, under spinal anesthesia, exploratory laparotomy was done and a tumor of the upper sigmoid colon was found. A first-stage Mikulicz's operation for excision of the sigmoid was done.

On August 10, 1931, a second-stage operation for removal of the sigmoid was done under spinal anesthesia.

On August 10, 1931, X-ray examination of the abdomen showed a marked gaseous distension of the colon.

On August 18, 1931, a caecostomy was done.

Histopathological report.—Sections made through the tumor mass removed from the sigmoid colon show an intact mucous membrane. Lying immediately be-

neath this, however, is a mass of cells of lymphoid origin. The individual cells are rather small in size, the nuclei are small and many show a pronounced hyperchromatism and there is a relatively liberal amount of cytoplasm. Many eosinophiles are scattered throughout the whole intestinal wall. In the sections examined the tumor tends to spread laterally just beneath the mucosa.

Diagnosis—Lymphosarcoma.—Convalescence following the series of operations was uneventful. The caecostomy and colostomy functioned well and all symptoms of obstruction subsided.

On September 4, 1931, the patient was transferred to the United States Naval Hospital, San Diego, Calif., where he received a series of 16 X-ray treatments between September 29, 1931, and October 26, 1931. Exposures were made in rotation over left anterior, right anterior, left lateral, and left posterior abdominal areas. Each treatment consisted of: 200 K. P. V. ------ 5 M. A. ------ 50 cm dist. ------ ½-cu/1-al M. M. filter ------ 15 minutes. He was discharged from treatment on March 2, 1932.

The onset of colicky pain followed by symptoms of chronic obstruction, nausea, and vomiting, as presented in this case, are the usual symptoms found in a case of sarcoma of the intestine.

Final diagnosis is made only by histopathological examination.

The treatment in such cases, is early resection of that portion of the intestine involved. Irradiation is an aid in treatment, as a supplement to surgery, but it is never a substitute for surgery.

Boyce and McFetridge, (1) in a recent review of primary sarcoma of the intestine, gave the number of reported cases, up to March 1934 as about 300. The ratio of incidence of sarcoma to carcinoma of the intestine is about 1 to 20. Furthermore, the case reported is one of sarcoma of the large intestine, whereas, sacroma is more often found in the small intestine, particularly in the ileum.

Farr, (2) in 1931, showed that only 35 percent of sacormata of the intestine is found in the large intestine, whereas 95 percent of carcinomata of the intestine is found in the large intestine. The ratio of incidence of sarcoma to carcinoma of the large intestine is about 1 to 55.

Lymphosarcoma usually arises in the submucosa, replaces the muscularis and subserosa, laterally, and the submucosa longitudinally. The mucosa and serosa are involved, late, if at all, so that the walls of the intestine become thickened and rigid. Direct permeation of the surrounding tissues occurs early. Metastasis occurs by blood stream through the portal system, to the liver.

Case no 2. Carcinoma of the third portion of the duodenum.—C. M. C. M. M. (F. N. R.), age 47. Admitted February 23, 1932.

Chief complaint.—Pain in epigastrium. Pain following meals. Loss of weight.

Nationality.—White, American.

Occupation.—Machinist.

Family history.—Father died, influenza, age 62. Mother died, childbirth. Wife living and well. Two children. No history of familial diseases.

Past history.—Measles, mumps, chicken pox in childhood. Typhoid fever at age 13.

Present illness.—In September 1930 he had an attack of severe epigastric pain, after eating walnuts. Since that time, pain recurred about one-half hour after ingestion of food. In August 1931 he entered the hospital for 1 month. At that time the stools were positive for occult blood.

The Histamine test for gastric secretion at that time was as follows:

No.	C. C.	Free H. C. L.	Total acidity	No.	C. C.	Free H. C. L.	Total acidity
1	32	35	42	4	20	17	29
2	23	42	51	5	10	5	15
3	20	32	44	Fasting	94	0	11

He was treated for duodenal ulcer with temporary relief, on a sippy diet. After leaving the hospital, symptoms continued and became worse with radiation of pain to the right shoulder.

Physical examination.—Revealed a well developed adult male, poorly nourished, and showing evidence of a recent loss of weight. Height 65 inches, weight 124 pounds (usual weight 162 pounds). Temperature, normal, pulse 90.

Head.—Normal.

E. E. N. T.—No diseases.

Neck.—Normal.

Chest.—Heart, normal. Blood pressure 120/84. Lungs, normal.

Abdomen.—There is tenderness, over epigastrium, which is not well localized.

Genitalia.—Normal.

Extremities.—Normal.

Laboratory reports.—R. B. C., 4,220,000; H. B., 80 percent; W. B. C., 7,950. Neutrophiles, 61; basophiles, 1; band forms, 2; polymorphs, 59; lymphocytes, 38. Blood Kahn test, negative. Urinalysis, normal findings. Stools, positive for occult blood. Gastric secretion (Histamine test).

No.	C. C.	Free H. C. L.	Total acidity	No.	C. C.	Free H. C. L.	Total acidity
1	10	0	11	4	12	0	9
2	12	0	14	5	14	0	10
3	10	0	14	Fasting	78	0	11

Electrocardiograph shows right ventricular extra systoles.

X-ray report upon a gastrointestinal series following a barium meal showed evidence of colonic irritability. There was no evidence of organic disease of the stomach, and no evidence of duodenal ulcer.

Under spinal anesthesia, exploratory laparotomy was done on March 15, 1932. A dilated, chronically inflamed, gall bladder was found. The liver had a mottled appearance. No pathology of the stomach and duodenum was found. A cholecystostomy, with drainage, was performed.

Post operative condition showed no change of symptoms. Nausea and vomiting were relieved by duodenal drainage by Rehfuss tube.

On March 25, 1932, a posterior gastroenterostomy was done. At the time of operation, hypertrophy of the pyloric sphincter was noted. No other evidence of disease of the duodenum was found.

Following gastroenterostomy there was relief from pain, nausea, and vomiting. The patient was comfortable and showed general improvement until the twelfth post operative day, when he suddenly developed acute dyspnoe, fell into a coma and died of myocardial failure.

Necropsy protocol.—The body is that of a fairly well-developed amaciated white male, that appeared to be about 55 years of age. Two practically healed operative scars just to the right of the midline above the umbilicus. Skin clear. Straight necropsy incision.

Chest.—Lungs fill the pleural cavities. No excess fluid. No adhesions. Rather marked anthracosis. Cut section showed no oedema or congestion. Pericardium normal. Heart about normal in size, somewhat fatty. Myocardium showed a marked fibrosis. Valves quite sclerotic. Coronary arteries very sclerotic. Lumen small. Aorta showed rather numerous atheromatous plaques.

Abdomen.—Omentum and peritoneum adhered to margins of liver and to several loops of intestine. Stomach large and filled with fluid and food. Oesophagus quite large in diameter. Upper two-thirds of duodenum markedly dilated. Part of the middle third and lower third adhered around the head of the pancreas. The third portion of the duodenum practically completely stenosed by an ulcerated mass about 5 cm long and completely encircling the lumen. The lumen in this tumor barely admitted a grooved director. Recent gastroenterostomy short circuited the loop containing the tumor. Gastroenterostomy in good condition. Liver somewhat large. Acutely congested. Spleen normal in size, somewhat fibrous. Both kidneys about normal in size. Capsules would not strip. Markings obliterated but surfaces dark red. G. U. tract practically normal. Calverium not opened.

Gross diagnoses:

(1) Carcinoma, third portion of the duodenum.
(2) Myocarditis, chronic.
(3) Coronary sclerosis.
(4) Arteriosclerosis, general.
(5) Nephritis, chronic.
(6) Dilation of G. I. tract above tumor.

Histopathological report.—The sections were made through the duodenal tumor and show an ulcerated epithelial surface underlying which anaplastic epithelium, reproducing glandular structure, is to be seen. The malignant cells in one area have infilterated the entire wall of the duodenum and are distinctly malignant with marked hyperchromatism and loss of polarity. A high degree of inflammatory reaction is present throughout the section.

Diagnosis.—Adenocarcinoma of duodenum.

Primary carcinoma of the duodenum is rare, being found in 0.033 percent of autopsies; and of the three divisions of the duodenum, it is most rarely found in the third portion. The usual type is the cylindrical cell adenocarcinoma which tends to encircle the intestine and to cause intestinal obstruction.

Metastasis from this type of carcinoma occurs late.

The symptoms are those of chronic upper intestinal obstruction, with blood in the stools. X-ray series after a barium meal will usually show defects in gastric carcinoma and in carcinoma of the first portion of the duodenum. Free H. C. L. disappears early in gastric carcinoma, and later in carcinoma of the duodenum. Bile and pancreatic juice may occur in the vomitus; and jaundice may appear in obstruction below the first portion of the duodenum.

Diagnosis of carcinoma of the third portion of the duodenum offers the most difficulties of all cases of carcinoma of the grastrointestinal tract.

Case no. 3. Nonspecific granuloma of the caecum.—J. F. P. (V. A. P.), age 37. Admitted, November 5, 1932.

Nationality.—White, American.

Occupation.—Salesman.

Chief complaint.—Pain in abdomen, localized in right lower quadrant.

Past history.—Not significant.

Family history.—Irrelevant.

Past illness.—About 4 months ago the patient had a sudden attack of pain in right lower abdomen. The attack subsided after several hours. Since that time, there have been recurring similar attacks of acute pain in the right lower abdomen. No nausea. No vomiting. No diarrhea. No constipation.

Physical examination.—The patient is a well-developed and well-nourished adult male. Temperature, 98.4°. Pulse, 90.

Head.—Normal.

Eyes.—Normal.

Nose.—Normal.

Ears.—Normal.

Pharynx.—Tonsils are large and cryptic.

Neck.—Normal.

Thorax.—Normal.

Heart and lungs.—Normal.

Abdomen.—Normal contour. No palpable abnormal masses. There is tenderness to deep palpation over region of the caecum.

Genitalia.—Normal.

Extremities.—Normal.

Urinalysis showed normal findings. W. B. C. 10,850. P. M. N. 78 percent.

Blood Kahn test.—Negative.

Pre-operative diagnosis.—(1) Appendicitis, chronic. (2) Tonsillitis, chronic.

On November 6, 1932, at operation, infiltration of the walls of the caecum was found. Anastomosis between the ileum and the transverse colon was done, and the terminal ileum, the caecum, and the ascending colon were excised.

Histopathological report.—Sections taken from various areas present the same microscopical picture, the mucus membrane is intact and is of adult type of cells. The walls of the caecum are edematous with infiltration of inflammatory cells. The blood vessels are congested. There is no evidence of malignancy.

Diagnosis.—Inflammation subacute, caecum (nonspecific) typhlitis.

Convalesence was uneventful and the patient was discharged to home on December 6, 1932.

A recent report from the patient shows that about 8 months after operation he had an attack of abdominal pain with acute diarrhea, which subsided without sequelae. At 18 months after operation, the patient is free of symptoms and carrying on his usual occupation.

Nonspecific granuloma of the ileum has been established as a clinical entity by reports of several individual cases. Ginsburg and Oppenheimer (2) referred to the condition as "regional ileitis." The condition is usually confined to the terminal ileum.

Dr. Ralph Colp (1) in April 1934 reported an unusual case of nonspecific granuloma of the terminal ileum which extended beyond the ileocaecal valve and involved the caecum. In this report we wish to add another case of this unusual type of granuloma about the terminal ileum and the caecum.

The case reported here was evidently an early case. The usual finding of ulceration of the mucosa were not present in this case, but the microscopial findings of the intestinal walls were similar to those of previously reported cases. The case substantiates the belief of Dr. Colp and others that nonspecific granuloma of the intestine is not limited to the terminal ileum.

Case no. 4. Neurofibromatosis with visceral manifestations.—N. A. P. (V. A. P.), age 43. Admitted United States Naval Hospital, November 10, 1932.

Chief complaint.—Abdominal distress, eructation, gas on stomach, and epigastric pain.

Family history.—Not significant.

Past history.—Multiple subcutaneous tumors have developed since 1919. No symptoms from these tumors. No serious illness previous to 1919. Denies venereal disease. In 1919 attacks of epigastric distress began, and recurred at irregular intervals. Usually the attacks followed meals and were not relieved by taking soda bicarbonate.

Present illness.—During the past year the attacks of epigastric pain have occurred more frequently, and they have been more severe with attacks of vomiting, following meals. A milk diet relieves symptoms. The patient is free of symptoms before breakfast and on a fasting stomach. About 2 hours after meals, he has epigastric pain, gas on stomach, eructation, and occasional vomiting. No hematemesis.

Physicial examination.—Well nourished, well developed adult male, not acutely ill. Height 68 inches, weight 157. Multiple subcutaneous tumors, varying from pea size to the size of a hen's egg are present, over face, shoulders, chest, back, abdomen, arm's and legs.

Head.—Normal.

Eyes, ears, nose, throat.—No disease.

Chest.—Heart and lungs are normal. Blood pressure 110/74, pulse 72.

Abdomen.—No areas of tenderness. No palpable masses.

Genitalia.—Normal.

Extremities.—Right thumb and ring finger missing (explosion). Reflexes are normal. Rhomberg is negative.

Laboratory examinations:

Urinalysis.—Normal findings.

Blood Kahn test.—Negative. R. B. C. 3,800,000, H. B. 61 percent (dare), W. B. C. 13,300, P. M. N. 67, Lymph. 28, Mono. 3, band forms 2.

Coagulation time.—3½ minutes.

Stools.—Positive for occult blood.

Histamine test.—Normal gastric secretion.

Report of biopsy of subcutaneous tumors.—All sections present the same appearance of bundles of hyaline tissue, between which there are concentric whorls of cellular tissue. The tumor is vasular. An occasional sebaceous gland is present. There is no evidence of malignancy.

Diagnosis: Neurofibromatosis (Von Recklinghausen's disease).—X-ray report: The roentgenological findings in a G. I. series after a barium meal, in this case, are definitely those of organic obstruction near the pyloric ring. The stomach is markedly dilated.

On January 7, 1933, at operation, the pyloric orifice was found to be practically occluded by a dense fibrous type of tissue. The same type of tissue was found in the gastrohepatic omentum.

A partial gastrectomy and anastomosis by the method of Bilroth was done.

The post-operative condition was good, and the convalescence of the patient was uneventful.

The histopathological report was as follows: The mucus membrane is apparently normal. Eosinophiles are present in the deep layers of the tunica propria. The submucosa is not remarkable. The muscle layer shows replacement by cellular connective tissue. In a few of the concentric whorls, occasionally, nerve cells can be distinguished. One type of nerve cell presents a large vesicular nucleus. A second type of cell presents a large, wavy nucleus. Eosinophiles are abundant throughout.

On March 4, 1933, the patient was discharged to home, symptom free.

A recent report from the patient shows that after 18 months there has been no recurrence of symptoms of pyloric obstruction.

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POLYORRHOMENITIS; A CASE REPORT

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Since the publication by A. O. J. Kelly (*1*) in 1903 of a monograph reviewing the subject of multiple serositis, very few cases have been reported, which doubtless accounts for the fact that little contributory thought has been added to his conclusion that Concato's disease must be differentiated from serositis of syphilis and of tuberculosis.

In a recent review of the subject the paucity of such case reports since 1917 is noteworthy and the analyses disappointing from the standpoint that no clear distinction is made between Pick's disease and the polyorrhomenitis described by Concato and bearing his name. William J. Mayo, in his discussion (*2*) of Concato's disease and after reviewing a series of case reports, concludes that serositis, wherever found in the body, is a manifestation of the same disease or process. Some classification of the inflammatory reactions of serous membranes based on etiology rather than on the degree of involve-

ment or on the specific membranes involved is believed to be indicated. Undue emphasis seems to have been placed on local thickenings of the spleen capsule, on "Zuckerguss leber", and on the hyalinization and hypertrophy of peritoneum or pleura as well as on the logical sequence of mediastino-pleuro-pericardial adhesions as manifested in congestion and pseudo cirrhosis of the liver.

It would appear that a clearer and more logical description of the various types of polyserositis might be found in a classification based on causes and that the concept of Pick's disease, or Concato's disease, as clinical entities might well be abandoned.

A basis for such a classification was suggested by Kelly 30 years ago, and a recent case which came to autopsy at this hospital emphasized the need for a system of clinical pathological differentiation.

CLINICAL REPORT

T. L. H., a Negro male, aged 46, was admitted to the hospital complaining of "rheumatism" of the joints of the lower extremities, "feeling as though he had no blood", cold feet, and diarrhea.

Family history.—Unimportant.

Personal history.—Not important.

Present illness.—Began about 4 months prior to admission to this hospital. The illness began with loss of weight, lassitude, and loss of appetite. His normal weight approximates 155 pounds, although on admission he weighs only 98 pounds. Diarrhea gradually developed during the past year and he was admitted to a New Jersey hospital 3 weeks prior to his admission here. He stated that he had been treated there for "rheumatism and pains in his abdomen."

Physical examination.—Patient is emaciated and has the appearance of a man 20 years older than his actual age. He is apparently dehydrated. Pupils equal and active. Marked arcus senilis. Throat shows a moderately pale mucosa. Teeth show many carious areas and several teeth are missing. Tonsils are hypertrophied and cryptic. There are a few shotty posterior and anterior cervical glands. Chest: No pathological signs elicited by percussion or auscultation. Chest expansion free and equal. Heart: There is a soft systolic murmur at the apex transmitted outward toward the axilla. No enlargement to percussion. Pulse weak and thready. B. P. 70/50. Abdomen: Flat and soft, no masses felt, no tender areas. Testes show gross atrophy. Temperature, pulse rate, and respiration normal. Except for extreme emaciation, the examination revealed nothing further of note.

Neuropsychiatric examination.—Essentially negative.

Laboratory findings.—R. B. C. 4,380,000, Hgb. 88.8 percent, W. B. C. 8,000, segmented 50 percent, lymph. 48 percent, eos. 2 percent. Urine: Albumin, plus-minus, specific gravity 1.024, Ph. 6, no casts. Stool: Occult blood a trace. Ova and parasites negative. Blood Kahn negative.

Patient's condition progressed steadily downward. Although the intake of fluids and soft diet was in good quantity, the dehydration continued. Bowel movements were 3 or 4 daily until on March 2, 1933, 14 days after admission, there were almost continuous involuntary bowel movements, which were only slightly checked by treatment.

Death occurred March 2, 1933. Patient was conscious until a few moments preceding death.

The red blood cells remained around 4,370,000 per cubic millimeter, Hgb. 88.8 percent, and the white blood cells and differential count continued the same as on admission. The gross atrophy of the testes and general dehydration with essentially normal blood count were considered the most significant features in this case.

Clinical diagnosis.—1. Colitis, chronic. 2. Inanition.

AUTOPSY REPORT

General appearance.—The body is that of an emaciated Negro male of the apparent age of 60 years, height 67 inches. There is a marked arcus senilis of both eyes and the hair is gray.

Rigor mortis is moderately well developed.

Inguinal lymph nodes are moderately enlarged.

Lungs.—The visceral pleura is adherent to the parietal pleura by dense, dry, and translucent adhesions over the entire surface of the right lung. The lower lobe of the right lung is adherent to a small portion of the external surface of the pericardium. The visceral pleura is markedly thickened and on section the lungs are congested and edematous. All lobes of each lung are intimately bound to one another by dense, white, and dry adhesions. The lungs are subcrepitant throughout. No areas of consolidation are noted. Posterior mediastinal lymph nodes are hypertrophic and dark in color on section. The anterior mediastinum contains dense, white, and dry adhesions binding the pleura to the sternum and costal cartilage.

The right lung weighs 580 grams, the left lung 540.

Heart.—The pericardium is smooth and glistening and adherent on the surface to the pleura at one point. The sac contains about 10 cc of clear straw-colored fluid. The heart is small with no evidence of any pericarditis. The muscle is normal. There is no evidence of endocarditis. Slight wrinkling of the thoracic aorta is noted, but the wall is normally elastic. All valves are normal, the coronaries are patulous with no evidence of sclerosis.

The left ventricle wall measures 1.8 centimeters in thickness, the right ventricle wall 0.2 centimeter in thickness. The nitral valve measures 9½ centimeters in circumference, the aortic valve measures 7½ centimeters in circumference, the tricuspid valve measures 11 centimeters, and the pulmonic valve 8½ centimeters in circumference. The heart weighs 240 grams.

Abdominal cavity.—The intestines are firmly bound to the parietal peritoneum and loops are bound to each other by dry, translucent, and firm adhesions. The hepatic flexure of the colon is intimately bound to the lower surface of Glisson's capsule.

Liver.—Glisson's capsule is intimately bound to the surrounding structures—the diaphragmatic peritoneum, hepatic flexure of the colon, to the lesser curvature of the stomach, and to the duodenum. Glisson's capsule is moderately thickened. Color of the liver on the surface is dark red to purple. On section the lobular markings are distinct.

There is a moderate degree of passive congestion and some evidence of granular degeneration in the parenchyma. The liver weighs 1,450 grams.

The gall bladder contains about 4 cc of canary yellow bile. The bile ducts are patent and the mucosa is essentially normal.

Spleen.—The capsule is thickened and adherent to the diaphragmatic pleura. It is red in color on the surface and on section is dark garnet. An increased amount of fibrous connective tissue and passive congestion are noted. The spleen weighs 160 grams.

Pancreas.—The pancreas is essentially normal in appearance and weighs 95 grams.

Gastro intestinal tract.—All loops of the small intestines are firmly bound to each other by dry, tenuous, and translucent white adhesions. Moreover, the colon and stomach are similarly bound to surrounding structures and to the parietal peritoneum, so that traction on one organ moves the entire contents of the abdomen. The serosa of the intestines is remarkably dry and dull in appearance. The stomach is essentially normal. Throughout the duodenum, jejunum, and ileum there are scattered areas of petechial hemorrhage in the submucosa and the mucosa is occasionally coated with a pink mucus material. The mucosa of the colon below the sigmoid is smooth and studded under the surface with small white circular areas which do not protrude above the surface.

Kidneys.—The left kidney is dark red in color on the surface. The capsule strips with slight difficulty, leaving, however, a smooth glistening surface. The stellate veins are prominent with marked passive congestion evident. The cortico medullary ratio is 1:2½. The left kidney weighs 100 grams.

The right kidney is essentially similar to the left kidney and weighs 140 grams. Both show a moderate degree of lobulation.

Adrenals.—Both adrenals are atrophic and the gross appearance indicates total loss of lipoid content.

The prostate and urinary bladder are essentially normal.

The right testicle is removed and appears atrophic. The parenchyma does not string. Fibrous connective tissue is moderately increased.

Thyroid.—Both lobes are one-third the normal size, dark brown in color, and cut with increased difficulty. On section there is increased fibrous connective tissue and the surface is lusterless and light brown in color.

Brain.—On opening the cranial cavity, it is noted that the meninges are moderately injected. The under surface of the temporal lobe is flattened. There is increased congestion in the region of the circle of Willis and the fourth and fifth ventricles and upper portion of the spinal cord show edema. The fossa hypophyseos is moderately enlarged. The pituitary is removed and is larger than normal, measuring 1.4 centimeters in diameter by 0.6 centimeter thick. On sectioning the brain, the ventricles are moderately swollen and contain an increased quantity of fluid.

Diagnosis.—

(1) Polyorrhomenitis involving mediastinum, pleura and peritoneum (Concato's disease).
(2) Ileo colitis chronic.
(3) Atrophy of adrenals and testicles.
(4) Involution of thyroid gland.
(5) Hypertrophy of pituitary gland.
(6) Congestion and edema of meninges.
(7) Dilatation of all ventricles of brain.

MICROSCOPICAL EXAMINATION OF TISSUES SHOW

Lungs.—There is moderate congestion throughout with occasional areas of oedema. A few pigment laden histiocytes are noted in the alveoli. The bronchi are essentially normal. The pleura is irregularly thickened in all sections examined by reason of increased fibrous connective tissue. No inflammatory cell infiltration is noted.

Aorta.—The aorta shows a moderate degree of atheromatous degeneration of the intima.

Heart.—The myofibrils show a moderate degree of granular degeneration with brown pigmentation at the nuclear poles. The pericardium is thickened by

ATROPHY OF THYROID GLAND.

HYPOPHYSIS SHOWING PARS DISTALIS, PARS MEDIA AND PARS NERVOSA.

reason of a reticular mass of fibrous connective tissue. The interstitial fibrous connective tissue shows a moderate degree of hypertrophy and fibroblastic proliferation in scattered areas of the sections examined. The vessel walls are essentially normal although the vascularity of the tissue appears to be decreased.

Liver.—Glisson's capsule is thickened by increased and hyalinized fibrous connective tissue. The capsular arteries show medial sclerosis and hyalinization. The sinuses are dilated and show marked passive congestion. The polygonal cells show moderate granular degeneration and there is a slight increase of interlobular fibrous connective tissue.

Spleen.—The capsule and trabeculae are thickened by increased fibrous connective tissue and the sinuses are dilated by a moderate degree of congestion.

Pancreas.—The secretory elements are essentially normal. There is an increase of fibrous connective tissue between the lobes and in the capsule. The lumina of medium sized arteries and of arterioles are narrowed by medial sclerosis of the walls.

Gastro-intestinal tract.—The wall of the colon, taken from the area described as having a smooth mucous surface, shows absence of lining mucosa. The tubular mucosa is atrophic and shows granular degeneration. The submucosa contains several hypertrophic lymph follicles with moderately active germinal centers and is thickly infiltrated with plasma cells, lymphocytes, eosinophiles and shows fibroblastic proliferation. Between the muscularis mucosa and muscularis there is a thickened layer of fibrous connective tissue with considerable fibroblastic proliferation. The muscularis is essentially normal, but the serosa is markedly and irregularly thickened by reason of increased fibrous connective tissue.

Kidneys.—There is marked passive congestion throughout. The medium-sized artery walls show medial sclerosis and hyaline degeneration. There is an occasional obliterated and hyalinized glomerulus, and the tubular epithelium is essentially normal.

Adrenal glands.—The adrenal glands show almost complete loss of lipoid from the cortical cells. The medulla is contracted, and no brownish pigment is evident. The capsule is moderately thickened by increased fibrous connective tissue.

Pituitary gland.—The pars nervosa is larger in comparison to the pars distalis than is normally seen, measuring 0.35 centimeter in diameter to 0.5 centimeter in diameter for the anterior portion in the shrunken microscopic section. Very few chromophobe cells are noted in the anterior portion and while the periphery contains many basophilic cells, the large majority of glandular elements are eosinophilic. The pars intermedia is essentially normal, containing a few acini with flattened epithelial cells surrounding a palestaining colloid.

In the pars nervosa there is moderate increase of vascularity, and there is a considerable quantity of brown pigmentation seen here and there amid the glial fibers. In the central portion of the posterior pituitary there is a large area occupied by large eosinophilic cells, polygonal in shape and having eccentric vesicular nuclei with considerable chromatin content and with an occasional nucleolus. These cells are occasionally multinucleated and the cytoplasm is granular and eosinophilic. They resemble, to a certain extent, those seen in the pars distalis but do not form the tubular glands which are sometimes seen in this portion, being arranged, rather, in irregular strands in a glial fibrous network.

Thyroid gland.—The acini lie widely spaced in a loose, fibrous connective-tissue reticulum and are surrounded by a thickened connective-tissue capsule. The colloid stains a moderately deep pink, is granular in some acini, and in others shows oval and circular areas of vacuolation in the center of the colloid. The epithelial lining is cuboidal and flattened in type. The medium-sized artery walls in the capsule show medial sclerosis and intimal proliferation.

Cerebellum.—The meninges and cortex show dilation and congestion of the blood vessels, combined with a moderate degree of edema.

Testicle.—The tunica albuginea is thickened to approximately twice its normal width by increased fibrous connective tissue. The spermatogenic elements are widely spaced in a loose connective-tissue reticulum and the cells of Sertoli show granular and fatty degeneration and in many instances are free of the basement membrane. The immature spermatocytes show granular degeneration of the cytoplasm and pyknosis of the nuclei.

Only a few islands of interstitial cells of Leydig are seen, and these show atrophy and granular degeneration.

Diagnosis.—

(1) Polyorrhomenitis (Concato's disease).
(2) Arteriosclerosis (Monckeberg type).
(3) Atheromatous degeneration of aorta.
(4) Mucus colitis subacute.
(5) Atrophy of testicle, thyroid, and adrenal medulla.
(6) Hypertrophy and hyperplasia of hypophyseal pars nervosa.
(7) Congestion and edema of meninges and cerebellar cortex.
(8) Passive congestion of lungs, liver, spleen, and kidneys.

COMMENT

The polyglandular dystrophy was doubtless the underlying cause of the faulty distribution of water and consequent dryness of the serous membranes, as explained by Elwyn in the following paragraphs of his book (3):

Of the glands of internal secretion it is especially the hypophysis which influences the water exchange. The intermediate and posterior lobes secrete a substance which produces a change in the colloidal system, and with it a change in the distribution of electrolytes of the cells, especially of those organs which form part of the mechanism for water regulation. We may consider that the center for water regulation (the hypophysis) influences the whole mechanism of water distribution and exchange through two channels: First, by impulses reaching the various parts of the whole mechanism, through nervous connection, by way of the vegetative nerves; second, by impulses through nerve fibers reaching the intermediate and posterior parts of the hypophysis and influencing the internal secretion.

Elwyn also observes that either more or less than the normal amount of water may be contained in the body cells, dependent on the action of the mechanism described.

It is believed that the serous membranes of this patient were affected by an abnormal influence from the hyophysis and tuber-cinereum so that the ability of the serous membrane to retain water was seriously altered, and that this dryness of the contiguous membranes resulted in irritation and consequent adhesions.

It is noted that, in a case of multiple serositis reported by Evans, (4) there was a history of rheumatism and myocardial degeneration. Boyd's contention (5) that rheumatism manifests itself in allergic reactions of serous surfaces provides us with an explanation for another type of polyserositis and this theory is further supported by

Dr. Mallory's analysis of a case reported in the New England Journal of Medicine (6) when he observed that slight degrees of multiple serositis are common in rheumatism and may predominate throughout the course of the disease.

Summary.—A case of multiple serositis is presented and an attempt made to show that the adhesions were the result of faulty water distribution, which in turn was due to hypophyseal dystrophy.

A review of the literature on the subject of polyserositis indicates that serous thickening and adhesions are the result of chronic inflammatory processes, allergic phenomena and faulty water distribution.

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(1) A. O. J. Kelly: On Multiple Serositis, American Journal of Medical Science, 1903, vol. CXXV 116–148.

(2) Mayo: Concato's Disease, American Surg. 1922 LXXVI 432.

(3) Elwyn: Edema and its Treatment.

(4) Evans: Multiple Serositis, American Journal of Medical Science, Phila., 1918; 553.

(5) Boyd: Pathology of Internal Diseases.

(6) Polyserositis Subacute; New England J. Med. 201–588, 1929.

ANURIA FOLLOWING THE ADMINISTRATION OF NEOARSPHENAMINE

Report of Case

By C. L. Andrus, Lieutenant Commander, Medical Corps, United States Navy

The patient was admitted to the United States Naval Hospital, Washington, D. C., as a stretcher case at 9 a. m. on June 1, 1933, with the diagnosis of syphilis.

Chief complaint.—Unable to urinate since 10:45 a. m., May 30, 1933; nausea and vomiting.

Family history.—Father died at 55 following a "stroke." Mother alive and well. Nine other children in the family, all alive and well. Married in 1911, wife alive and well. Two children, a girl 16 and a boy 13, both well.

Past history.—(Diseases) measles, mumps, chicken-pox, and typhoid fever in childhood. Gonorrhea 1918. Syphilis 1929. Malaria 1931. (Injuries.) Ventral hernia since 1918 which followed a blow in the abdomen received during a shipwreck. Operations: none.

According to the patient's statement the diagnosis of syphilis was made in September 1929 and was based upon a blood test reported 4 plus. The place and date of acquiring the infection are unknown. The medical record states that there was no evidence of chancre or other initial lesion and no evidence of a skin manifestation. The infection was supposedly congenital in origin. This supposition does not seem consistent with the family history. After the diagnosis was made treatment was instituted and following the sixth injection of "salvarsan" he had a severe reaction. Since that time he has received bismuth and mercury but no arsenical treatment until May 9, 1933, when the present course was started.

Present illness.—The onset and course of the present illness up to the time of admission into the hospital are given in the Health Record as follows: "About April 15, 1933, the patient developed a skin ulcer on the anterior surface of his left leg which failed to respond to usual local treatment. On April 25, 1933, a

Kahn blood test was reported as 2 plus and a Wassermann blood test was reported positive. For this reason arsenical treatment was instituted on May 9, 1933, at which time he received a 0.2-gram intravenous injection of neoarsphenamine. On May 16, he received a 0.4-gram intravenous injection and on May 23, and May 30, 1933, he received 0.6-gram intravenous injections of neoarsphenamine. The skin ulcer showed improvement shortly after the first injection of neoarsphenamine and it was entirely healed after the third injection of neoarsphenamine. As concurrent treatment the patient was administered one-fifth grain intramuscular injections of mercury succinimide on May 12, 15, 19, 22, 26, and 29, 1933. The dilution of the last injection of neoarsphenamine was 0.6 gram of neoarsphenamine dissolved in 20 cubic centimeters of sterile water. The rate of injection was stated as approximately 6 or 7 minutes for the 0.6 gram dose administered.

"Approximately 12 hours after the last injection of neoarsphenamine the patient stated that he developed a headache, slight nausea, and loss of appetite. He had the desire to urinate but could not void. As treatment the patient was given forced fluids which consisted of 6 ounces of water every hour. At about 9:30 a. m., May 31, 1933, the patient was catheterized using a number 10 soft rubber French catheter which was passed with only slight difficulty. No urine was obtained. At 10 a. m. the patient was given an intravenous injection of 1 gram of sodium thiosulphate. At 10:30 a. m. he was given 1 dram of potassium bitartrate in a pint of hot water. At about 12:30 p. m. he vomited about 500 cubic centimeters of bile-colored fluid and shortly thereafter he had a large watery bowel movement. At 1 p. m. he was given a pint of warm saline solution as a high colonic irrigation. Thirty minutes later he had a constipated bowel movement. At 8 p. m. he voided about 50 cubic centimeters of blood-tinged urine."

Physical examination.—Forty-three years of age. Stated weight 160 pounds. His temperature was 98.4° F., pulse 54 and respiration 16. He presented himself as a well-developed, well-nourished colored man who had hiccoughs and who appeared to be somewhat weakened and anxious but otherwise not ill. No icterus of the sclerae was noted. His pupils reacted to light and accommodation. The axillary and inguinal lymph glands were palpable. Heart sounds were normal. Blood pressure was 140/70. Breath sounds were normal. His abdomen was essentially negative, there was no tenderness over either costo-vertebral angle or the kidneys. A small ventral hernia in the epigastrium was noted. Pressure over the bladder region caused a desire to urinate, although the bladder was not distended. His prostate was not enlarged or tender. Examination otherwise negative.

Progress notes.—He was admitted to the urological service and placed on a liquid diet with orange juice and water in small quantities as frequently as he could take them without inducing vomiting. Soda bicarbonate was given by mouth. A soap-suds enema was given. Sodium thiosulphate, 1 gram was given intravenously. At 3 p. m. an intravenous injection of 900 cubic centimeters of Fischer's solution (sodium carbonate crystallized C. P. 10 g; sodium chloride, C. P. 14 g; water distilled qsa d 1,000 cc) was administered in 1 hour and 15 minutes. A proctoclysis of 500 cubic centimeters of normal salt solution was also given. At 10 p. m. he voided 15 cubic centimeters of blood.

June 2. The patient had a restless night. At 1:25 a. m. he voided 25 cubic centimeters of blood. At morning sick call his temperature was 98° F., pulse 58, respirations 18. His blood pressure was 138/62. At 11 a. m., a normal saline proctoclysis of 500 cubic centimeters was administered. At 4 p. m. he was given 1,000 cubic centimeters of Fischer's solution intravenously. Milk of magnesia and soda bicarbonate were given by mouth. Catheterization was effected by the

use of a soft rubber catheter but only a few cubic centimeters of blood were obtained. The bladder was irrigated with normal salt solution and argyrol 5-percent solution was instilled. Laboratory findings this date were as follows: Blood chemistry: Nonprotein nitrogen, 112.5 milligrams per 100 cubic centimeters of whole blood, and sugar, 100 milligrams per 100 cubic centimeters of whole blood. R. B. C., 3,750,000; W. B. C., 13,600; Hgb, 70 percent; band forms, 16; segmented, 41; lymphocytes, 29; monocytes, 14. A Kahn test was reported 1 plus.

June 3. The patient had a restless night until 12:45 a. m. at which time he was given a hypodermic injection of one-eighth grain of morphine sulphate. Following this medication he rested and slept some. In the morning he passed 90 cubic centimeters of bloody urine. He vomited small amounts on two occasions during the preceding 24 hours. A hot tub bath was given after which he perspired freely. At 1:20 p. m. his blood pressure was 134/60, pulse 84. 1,000 cubic centimeters of 2-percent sodium chloride and 5-percent glucose was given intravenously in 1 hour and 10 minutes. Following this intravenous injection his blood pressure was 134/50, pulse 94. He had hiccoughs at intervals and after 6:45 p. m. he vomited more frequently in amounts from 150 cubic centimeters to 500 cubic centimeters. He seemed mentally clear, his tongue was moist and his temperature was recorded as 100.2° F., pulse 96. Laboratory findings this date were as follows: Blood chemistry: Nonprotein nitrogen, 95; urea nitrogen, 75; uric acid, 6; creatinine, 7.5; sugar, 115; chlorides, 540; cholesterol, 166; indican, 1 plus.

June 4. After midnight the patient rested fairly well, but in the morning he felt weaker. His blood pressure was 130/60. His abdomen was somewhat distended but there was no tenderness. The percussion note was dull in his right flank. There was no edema of his ankles. His tongue was slightly coated and moist. He had hiccoughs at intervals and at times nausea followed by vomiting. A hot sponge bath between blankets was given, and hot-water bottles were used to induce skin elimination; profuse perspiration followed. He was given an intravenous injection of 1,000 cubic centimeters of normal salt solution with 5-percent glucose in 1 hour and 25 minutes. He voided 60 cubic centimeters of bright red blood. Milk of magnesia and sodium bicarbonate by mouth were continued. Caffeine sodium benzoate, 3 grains was given hypodermically 3 times a day.

June 5. The patient did not vomit during the night. In the morning he felt weak but his general condition seemed to be about the same. His temperature was 99° F., pulse 100, and respirations 20. Blood pressure was 130/54. His abdomen was somewhat distended with dullness in both flanks. He had voided 175 cubic centimeters of bloody urine during the preceding 24 hours. At 3 p. m., he had not vomited duing this date, had been free of hiccoughs since early morning and was resting comfortably. A soap-suds enema with 1½ ounces of magnesium sulphate added was given and followed by a return of yellowish colored fluid. He voided 90 cubic centimeters of urine which was highly colored but not as blood red as on previous occasions. He was given, intravenously, 1,000 cubic centimeters of normal salt solution with 5-percent glucose. Hot packs were applied for skin elimination. Laboratory findings this date were as follows: Urinalysis: Amount, 175 cubic centimeters; appearance, iron-red in color; reaction, acid; specific gravity, 1.016; albumin, 3 plus; sugar, negative; occult blood, 4 plus (acid to neutral red); leucocytes, 0 to 1 per high dry field; erythrocytes, too numerous to count; epithelium occasional squamous.

June 6. In the morning the patient felt somewhat better although he experienced some difficulty in breathing. There was some dullness over both lung cavities posteriorly. The dullness in his flanks was increased and there was some

edema of his ankles. Although the patient had not appeared apathetic, he stated that he did not remember events after coming into the hospital but now felt that his mind was clear. His blood pressure was recorded as 130/70. He was given an intravenous injection of 1,000 cubic centimeters of normal salt solution with 5-percent glucose. Hot packs were discontinued. Other medications were continued. Laboratory findings this date were as follows: Blood chemistry: Nonprotein nitrogen, 125 plus; urea nitrogen, 112; uric acid, 10; creatinine, 11.5; sugar, 150; chlorides, 510. Microscopic urine showed leucocytes 6 to 8 per high dry field, erythrocytes too numerous to count. Urea nitrogen 300 mg per 100 cc of urin. Indican, 3 plus.

June 7. The patient was mentally clear. He did not experience any hiccoughs or vomiting. Marked dullness in his flanks, edema of his penis, scrotum, and legs was noted. Temperature 99.6, pulse 114; respiration 23. His blood pressure was 130/72. Water intake was restricted to an amount between 1,000 and 1,500 cc. Laboratory report of urinalysis was as follows: Color, blood tinged; reaction alkaline; albumin 2 plus; specific gravity 1.006; occult blood 4 plus; leucocytes 4 to 5 per high dry field; erythrocytes approximately 300 per high dry field. Urea nitrogen 268 mg per 100 cc of urine. Creatinine clearance 27.

June 8. The patient felt definitely better although the edema was more marked. His urinary output was gradually increasing in amount. Blood pressure was 130/70. Laboratory report of urinalysis was as follows: Color, cloudy stra; alkaline; specific gravity 1.018; albumin 1 plus; occult blood, 1 plus; leucocytes 4 to 6 per high dry field; erythrocytes 25 to 35 per high dry field. Estimated urea output for 24 hours 2¼ g.

June 9. Laboratory findings this date were as follows: Blood chemistry: Nonprotein nitrogen 112.5; urea nitrogen 62.5; uric acid 7.5; creatinine 9.0; sugar 107; chlorides 530. Urinalysis: Color cloudy; alkaline; specific gravity 1.009; albumin 1 plus; occult blood 1 plus; leucocytes 0 to 2 per high dry fields; erythrocytes 0 to 2 per high dry field.

June 10. Urinalysis was reported as follows: Color, straw, clear; alkaline; specific gravity 1.008; albumin plus (negative in 1 to 10); occult blood negative; leucocytes 1 to 3 per high dry field; erythrocytes 1 to 2 per high dry field.

June 12. The patient was gaining each day. His temperature, pulse, and respirations were normal and he felt much better. The edema of his penis and scrotum had subsided and there was less dullness in his flanks. Some edema of the lower extremities persisted but this condition was gradully decreasing in amount. His daily output of urine was 2 to 4 times greater than his intake of fluids. Laboratory findings this date were as follows: Blood chemistry: Nonprotein nitrogen 73; urea nitrogen 56; uric acid 4.6; creatinine 2.8; sugar 107; chlorides 540. Urinalysis: Color, straw, clear; alkaline; specific gravity 1.006; albumin 1 plus; occult blood negative; leucocytes 2 to 4 per high dry field; erythrocytes none. Urea nitrogen 425 mg per 100 cc of urine. Urea estimation about 0.9 of 1 percent. Urine output 4,025 cc with estimated loss of 17 gms of urea.

June 15. The patient's general condition was improving more rapidly, his temperature, pulse and respirations continued normal and only slight edema remained. His blood pressure continued about the same from day to day. Caffeine sodium benzoate treatment was discontinued.

June 16. Result of blood chemistry examination was reported as follows: Nonprotein nitrogen 38; urea nitrogen 16; uric acid 2.9; creatinine 1.7; sugar 88; chlorides 540. Urine 400 cc. Urea output 75.5 g in 24 hours due to polyuria. Urea 1.89 percent.

June 17. Continued improvement and patient had no complaints. Dullness in flanks and edema of lower extremeties entirely subsided. His blood pressure

was recorded as 142/70. His output still exceeded his intake. He was allowed up in a wheel chair.

June 19. This date the patient had no complaints and he was allowed up and about the ward. He was placed on a soft diet and given milk of magnesia three times a day.

June 21. The patient's temperature, pulse, and respirations continued normal.

June 23. The patient had no complaints. His weight was 151½ pounds. The result of a phenolsulphonphthalein test was as follows: First specimen 130 cc, 19 percent of dye excreted; second specimen 150 cc, 15 percent of dye excreted; total 34 percent of dye excreted. Urinalysis was reported as follows: Color, straw, clear; alkaline; specific gravity 1.007; albumin negative; occult blood negative; leucocytes 1 to 2 per high dry field; erythrocytes none.

June 27. Report of urinalysis was as follows: Appearance clear straw; reaction acid; specific gravity 1.006; albumin and sugar negative; leucocytes 0 to 1 per high dry field; erythrocytes none; epithelium few squamous; crystals few amorphous urates. Urea clearance test 46.3 percent of normal; creatinine clearance 67 percent.

On June 30, 1933, 31 days after the onset of first symptoms patient looked and felt well. Fluid intake exceeded urine output. It was considered that he had made a satisfactory functional recovery from the effects of his arsenical kidney damage. He was discharged to duty without complaints and in good condition.

The following is the record of intake and output during the period of hospitalization and shows the accumulation of fluids in the body with subsequent depletion when kidney function returned and the fluid intake was restricted.

Date	Intake (cc)	Output (cc)	Plus or minus output (cc)	Appearance	Remarks
June 1	2,400	55	2,345	Blood	Hiccoughs and vomiting.
June 2	2,550	60	2,490	do	Do.
June 3	2,685	90	2,595	do	Do.
June 4	3,695	57	3,638	do	Do.
June 5	4,400	175	4,225	Bloody urine	Do.
June 6	3,225	200	3,025	do	No hiccoughs and vomiting
June 7	3,230	600	2,630	Blood tinged	None.
June 8	1,730	1,135	595	Cloudy straw	None.
June 9	1,990	1,849	141	Cloudy	None.
June 10	1,040	3,205	2,306	Light amber	None.
June 11	1,550	2,685	1,135	Pale straw	None.
June 12	1,175	4,025	2,850	Clear	None.
June 13	1,630	3,480	1,850	do	None.
June 14	1,505	3,225	1,720	do	None.
June 15	1,090	2,375	1,285	do	None.
June 16	1,000	4,325	3,325	do	None.
June 17	1,375	3,380	2,005	do	None.
June 18	1,000	3,180	2,180	do	None.
June 19	1,260	2,650	1,390	do	None.
June 20	1,200	3,650	2,450	do	None.
June 21	1,245	3,100	1,850	do	None.
June 22	990	3,375	2,385	do	None.
June 23	1,820	3,495	1,675	do	None.
June 24	1,620	3,525	1,905	do	None.
June 25	2,942	4,135	1,193	do	None.
June 26	3,240	3,485	245	do	None.
June 27	2,538	3,825	1,287	do	None.
June 28	2,575	3,625	1,050	do	None.
June 29	3,965	3,575	390	do	None.
June 30	3,000	2,553	447	do	None.

Summary of laboratory findings—Blood chemistry

Date	N. P. N.	Urea N.	Uric Ac.	Creat.	Sugar	Chloride
June 2, 1933	112.5				107	
June 3, 1933	95	75	6.0	7.5	115	540
June 6, 1933	125+	112	10.0	11.5	150	510
June 9, 1933	112.5	62.5	7.5	9.0	107	530
June 12, 1933	75	56	4.6	2.8	107	540
June 16, 1933	38	16	2.9	1.7	88	540

Urinalyses

June 5, 1933. First specimen obtained this date.

Date	Color	Sp. Gr.	Albumin	Occult Bld.	Leuco	Erythrocytes
June 5, 1933	Iron red	1.016	3 plus	4 plus	0-1	Too numerous to count.
June 6, 1933					6-8	Do.
June 7, 1933	Bld. tinged	1.006	2 plus	4 plus	4-5	Approx. 300 per H. D. F.
June 8, 1933	Cld. straw	1.018	1 plus	1 plus	4-6	25-35 per H. D. F
June 9, 1933	Cld. lt. amber	1.009	do	do	0-2	0-2.
June 10, 1933	Straw clear	1.008	Plus (neg. 1-10 dil.)		1-3	1-2.
June 12, 1933	do	1.006	1 plus		2-4	
June 23, 1933	do				1-3	
June 27, 1933	do	1.006			1-3	
June 30, 1933	do				0-1	

NOTES ON TREATMENT OF FRACTURE OF MANDIBLE IN A CHILD

By W. F. Murdy, Lieutenant Commander, Dental Corps, United States Navy

The patient, D. L., aged 5, son of a petty officer, stationed at Navy Yard, Cavite, P. I., while playing on banister fell to concrete walk, a distance of about 10 feet. He struck his chin, causing a compound fracture of the mandible and severe laceration of skin. The wound on face was sutured at the yard dispensary, but as the boy complained of soreness in his mouth and difficulty in chewing, he was sent to the dental office for treatment of a suspected fracture of the jaw. The case presented most of the cardinal symptoms of fractured mandible—some mobility of fractured ends, malocclusion of teeth, swelling, wound of gums, and tenderness and swelling about temporo-mandibular joints. Radiographic examination showed a single fracture near symphysis with slight displacement of fractured ends of bone.

On account of the child's age, it would have been difficult to reduce a fracture of this kind by the method of interdental ligation, Baker intermaxillary anchorage, splints, etc. It is difficult enough to persuade even older children to have necessary fillings or treatments, yet alone to submit them to the tedious work of taking an impression for a dental splint or passing wires around their teeth for necessary ligation, without the use of a general anaesthesia or a conduction anaesthesia. The danger of choking from vomiting and paroxysm of coughing with a fixed appliance in place was also given consideration. The application of a Barton bandage, reinforced by elastic webbing bandage from chin to crown of head for slight traction, was

decided upon as better adapted as a method to fit the needs of this particular case. The bandage had to be removed several times to treat the wound on the skin. The case progressed nicely under this treatment, and, at the end of the twenty-fourth day the bandage was removed. The next day the boy came to the office chewing gum. There was good union, good occlusion of teeth, and no displacement of broken ends of bone.

While notes on the treatment of this single fracture are hardly worth repeating, the case demonstrates again that fractures of bone in the healthy young heal quickly, if properly immobilized, and that a single compound fracture of the jaw with practically no displacement of the parts can well be treated by the simplest form of fixation.

NAVAL RESERVE

MEDICAL CORPS

APPOINTMENTS, THIRD QUARTER, 1934

Name	Rank	Appointed
Vail, Harris H	Lieutenant Commander, MC–V(S), U. S. N. R	June 21
Fothergill, LeRoy D	do	June 26
Shackford, Bartlett C	do	June 26
Farnsworth, Tom K	do	June 27
Mayfield, Claud	do	June 27
Shambaugh, Noel F	do	June 27
McElhinney, Philip P. B	do	June 28
Smith, Sydney K	do	June 29
Walker, Joe E	do	July 3
Livingston, Edward M	do	July 12
Clark, Harold E	do	July 13
Lokrantz, Sven R	do	July 14
Coffin, Whitman K	do	July 16
Cecil, Arthur B	do	July 17
Delzell, William R	do	July 20
Harner, Clyde E	do	July 20
Orme, Eugene	do	July 21
Bowman, Karl M	do	July 23
Raeder, Oscar J	do	July 25
Craig, Alfred L	do	July 31
Withington, Paul	do	July 31
Kelly, Harvey A	do	Aug. 1
MacColl, Douglas R	do	Aug. 6
Weber, William L	do	Aug. 6
Alter, Nicholas M	do	Aug. 7
McKinney, Frank S	do	Aug. 9
Hammond, Roland	do	Aug. 16
Brett, Afley L	do	Aug. 17
Colonna, Paul C	do	Aug. 22
Morrison, Wayland A	do	Aug. 22
Porter, Lewis B	do	Aug. 22
Turner, Howard K	do	Aug. 23
Chandler, Loren R	do	Aug. 28
Lounsberry, Ray Chancel	do	Aug. 28
Sanford, Conley H	do	Aug. 29
de La Chapelle, Clarence E	do	Sept. 6
de Yoanna, Saverio A	do	Sept. 6
Hyslop, George H	do	Sept. 6
Loewe, Walter R	do	Sept. 6
McNulty, Albert H	do	Sept. 7
Milam, Ernest B	do	Sept. 14
Walker, Henry M	Lieutenant, MC–V(G), U. S. N. R	June 14
Williams, Henry L	Lieutenant, MC–V(S), U. S. N. R	June 16
Toffelmier, Douglas D	do	June 28
Buckley, Thomas I	do	July 9
Jensen, Clyde R	do	July 17
Morrow, Cecil L	do	July 25
Trexler, Clarence W	do	July 31
Phillips, Richard B	do	Aug. 3
Rawlins, Aubrey G	do	Aug. 14
Lieber, Hyman	do	Aug. 16
Isquith, Samuel A	do	Aug. 17
Gerstle, Mark L., Jr	do	Aug. 18
Ecklund, Archibald M	do	Aug. 21
Wood, David A	do	Aug. 28
Boyd, Douglas	do	Sept. 4
Murphy, Wallace B	do	Sept. 4
Dawson, Terence Thomas	do	Sept. 7
Klopfenstein, Alpha R	do	Sept. 7
Nock, Randolph M	do	Sept. 11
Burke, John	Lieutenant (junior grade) MC–V(G) U. S. N. R	June 19
Baker, Samuel R	Lieutenant (junior grade) MC–V(S) U. S. N. R	June 20
Wilson, James E., Jr	Lieutenant (junior grade) MC–V(G), U. S. N. R	July 12

MEDICAL CORPS—Continued

APPOINTMENTS, THIRD QUARTER, 1934—Continued

Name	Rank	Appointed
Gerlach, Lawrence A	Lieutenant (junior grade) MC-V(G), U. S. N. R	July 25
Giannestras, Nicholas J	do	July 27
Grendon, David A	do	July 27
Baker, George I	do	Aug. 2
Ainsworth, Leonard B	do	Aug. 8
Wade, Burt O	do	Aug. 8
Inman, Jesse H	do	Aug. 12
Daniels, Donald H	do	Aug. 13
Gill, Richard S	do	Aug. 23
Pendergrass, Clayton I	do	Aug. 30
Gindhart, Floyd D	do	Sept. 12

PROMOTIONS

Name	From—	To—	Appointed
Michael, Paul	Lieut. (junior grade) MC-V(G)	Lieutenant MC-V(G)	Mar. 2
Arnold, Hermann B	Lieut. (junior grade) MC-F	Lieutenant MC-F	July 19
Boudry, Marshall O	Lieut. (junior grade) MC-V(G)	Lieutenant MC-V(G)	July 25
Strine, Howard H	do	do	Aug. 2

DENTAL CORPS

APPOINTMENTS

Name	Rank	Appointed
Wells, C. Raymond	Lt. Comdr., DC-V(S), U. S. N. R	Sept. 6, 1934

PROMOTIONS

Name	From	To
Erickson, Hilmer Alvin	Lt. (j. g.) DC-V(G), U. S. N. R	Lt. DC-V(G), U. S. N. R.
Lucas, Lester Miles	Lt. (j. g.) DC-V(G), U. S. N. R	Lt. DC-V(G), U. S. N. R.

PHINEAS J. HORWITZ.
CHIEF OF THE BUREAU OF MEDICINE AND SURGERY, 1865-1869.

NOTES AND COMMENTS

PHINEAS J. HORWITZ

THE FOURTH CHIEF OF THE BUREAU OF MEDICINE AND SURGERY

Phineas J. Horwitz was born at Baltimore, Md., on March 3, 1822. He was graduated from the University of Maryland in 1845. Dr. Horwitz was appointed an assistant surgeon in the Navy November 8, 1847, during the war with Mexico, and while serving with the Gulf Squadron he was placed in charge of a temporary naval hospital at Tabasco, Mexico. He performed his duties there so well that he received the commendation and thanks of the commander of the squadron, Commodore M. C. Perry. He was promoted to passed assistant surgeon in January 1853. He served much at sea and saw duty on the Brazilian and African stations. In 1859 he was made assistant to the Chief of the Bureau of Medicine and Surgery and, on the death of Dr. Whelan who had been Chief of the Bureau for three terms, he was appointed Chief of the Bureau on July 1, 1865, and held office until July 1, 1869. While Assistant Chief of Bureau it was stated that "the whole system of tabulating the casualties of the war, of indexing the books of reference, reports of survey, certificates of disability and of diseases, was designed and carried forward by Dr. Horwitz, so that there was probably no case of injury, disease, or disability that occurred during the doctor's connection with the Bureau that will not be found in its appropriate place in the Surgeon General's office; the immense number of pension cases accruing during the war were all examined, adjusted, and prepared by the doctor, and every official letter that left the Bureau was written by him. All this was done without the aid of a single additional writer or clerk." He had been promoted surgeon in 1861, and when he ceased to be Chief of Bureau, Congress voted the highest shore pay of his grade during his term of office. Until his retirement in 1884, he served principally in Philadelphia, as medical officer in command of the naval hospital there, at the naval asylum, and as president of the examining board. He was promoted medical inspector on March 3, 1871, and medical director December 19, 1873. He died September 28, 1904, and is buried at Philadelphia.

MEDICAL OFFICERS RECOMMENDED FOR FELLOWSHIP IN THE AMERICAN COLLEGE OF SURGEONS IN 1934

The following medical officers have been recommended by the Surgeon General of the Navy for Fellowship in the American College of Surgeons:

Lt. C. J. Stuart (M.C.), U. S. N.
Lt. Comdr. F. F. Lane (M. C.), U. S. N.
Lt. Comdr. C. H. Savage (M. C.), U. S. N.
Lt. H. L. Pugh (M. C.), U. S. N.
Lt. Comdr. C. R. Tatum (M. C.), U. S. N.
Lt. T. F. Cooper (M. C.), U. S. N.
Lt. A. T. Walker (M. C.), U. S. N.
Lt. Comdr. N. Roberts (M. C.), U. S. N.
Lt. J. M. Brewster (M. C.), U. S. N.
Lt. Comdr. C. L. Andrus (M. C.), U. S. N.

AN AMERICAN CONTRIBUTION TO NAVAL HYGIENE

The American Navy can point to no such contributions to the advancement of naval hygiene as were made by Lind, Blane, and Trotter, but there is one field of naval hygiene in which our Navy has led the way. This is in respect to the Navy ration. Gatewood made probably the first thorough study of the Navy ration, definitely estimating the food requirements as established by the best scientific knowledge of the times, and secured the actual adoption of this ration. Furthermore, he introduced the idea of substitution of one food for another if more readily available or if better liked by the men, little check being placed on the variety of food as long as the caloric requisites and balance of their ration was not measurably affected. This was a most important principle for it allowed of the great diversity of food which adds so much to the value of a diet, particularly when a large number of people are included whose food habits are of so varied a character. Variety in food is, as we know, a great factor in the maintenance of health both by increasing appetite and digestion and by lessening the probabilities of food deficiencies.

An even greater contribution perhaps was the development of the general mess system. Formerly in our Navy, as well as in foreign navies, the individual mess system prevailed. Indeed it was still in use in the British Navy to some extent up to almost the present time. By this system the crew was broken up into groups of 15 or 20 men to whom the uncooked food was served out and who elected one of their number to act as cook. The disadvantages of such a system were many, the most important being that the food was cooked by inexperienced amateurs, there was no uniformity, and no satisfactory supervision of the messes was possible. The general

mess system, with a centralized galley and group of experienced cooks and bakers, of course permits of supervision and control, assures that a proper diet is received, and is more economical. Gatewood was a pioneer in urging this system and our supply officers the first of any Navy to use it uniformly throughout the entire service.

The development of a scientifically planned and adequate ration constitutes an achievement in the field of naval hygiene in which the United States Navy may be said to have been the leader among navies, and one to which our Medical Corps can point with justifiable pride.

SCURVY AND THE VITAMIN C REQUIREMENTS OF MAN

With the discovery of vitamin C the conquest of scurvy, the classic disease of mariners, may be said to have been virtually completed. This "Plague of the Sea and Spoyle of Mariners", as one early writer calls it, will scarcely be seen again as a serious disorder of shipboard life. Indeed, its ravages are not appreciated by us now, though its power to kill men and cripple the movements of ships and fleets was well known to our forebears. Sir Richard Hawkins says that during his 20 years at sea it killed 10,000 men. In Rodney's fleet in the West Indies in 1781 out of a force of 12,000 men 1 in 7 died from scurvy in a twelvemonth. A naval surgeon of the Napoleonic Wars, R. Finlayson, says "It is the opinion of some of the most experienced officers that the blockading system of warfare that annihilated the naval power of France could never have been carried on unless sea scurvy had been subdued." The history of the effects of this disease upon naval warfare and its influence upon naval strategy has never been completely investigated and would no doubt reveal many remarkable ways in which scurvy affected important aspects of maritime history.

The "subduing" of the disease mentioned by Finlayson refers to the Admiralty order made at the urgent recommendation of Sir Gilbert Blane that all men of the Royal Navy, after being at sea 2 weeks, should receive a fluid ounce of orange or lemon juice daily. Not only did this prove a method that effectually prevented the disease but recent research indicates that it is almost exactly the amount necessary to meet the requirements of an adult today.

This accurate estimation, which waited nearly 140 years, was made possible by the discovery in the last few years of the chemical nature of vitamin C and the development of a chemical method for its estimation. The isolation of ascorbic acid, its identification as one of the hexuronic acids, and its synthetic preparation, are all recent triumphs of medicine. It has recently been shown that the ascorbic acid content of 1 cc of lemon juice or orange juice is approximately 0.6 to 0.7 milligrams. There is some difference in potency in orange

and lemon juice from various sources but good lemon juice contains about this amount. It has also been found that the amount of ascorbic acid excreted by the urine is remarkably constant, about 30 milligrams daily. This remains the same even if a vitamin C diet is given. It is estimated that the minimum daily requirement is 1 ounce (30 cc) of orange or lemon juice, or about 20 milligrams of ascorbic acid. Thus Blane's ounce of orange juice recommended in 1795 finds remarkable justification in 1934.

NEW VIEWPOINTS ON THE PROPHYLAXIS OF VENEREAL DISEASE

After about 30 years devoted to the study and use of various methods of venereal prophylaxis the naval service is confronted with the fact that the problem of the prevention of venereal disease is still unsolved, and the diminution in venereal rates has not been in accordance with expectations. There is a feeling that the situation at present is one of stalemate, a sort of trench warfare of fixed positions, and that some totally new weapon or mode of attack must be developed in order to bring about any decisive change. Some believe that it should come through efforts to eliminate "repeaters" in venereal disease, which one medical officer of the Navy has aptly termed "venereal addicts." The practical application of this plan, however, is one of great difficulty. A more simple remedy is suggested by another naval medical officer who would emphasize a method neglected and even tabooed in the past. This is the use of the condom. He would officially authorize their sale and urge their use. His comparison of this method of prophylaxis used as it is before exposure, with chemical methods used after exposure, is interesting: "This difference impresses me as being comparable to that of the difference between a fireproof building and one not fireproof but with a fire extinguisher present."

THE DURATION OF IMMUNITY AGAINST SMALLPOX

The traditional period of immunity against smallpox conferred by vaccination with cowpox virus has long been considered as from 7 to 10 years. In 1934 Rosenau and Dearing published the result of their studies on the vaccinations of 557 medical students who had all been previously vaccinated. Of 337 students who had been vaccinated within 10 years only 1 had a primary take. Of the 336 remaining, 321 gave immediate reactions, and 15 gave accelerated takes. There were 168 students vaccinated from 10 to 19 years before and of these only 6 gave primary takes. After 20 years, out of 55 students 4 gave primary takes. The following summary explains the percentage of primary takes: Vaccinated 7 to 10 years before, 0.3 percent; vac-

cinated 10 to 19 years before, 3.5 percent; vaccinated 20 or more years before, 8 percent. These figures indicate the decrease of immunity with the passage of time yet they also tend to show that some measure of immunity exists for more than 20 years in most cases. Rosenau and Dearing also recorded the vaccination results with nine medical students who had never been vaccinated but had had smallpox. Four of these had primary takes, four accelerated takes, and one an immediate reaction. It has long been known that smallpox confers excellent immunity against itself as second attacks are extremely rare. These results would indicate that smallpox does not confer lasting immunity against cowpox. In every epidemic known the converse of this has been found to be true, viz, that cowpox confers immunity against smallpox.

ADMISSION TO AMERICAN SOCIETY OF CLINICAL PATHOLOGISTS WITHOUT INITIATION FEE

The American Society of Clinical Pathologists have passed an amendment to their constitution permitting the admission of pathologists from the Navy into the Society without the payment of the initiation fee.

91829—34——9

BOOK NOTICES

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

The Editor, United States Naval Medical Bulletin,
Bureau of Medicine and Surgery, Navy Department,
Washington, D. C.

The Shoulder. Rupture of the Supraspinatus Tendon and Other Lesions in or About the Subacromial Bursa. *By E. A. Codman, M. D., consulting surgeon, Massachusetts General Hospital, Boston, Massachusetts.* Thomas Todd Company, Boston. Price, $10.00.

This is a truly remarkable medical book in more ways than one. In the first place, it is not customary for the author of a medical book to begin it with an autobiographic preface and end it with an epilogue, but after reading the preface, the book, and the epilogue the reviewer is sorry that all medical writers do not produce works which are at once entertaining and at the same time contain so much grain and so little chaff. Dr. Codman expresses the real origin of his book in a sentence in the introduction, "Already in 1900 I had become interested in what I have called the 'end result idea', which was merely the common-sense notion that every hospital should follow *every* patient it treats, long enough to determine whether or not the treatment has been successful, and then to inquire 'if not, why not?' with a view to preventing similar failures in future." He applied this end-result system to injuries of certain parts of the human shoulder and found an astonishing lack of satisfactory results as regards *the patients*. His monograph of 500 pages, magnificently illustrated and printed, followed after long experience as a surgeon taught him certain facts not readily appreciated by the average general surgeon. There are remarkable chapters on the anatomy, normal motions, and pathology of the shoulder joint, accompanied by highly original drawings. This is followed by a description of arthritis, periarthritis, and bursitis. Several chapters then follow dealing particularly with the supraspinatus tendon, not only rupture but its role in fractures and dislocations. The nonoperative treatment is discussed in detail as well as the operative treatment. There is a chapter on neurasthenia, neurosis, and malingering that alone is worth $10 although it contains only 10 pages, in fact is the shortest chapter in the book.

Dr. Codman states that "since I have practiced surgery, my attention has been riveted on so managing my life that I could get 'days off', during the spring for trout fishing and a month in the fall for partridge and woodcock. * * * Perhaps I have sacrified my success as a distinguished surgeon to these pursuits. I have loved them better than teaching dozing medical students, the pride of amphitheater dexterity, or the hushed dignity of the consultant at the bedsides of important persons. On many a bright October day I have been glad that my talents as a teacher were not in demand."

His talents as a teacher are notably displayed, however, in this book and one cannot but urge all medical men to read it using Dr. Codman's own words: "Now start in and read the best book there is on the human shoulder (it is the only one) and do not fail to note that it shows that only 10 neglected cases of the injury, prominently mentioned in its title, may cost our community more than would the distribution of 3,000 copies of this book at $10 a copy, preface, epilogue, cartoons, and all!"

SURGERY OF A GENERAL PRACTICE. By *Arthur E. Hertzler, chief surgeon, Halstead Hospital, Professor of Surgery, University of Kansas;* and *Victor E. Chesky, chief resident surgeon, Halstead Hospital.* Illustrated. The C. V. Mosby Co., St. Louis, 1934

The author finds it difficult to define minor surgery. He very properly states that what may appear minor to the surgeon seldom is a matter of minor importance to the patient. He believes that many surgical procedures are minor in that the equipment and apparatus needed is simple and the expenses of these procedures when indicated by general practitioners is much less than when the patients are hospitalized. State medicine, he says, would not be so threatening if doctors would do more in their office practice. For each condition described he gives a definite remedy which has proven effective rather than several remedies from which to select. The importance of early diagnosis and complete treatment at once is stressed so the practitioner may not make the not infrequent error of applying a partial remedy or of assuming an attitude of temporizing with a condition which may prove serious or become malignant later on. A book of this sort is not a treatise for a surgeon but it may prove a valuable guide for the physician who wishes to handle the described surgical conditions himself. It raises some of the warning signals of danger ahead, and advises hospitalization when these signals are observed. It is a valuable book though in a limited field for the student and general practitioner.

SPINAL ANESTHESIA. TECHNIC AND CLINICAL APPLICATION, by *George Rudolph Vehrs, M. D.*, Salem, Oreg. The C. V. Mosby Co. 1934. Pp. 257. Ill. 81

This is an excellent summary of today's practice of spinal anesthesia. The beginner will find here all that he needs to know in order to use spinal anesthesia successfully and to treat the usual complications. More advanced practitioners will find thorough discussions of the controversial points with the author's evaluation of them.

Nearly one-half of the book is devoted to anatomy, physiology, and pharmacology. Efforts at condensation have produced a style that, in many passages, is not easily understood. Figure 41, which "represents the improper method of injecting novocain in the subarachnoid space in spinal anesthesia" is identical with figure 73 which represents its proper use in a successful case.

But these minor points of criticism detract very little from the value of the book. It is the equal of any of the works on this subject that have come to the notice of the reviewer.

ATLAS FUNDUS OCULI, by *William Holland Wilmer, M. C.*, Professor of Opthalmology, Johns Hopkins University, Brigadier General, U. S. Army, Aux. Res. The Macmillan Co., New York. $35

Thirty-five dollars may seem like a large sum for a book but after seeing this magnificent atlas of the fundus oculi the opposite view is at once taken. The book seems cheap at that price. Each of the 100 color plates is a work of art, a real contribution to medical illustration comparable to some of the great atlases of skin diseases and on a par with the work of the great anatomic illustrations of the 18th and 19th centuries. The first 13 plates depict the normal fundus in the different races, and in the blonde, the brunette, and the albino of the white race, as well as the fundus of the dog, cat, rabbit, and guinea pig. The remaining 87 plates cover all the important local pathological conditions and their changes in the fundus characteristics of general disease such as arteriorsclerosis, renal disease, tuberculosis, malignancy, and other constitutional diseases. Such a book cannot but be invaluable to the ophthalmologist and the internist, and is almost a necessity to any large hospital staff.

COLLECTED PAPERS OF THE MAYO CLINIC AND THE MAYO FOUNDATION. Vol. XXV, 1933. W. B. Saunders Co., Philadelphia. $11.50

These annual volumes from the Mayo Clinic are distinguished by their presentation of new treatments, drugs, operations, diets, and diagnostic methods, tested and tried by the finest clinicians and in a most scientific manner. It is not surprising that general practitioners and specialists alike purchase it and read it to find what the new discoveries in medicine are and what is thought of them by the clinicians at Rochester. This volume contains 1,300 pages and 400

illustrations. This number contains a memoir of Mrs. Maud Mellish-Wilson, long the editor of the Mayo Clinic volume, whose death occurred in 1933.

TUBERCULOSIS IN THE CHILD AND THE ADULT, by *Francis M. Pottenger, M. D.*, Clinical Professor of Medicine (Department of Chest), University of Southern California, the School of Medicine. The C. V. Mosby Co., St. Louis, Mo. $8.50

This is a carefully written, well-printed, well-illustrated book on tuberculosis, covering the field as to etiology, diagnosis, course, and treatment of this important disease. The diagnostic features are described with great detail and the value of the X-ray particularly stressed. Of interest to the laboratory worker is the excellent description of the technique in making examinations for tubercle bacilli. In the treatment much space is given to compression therapy, the indications and contraindications for its use. The book lays considerable stress on sanatorium treatment. In the section on childhood tuberculosis the value of pastuerization of milk in preventing juvenile infection is emphasized.

THE SIGNIFICANCE OF NITROGEN, by *J. Enrique Zanetti*, Professor of Chemistry, Columbia University. The Chemical Foundation, Incorporated, New York

This is a short history of the nitrogen industry and its importance, particularly in the munitions industry, though its agricultural value and other uses are mentioned. The effects of the Chilean monopoly both during and after the war, and the crushing blow to that monopoly as the result of the perfecting of processes for the fixation of atmospheric nitrogen, are told in dramatic fashion. The writer pictures the struggle as still going on but it is evident that the discovery of a synthetic process is as certain to end foreign control of the industry as the discovery of a method of synthesis of alizarin by Perkins ended the madder growing industry. The disappearance of the indigo plantation followed in the same way the making of synthetic indigo. The battle over nitrogen, however, is still not over and the warnings of this book should be heeded. One feels after reading it that the lesson learned during the World War, when we paid an exorbitant price for nitrogen, should not be forgotten, and our own nitrogen industry should be encouraged in every possible manner.

A TEXTBOOK OF BACTERIOLOGY, by *Hans Zinsser, M. D.*, Professor of Bacteriology and Immunology, Harvard University, and *Stanhope Bayne-Jones, M. D.*, Professor of Bacteriology, Yale University Medical School. D. Appleton-Century Co., New York and London. $8

This is a comprehensive manual dealing with the pathogenic bacteria, and the subject of immunology, specific therapy, and epidemology as well. Furthermore, the pathogenic protozoa are

included as well as such higher plants as fungi. The latest knowledge on such important matters as bacterial variability, ultra microscopic agents, chemistry of antigens and the bacteriophage are to be found. The book is handsomely printed and illustrated and contains a total of 1,226 pages, of which 71 pages are used for a very comprehensive index, a most valuable feature in a book of this character. The title is a little misleading as only pathogenic bacteria are included.

LABORATORY MANUAL OF BIOLOGICAL CHEMISTRY, by *Otto Folin*, Hamilton Kuhn Professor of Biological Chemistry in Harvard Medical School. Fifth Edition. 368 pages. D. Appleton-Century Co., New York and London. $3

This is a revised edition of this well-known and valuable handbook covering all the important laboratory procedures of physiological chemistry. The laboratory methods for nitrogen determination, the chemistry of fats, sugars, and amino acids, are given in detail, yet in a very concise way. The chapters on the analysis of the urine, blood, and milk are equally practical. There are a number of drawings and charts and an excellent index. The text is printed on one side of the page only, thus permitting notes to be taken on the blank side.

DENTAL HISTOLOGY AND EMBRYOLOGY, by *Theodore B. Beust, M. D., D. D. S.*, Professor of Dental Histology and Comparative Anatomy, School of Dentistry, University of Louisville. W. B. Saunders Co., Philadelphia, 1934

A textbook for dental students, the result of the author's desire to present a classroom guide that adequately correlates the microscopic anatomy of the hard tissues of the teeth with questions of a biologic nature. Sclerosis of the dentine is defined as a physiological reaction to irritation and to age, which results in the formation of an impermeable barrier laid down by the defense forces of the organism.

THE LABORATORY NOTEBOOK METHOD IN TEACHING PHYSICAL DIAGNOSIS AND CLINICAL HISTORY RECORDING, by *Logan Clendening, M. D.* The C. V. Mosby Co., St. Louis. Price 50 cents

A small paper pamphlet for use in teaching case taking and physical diagnosis.

THE SPASTIC CHILD, by *Marguerite K. Fischel.* C. V. Mosby Co., St. Louis. $1.50

A small volume in which the writer describes her personal experiences with her two children who were victims of Little's disease. She gives in detail schemes of rehabilitation resulting in the patient regaining muscle control. It is really a valuable little book about a little-understood pathological condition. Not the least valuable feature is some rather artless and perfectly justified criticism of the

medical man faced with a discouraging condition such as these spastic palsies of infancy and childhood present.

NOTE.—If you have need for a particular medical book for a limited period, please send your request to the editor of the BULLETIN. He will endeavor to obtain the book from one of the medical libraries in Washington having a loan service and send it to you by mail. After you have finished with the book please return by mail to the editor.

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, 1933

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

Since November 1924 medical officers of the Navy have been required to make monthly reports of the number of doses of arsenicals administered and a separate account of every case in which ill effects are noted. During the 9 years in which this information has been compiled 839,981 doses of arsenicals have been administered and 614 reactions have been reported.

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

The reactions during the year 1933 are as follows:[2]

Classification	Cases	Deaths
Arsenical dermatitis [1]	33	2
Vasomotor phenomena	32	1
Table reactions	11	0
Acute renal damage	5	2
Liver damage:		
Acute yellow atrophy of the liver	1	1
Jaundice	4	0
Blood dyscrasias	4	0
Jarisch-Herxheimer	2	0
Hemorrhagic encephalitis	1	1
Border-line, hemorrhagic encephalitis	1	0
Liver damage (doubtful reaction)	1	0
Total	95	7

[1] Case histories were published in the October 1934 number of the Bulletin.

[2] This is a revision of the classification as published in the October 1934 number of the United States Naval Medical Bulletin.

Proportion of reactions of various types, 1929 to 1933, inclusive

Type	Number of reactions	Percent of total reactions
Vasomotor phenomena	190	50.26
Arsenical dermatitis	118	31.23
Reactions of minor importance	17	4.50
Liver damage	12	3.18
Blood dyscrasias	11	2.91
Table reactions [1]	11	2.91
Jarisch-Herxheimer	7	1.85
Acute renal damage	6	1.59
Hemorrhagic encephalitis	3	.79
Polyneuritis	1	.26
Border-line, hemorrhagic encephalitis	1	.26
Liver damage (doubtful reaction)	1	.26
Total	378	100.00

[1] First reported during the year 1933.

Arsenicals administered during the year 1933 for all diseases including syphilis

	0.9 gram to 3.0 grams	0.9 gram	0.6 gram to 0.9 gram	Less than 0.6 gram	Total
Arsphenamine:					
United States Navy	0	0	0	77	77
All others	0	0	0	12	12
Neoarsphenamine:					
United States Navy	0	1,541	48,013	63,433	112,987
All others	0	86	3,979	21,438	25,503
Tryparsamide:					
United States Navy	2,763	0	0	0	2,763
All others	1,916	0	0	0	1,916
Sulpharsphenamine:					
United States Navy	0	8	18	581	607
All others	0	3	5	2,306	2,314
Silver arsphenamine:					
United States Navy	0	0	0	4	4
All others	0	0	0	30	30
Bismarsen:					
United States Navy	0	0	0	22	22
All others	0	0	0	62	62
Acetarsone:					
United States Navy	0	0	0	0	0
All others	0	0	0	332	332
Total	4,679	1,638	52,015	88,297	146,629

The following table shows the number of deaths and severe reactions which followed the administration of 762,342 doses of neoarsphenamine during the 9 years, 1925–33, and the ratio of deaths and severe reactions to the total number of doses administered:

Classification	Deaths		Severe reactions		Deaths and severe reactions	
	Number	Ratio to doses	Number	Ratio to doses	Number	Ratio to doses
Hemorrhagic encephalitis	13	58,642	0	----------	13	58,642
Arsenical dermatitis	7	108,906	117	6,516	124	6,148
Vasomotor phenomena	6	127,057	49	15,558	55	13,861
Blood dyscrasias	3	254,114	7	108,906	10	76,234
Acute renal damage	2	381,171	4	190,586	6	127,057
Acute yellow atrophy of the liver	2	381,171	0	----------	2	381,171
Jaundice	0	----------	10	76,234	10	76,234
Polyneuritis	0	----------	1	762,342	1	762,342
Border-line, hemorrhagic encephalitis	0	----------	1	762,342	1	762,342
Total	33	23,101	189	4,034	222	3,434

Deaths charged to the administration of arsenical compounds during the past 15 years were recorded as follows:

Year	Arsphenamine	Neoarsphenamine	Kind not specified	Total	Year	Arsphenamine	Neoarsphenamine	Kind not specified	Total
1919	2	0	1	3	1928	0	6	0	6
1920	1	1	0	2	1929	0	3	0	3
1921	3	1	0	4	1930	0	3	0	3
1922	0	4	0	4	1931	0	0	0	0
1923	0	1	0	1	1932	0	4	0	4
1924	1	2	0	3	1933	0	7	0	7
1925	0	2	0	2					
1926	0	4	0	4	Total	8	42	1	51
1927	1	4	0	5					

ANNUAL CENSUS OF PERSONS TREATED FOR SYPHILIS AND FOR DISEASES OTHER THAN SYPHILIS

In order to obtain information as to the number of persons in the naval service who have syphilis a census is taken on December 31 of each year. On this date each activity records (form A) and reports to the Bureau of Medicine and Surgery the number of individuals in that command who have a history of syphilis in their health records. This census does not take into account those individuals who have left the service during the year.

SYPHILIS AND ARSENICALS, UNITED STATES NAVY, 1933

Summary of census taken on Dec. 31, 1933

	United States Navy	All others	Total (persons)
Strength, Dec. 31, 1933	105,691	.....	105,691
Syphilis census, Dec. 31, 1933	15,111	.....	15,111
1. Number of persons treated for *syphilis* with:			
(*a*) Arsenicals:			
Arsphenamine	10	0	10
Neoarsphenamine	7,781	1,109	8,890
Tryparsamide	129	161	290
Sulpharsphenamine	92	69	161
Silver arsphenamine	0	5	5
Bismarsen	5	17	22
Total	8,017	1,361	9,378
(*b*) Other treatment:			
Bismuth compounds	6,777	1,258	8,035
Mercury compounds	2,170	164	2,334
Potassium iodide	454	99	553
Mixed treatment (specific mixture, etc.)	140	19	159
Sodium iodide	1	0	1
Malaria treatment	1	0	1
Total	9,543	1,540	11,083
Total of (*a*) and (*b*)	17,560	2,901	20,461
2. Number of persons treated for *diseases other than syphilis* with:			
Neoarsphenamine	499	3,012	3,511
Sulpharsphenamine	0	261	261
Acetarsone	0	43	43
Fowler's solution	4	0	4
Bismosol	1	76	77
Total	504	3,392	3,896
Grand total	18,064	6,293	24,357

Additional information is compiled at the same time with respect to the number of these individuals who were treated during the year for syphilis and other diseases with arsenicals and heavy metals. It will be noted in the table which follows that treatment data have been separated into that given to active naval personnel and that given to all others. The term "all others" includes dependents of naval personnel, Veterans' Administration patients, retired naval personnel, and native populations of insular possessions.

Of the 504 naval personnel treated for diseases other than syphilis, 455 were treated for Vincent's infection, 18 for yaws, and 31 for other diseases.

Of the 6,293 persons in the group "all others", 2,901 were treated for syphilis, 3,365 for yaws, 22 for Vincent's infection, and 5 for other conditions.

HEMORRHAGIC ENCEPHALITIS

Ratio of deaths from hemorrhagic encephalitis to persons treated for syphilis with arsenical compounds, 1931, 1932, 1933

	Number of persons	Deaths	Ratio
1931	15,763	0	
1932	12,245	2	1:6,123
1933	9,378	1	1:9,378
Total	37,386	3	1:12,462

In comparing deaths from hemorrhagic encephalitis with the number of doses of arsenicals administered, all diseases for which these compounds were given are considered.

	Number of doses of neoarsphenamine	Deaths	Ratio
1931	95,442	0	
1932	128,540	2	1:64,270
1933	138,490	1	1:138,490
1925–33	762,342	13	1:58,642

During the 9-year period 1925–33 there were 30,652 doses of arsphenamine administered with 1 death from hemorrhagic encephalitis. In this period 31,187 doses of tryparsamide and 14,655 doses of sulpharsphenamine were given with no resulting deaths.

Neoarsphenamine.—(1–1933.) The patient was given a diagnosis of syphilis in December 1932 because of two consecutive four plus Kahn blood tests.

From November 23, 1932, until January 10, 1933, he received one 0.3 gram and five 0.6 gram intravenous injections of neoarsphena-

mine. On March 4, 1933, he received 0.3 gram of neoarsphenamine and on March 11 a 0.45 gram injection. The latter dose was dissolved in 9 cubic centimeters of double distilled sterilized water and administered in 3 minutes.

Upon completion of the treatment the patient walked out on deck but returned in 5 minutes complaining of nausea. He immediately collapsed, presenting signs of severe shock. Although pulseless he was conscious and complained of tightness in the upper abdomen. Pulmonary edema ensued and death occurred in 45 minutes.

Post-mortem examination showed marked congestion and hemorrhagic infiltration of all tissues, especially the lungs, endocardium, kidneys, pancreas, and meninges.

The reporting medical officer stated that the neoarsphenamine solution was prepared by dissolving five ampules of 0.9 gram each in 90 cubic centimeters of water. The water had been double distilled and then sterilized the night before in the autoclave. During the morning 9 patients received injections, of whom this patient was the fifth. None of the other men suffered any reaction. This patient stated that he had suffered no ill effects following any of his previous injections.

(2–1933.) In another instance a severe reaction occurred with symptoms indicative of cerebral irritation. As other symptoms were observed the case has been classified as a border-line type of reaction. The history follows: Primary lesion appeared on June 7, 1933. The patient received his first injection of neoarsphenamine, 0.3 gram, on June 19. A febrile reaction was noted. On June 24 he was given 0.6 gram of neoarsphenamine. He also received intramuscular injections of bismuth salicylate on June 19 and 24. Twenty-four hours after the second injection of neoarsphenamine a faint morbilliform rash appeared, increased in intensity for a few days, and disappeared within 6 days. Associated with the rash was conjunctival injection and a subconjunctival hemorrhage in one eye.

On June 29, the day before the rash disappeared, the patient had two epileptiform convulsions. Following these he was semiconscious for about 18 hours, and also had dilatation of the right pupil. His blood pressure was 146/85, temperature 98° F. to 100.2° F., urine strongly acid, albumin 1+, and many leukocytes. All of these signs and symptoms disappeared within 4 days.

It is of interest to note that the patient had some febrile reaction from June 19 to July 3, the temperature ranging from 98° F. to 104° F. On June 30 examination of the spinal fluid revealed: Kahn, negative; Pandy's test and ammonium sulphate strongly positive for globulin; Autenreith test, negative for arsenic; colloidal gold curve 1111111100.

Recovery from reaction in 7 days.

ACUTE RENAL DAMAGE

(83–1933.) A patient who was infected on March 25, 1928, received arsenical treatment as follows:

April 10, 1928, to May 29, 1928—8 injections neoarsphenamine.

July 11, 1928, to November 26, 1928—16 injections neoarsphenamine.

On September 26, 1933, he received 0.3 gram of neoarsphenamine and on October 3, 0.6 gram. The latter dose was dissolved in 15 cubic centimeters of water and injected slowly. About 30 minutes later the patient developed symptoms of profound shock, which were followed by elevation of temperature and anuria.

During the succeeding 3 days he voided less than 200 cubic centimeters of urine daily and all specimens contained much albumin and many granular casts. A physical examination on October 6, 3 days after the injection, revealed the following findings: Facial edema and labial herpes; blood pressure 140/90.

October 7.—Urine, specific gravity 1.010; albumin 4+; leukocytes 8 to 10 per high dry field; temperature 98° F. to 99° F.

October 10.—Daily output of urine increasing. There is much albumin but no casts in his urine.

October 12.—Urine output in 24 hours, 2,925 cubic centimeters.

October 15.—Patient feels well. Edema has disappeared. Urine is negative.

Treatment.—Ten minims of adrenalin subcutaneously and 1 gram of sodium thiosulphate intravenously at onset of symptoms; subsequently forced fluids, milk, and eggnogs.

October 20.—Recovery after 17 days of illness.

(84–1933.) A patient whose initial lesion appeared on December 15, 1927, began treatment on December 17, and received 8 injections of neoarsphenamine and 10 injections of mercury between that date and March 24, 1928. Treatment was resumed on April 28, and 8 injections of neoarsphenamine and 10 injections of mercury were given between that date and July 12. In his third course, August 24, 1928, to November 27, 1928, he received 8 injections of neoarsphenamine and 2 injections of mercury.

On May 1, 1933, his fourth course was started, and 5 injections of bismuth were given up to May 23, when he received 0.3 gram of neoarsphenamine. His second injection of neoarsphenamine, on June 6, consisted of 0.45 gram in 10 cubic centimeters of freshly distilled water and injected in 1 minute. About 30 minutes later he developed symptoms of shock and also had severe pains in his back and legs. After recovery from shock he was transferred to a hospital ship.

The following day, June 7, the patient felt better although he had not voided in 24 hours. He was catheterized and 5 cubic centimeters

of urine obtained. This urine was loaded with albumin and contained many hyaline and waxy casts.

Blood.—Red blood count, 4,660,000; hemoglobin 90 percent; white blood count, 31,000; myelocytes 2, juveniles 7, band forms 14, segmented 62, lymphocytes 13, and eosinophiles 2.

Blood chemistry.—Nonprotein nitrogen, 75 milligrams per 100 cubic centimeters; urea nitrogen, 45 milligrams per 100 cubic centimeters.

June 8.—The patient voided 300 cubic centimeters of urine in the previous 24 hours. The urine contained much albumin and many fine and coarse hyaline and waxy casts. Electrocardiogram: Rate 105 and regular; P waves normal; PR interval 0.16 of a second; QRS occupies 0.06 of a second and of normal contour; T waves, upright in leads 1 and 2, inverted with slight convexity upward of S–T interval in lead 3. Sinus tachycardia.

June 9.—Physical examination: Blood pressure 140/90; liver slightly enlarged; neurological negative. Urine: Specific gravity 1.007, albumin 2+, few granular, hyaline, and waxy casts. Blood: Kahn, negative; white blood count, 23,900; juveniles 3, band forms 7, segmented 70, lymphocytes 19, and eosinophiles 1. Blood chemistry: Urea nitrogen, 56 milligrams per 100 cubic centimeters; creatinine, 4.5 milligrams per 100 cubic centimeters.

July 8.—Recovery in 32 days.

(3–1933.) A patient, who was first infected June 11, 1921, received arsenical treatment as follows:

> July 29 to September 14, 1921—8 injections of neoarsphenamine (5.7 grams).
> January 9 to March 20, 1922—8 injections of neoarsphenamine (6.0 grams).

On March 20, 1925, there was history of exposure which was followed by an indurated chancre on the prepuce and marked enlargement of the inguinal glands. Treatment was as follows:

> March 19 to June 17, 1925—6 injections of neoarsphenamine and 6 injections of mercury.
> October 28 to December 9, 1926—12 injections of mercury.
> July 11, 1933—1 injection of neoarsphenamine (0.3 gram).

On the morning of July 18, 1933, he received 0.6 gram of neoarsphenamine dissolved in 10 cubic centimeters of water and injected in 3 minutes. One hour later he was suddenly seized with violent pains in the lumbar region. This was followed by vomiting and collapse. His respirations were shallow, pulse rate 90, and very feeble, blood pressure 70/50. Throughout the afternoon he was practically pulseless. At 6:50 p. m. he was transfused with 500 cubic centimeters of citrated blood. There was almost immediate improvement in his general condition. He was catheterized and 50 cubic centimeters of fairly clear urine obtained. This contained albumin, a small amount of mac-

roscopic blood and a number of red blood cells. About 45 minutes after the transfusion was fairly good through the night with a strong pulse of about 90 and blood pressure 104/68. The next morning, July 19, he was catheterized and 60 cubic centimeters of chocolate-colored urine obtained.

He continued to have hematuria and backache until his death on July 27, 9 days after the injection.

Autopsy.—Pericardial adhesions over the surface of the right auricle and ventricle; marked dilatation of the right side of the heart with dilatation of the tricuspid ring. Petechial hemorrhages in the myocardium. Slight edema of the lungs and marked acute passive congestion of the liver and kidneys.

(82–1933.) This patient had a history of two infections, the first after exposure on November 6, 1927, and the second after exposure on November 5, 1930. Treatment was as follows:

First series.—

December 12, 1927, to January 9, 1928—9 injections of neoarsphenamine (4.05 grams).
December 13, 1927, to April 24, 1928—18 injections of bismuth.
May 28 to June 12, 1928—8 injections of neoarsphenamine (3.6 grams).
May 30 to September 12, 1928—12 injections of bismuth (1.2 grams).
December 20, 1928, to March 10, 1929—6 injections of neoarsphenamine 2.7 grams).

Second series.—

November 28, 1930, to June 26, 1931—10 injections of neoarsphenamine.
November 28, 1930, to June 26, 1931—13 injections of bismuth.
January 5 to February 23, 1932—8 injections of neoarsphenamine.
January 5 to February 23, 1932—10 injections of bismuth.
June 10 to July 1, 1933—4 injections of bismuth.
November 25, 1933—1 injection of neoarsphenamine (0.3 gram).
November 29, 1933—1 injection of neoarsphenamine (0.45 gram).
December 2, 1933—1 injection of neoarsphenamine (0.6 gram).
December 9, 1933—1 injection of neoarsphenamine (0.6 gram).

The injection of December 9 was dissolved in 20 cubic centimeters of water and given in 1 minute. One hour after the injection the patient had severe pains in his back and legs. Later in the day he vomited several times and in the afternoon blood was seen in the urine.

December 10.—Patient complains of no pain. Slight jaundice.

December 11.—Patient nauseated, vomiting, and perspiring. Blood pressure 130/60. Voided 35 cubic centimeters of urine.

December 12.—Vomiting and perspiring. Slight nose bleed. Voided no urine.

December 13.—Vomiting and perspiring. Blood pressure 124/64.

December 14.—Vomiting and perspiring. Blood pressure 130/60. Temperature, pulse, and respirations, normal. White blood count 8,000, polymorphonuclears 61, lymphocytes 26, mononuclears 3, and eosinophiles 8. Blood chemistry: Urea nitrogen, 100 milligrams per 100 cubic centimeters. Creatinine, 7.5 milligrams per 100 cubic centimeters. Icterus index 20. Voided 20 cubic centimeters of urine.

December 15.—Blood pressure 120/62. Cutaneous hyperesthesia. Subconjunctival hemorrhages.

December 16.—Voided 25 cubic centimeters. Kahn blood test 4+.

December 17.—Severe pain over kidneys, slight nose bleed.

December 18.—Severe pain over kidneys, nausea, perspiring freely. Voided 40 cubic centimeters. Urine: clear amber, specific gravity 1.014, heavy trace of albumin, many white blood cells, and an occasional red blood cell.

December 19.—Voided 40 cubic centimeters. Severe backache.

December 20.—Severe pain over kidneys. Hiccoughs and nose bleed. Voided 130 cubic centimeters of bloody urine. Blood pressure 150/80.

December 21.—Patient raving from severe pain over kidneys. Blood pressure 130/80. Blood chemistry: Urea nitrogen, 150 milligrams per 100 cubic centimeters; creatinine 11.2 milligrams per 100 cubic centimeters; cholesterol, 210 milligrams per 100 cubic centimeters; non protein nitrogen, 450 milligrams per 100 cubic centimeters. Died at 1:30 p. m.

Autopsy.—Lungs, dark red; heart, normal in size and valves are normal. The aorta contains several atheromatous patches. Liver, smaller than normal and has a nutmeg mottling; spleen, intestines, and pancreas, normal; kidneys, small hemorrhagic area in the lower pole of left kidney. Both kidneys are large and pale, and the cortex of both is very opaque and soft.

(85–1933.) This case is reported by Lt. Comdr. C. L. Andrus, Medical Corps, United States Navy, on page 109.

LIVER DAMAGE

(88–1933.) A patient, who was exposed on August 28, 1933, developed a penile lesion on October 2. The following day he received 0.6 gram of neoarsphenamine and on October 10 another injection of 0.6 gram. Four days later he complained of nausea and loss of appetite. This was not considered at the time as resulting from treatment so he was given a third injection of neoarsphenamine on October 18, this dose consisting of 0.6 gram in 10 cubic centimeters of sterile distilled water and given in 30 seconds. About 1 hour later the patient had a chill followed by nausea, vomiting, and an elevation of temperature to 102° F. During the afternoon he had general erythema and profuse sweating.

The next morning, October 19, his temperature was 100.2° F. He appeared drowsy, had erythema of the entire body with injection of the pharynx and conjunctivae. Blood pressure 90/50. No other abnormalities were noted. The blood Kahn was 2+, his urine contained many calcium carbonate crystals and was strongly positive for arsenic.

The following day there was marked improvement and his temperature remained normal throughout the day. He was kept in bed under observation but showed nothing of note until October 24, 5 days after the injection, when jaundice appeared. At this time his urine was highly colored with bile.

Although he was not seriously ill the jaundice persisted for more than a month. During his stay in the hospital he received 10 intravenous injections of sodium thiosulphate of 1 gram each and intramuscular injections of bismuth.

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December 9, 1933.—Recovery in 52 days.

(89–1933.) A patient, who was infected in July 1933 received nine injections of neoarsphenamine for a total of 3.6 grams during August and September. A second course of treatment, consisting of five injections of arsphenamine for a total of 1.2 grams, was given in November. On December 12 a Kahn blood test was 3+. On December 13 he began a third course of arsenical treatment, receiving a 0.3-gram injection. This dose was dissolved in 6 cubic centimeters of water and given in 3 minutes. About 2 days later the patient complained of general malaise, which was followed in the next few days by vomiting and then pain and tenderness in the epigastrium.

On December 18, 5 days after the injection, he was admitted to a naval hospital with jaundice, pain, and tenderness over the liver. At this time he was mildly jaundiced, his liver was enlarged and tender, and there was a fading scarlatinal rash. Within 2 days the tenderness over the liver disappeared. His icterus index on December 21 was 100; on December 30, 100; and on January 19, 10.

Recovery in 20 days.

(90–1933.) A patient, who was exposed on December 29, 1932, developed an initial lesion late in January 1933 and began treatment on February 8. He received 0.3 gram of neoarsphenamine on February 8; 0.3 gram on February 11; and on February 18, 0.45 gram dissolved in 13.5 cubic centimeters of water and given in 3 minutes. About 4 hours later he had a severe chill followed by fever. On February 21 he developed jaundice, vomiting, and backache. Laboratory reports were as follows: Blood, white blood count, 6,900; polymorphonuclears, 41; lymphocytes, 46; transitionals, 5; mononuclears, 2; eosinophiles, 4; and basophiles, 2. Urine, specific gravity, 1.012; reaction, acid; albumin, positive; 5 to 6 white blood cells per high power field, occasional red blood cells, and many casts.

February 24.—Physical examination negative except for jaundice, general adenopathy, and tenderness on palpation over kidneys. Laboratory, urine positive for bile, negative for albumin, casts, and arsenic. Blood count, normal with 85 percent hemoglobin. Van den Bergh's, direct immediate reaction. Icterus index, 30, bilirubin 3.3 milligrams per 100 cubic centimeters of blood.

Recovery in 46 days.

(91–1933.) A patient, who was exposed about March 12, 1933, developed a chancre early in April and began treatment on April 17. He received 0.3 gram of neoarsphenamine on April 17; 0.45 gram on April 20; and on April 28, 0.6 gram dissolved in 12 cubic centimeters of freshly double-distilled water and given in 3 minutes. About 3 hours later he developed a headache and fever. A fine red rash appeared and covered the entire body surface. There was also some injection of the sclerae and edema of the eyelids. Urinalysis was negative.

April 29.—The following day, his temperature range was 100.8° F. to 103.4° F.

April 30.—Temperature 101.6° F. to 102.4° F. Urine shows a trace of albumin and many epithelial cells.

May 1.—Patient feels better. Van den Bergh's, direct immediate reaction.

May 2.—Temperature 99.4° F.

May 6.—Slight icterus.

May 11.—Blood icterus index, 38.

May 15.—Marked icterus of entire body; icterus index, 107.

May 17.—Slight fading of icterus.

May 23.—Icterus index, 68.

May 29.—Icterus index, 20.

June 6.—Icterus index, 7.

June 16.—Icterus index, 7.

Blood

	Red blood count	Hemoglobin	White blood count	Juveniles	Band forms	Segmented	Eosinophiles	Basophiles	Lymphocytes	Mononuclears
Apr. 29	4,970,000	85	10,600	1	58	30	----	1	5	5
Apr. 30	4,800,000	0	8,700	----	54	17	1	----	18	10
May 2	4,870,000	80	5,300	----	48	22	----	1	23	6
May 3	4,890,000	85	6,350	----	30	17	4	----	36	[1] 12
May 4	4,850,000	80	5,200	1	37	39	5	----	13	5
May 8	4,000,000	70	4,600	----	20	37	12	5	15	11

[1] Turck's cell, 1.

June 23.—Recovery in 56 days.

(86–1933.) A patient whose diagnosis of syphilis was established on February 6, 1933, began treatment on February 14, when he received 0.3 gram of neoarsphenamine. This was followed by 0.6 gram injections on February 21, 28, and March 7. The last dose was dissolved in 12 cubic centimeters of water and injected in 1 minute. An hour later he became nauseated, vomited, and complained of severe pain in his abdomen and right loin. His temperature rose during the day to a maximum of 102.5° F. at 8 p. m. During the night he voided a small quantity of bloody urine.

March 8.—Twenty-four hours after the injection the patient is deeply jaundiced. Blood pressure 90/70. Taking fluids freely and has no pain. Fluid intake 1,500 cubic centimeters, output 55 cubic centimeters. Laboratory reports, van den Bergh, direct immediate reaction. Urine contains blood. Blood, icterus index, 75; red blood count, 2,580,000; white blood count, 8,250; hemoglobin, 54 percent; differential, band forms, 4; segmented, 26; lymphocytes, 66; mononuclears, 2; and eosinophiles, 2. Red cells show slight achromia and polychromatophilia. There are many normoblasts and a few stippled red cells.

March 9.—(Second day.) Conscious and rational in the morning, becoming restless, with hiccoughs, in the afternoon. Voided 35 cubic centimeters of blood. Temperature 99.5° F., pulse 85, and respirations 18. Hypodermoclysis of 2,000 cubic centimeters of normal saline solution and later 1,000 cubic centimeters of a 1-percent glucose solution. One gram of sodium thiosulphate intravenously. Blood count: White blood count, 5,600; band forms, 3 percent; segmented, 50 percent; eosinophiles, 3 percent; mononuclears, 3 percent; lymphocytes, 41 percent. Blood chemistry (milligrams per 100 cubic centimeters), urea nitrogen, 56; sugar, 114; creatinine, 4.5; nonprotein nitrogen, 95; and chlorides, 460.

March 10.—(Third day.) Expectorating dark red blood. Temperature 98 to 100° F., pulse 85, respirations 30. Fluid intake 2,600 cubic centimeters and output 110 cubic centimeters, which was the total amount obtained by voiding and bladder irrigation. Urine, bloody, albumin 4+, no casts; blood, icterus index, 150; van den Bergh's, immediate direct reaction; treatment, rectal irrigation with hot sodium bicarbonate solution, 1,000 cubic centimeters of glucose in saline solution by hypodermoclysis. Sodium thiosulphate, 1 gram intravenously. Forced fluids by mouth.

March 11.—(Fourth day.) Cyanosis of lips and fingers, epistaxis, blood in stools and urine. Fluid intake 3,000 cubic centimeters, output 50 cubic centimeters. Blood chemistry (milligrams per 100 cubic centimeters), urea nitrogen, 140; sugar, 150; creatinine, 7.4; nonprotein nitrogen, 175; chlorides, 400. Blood, red blood count, 1,590,000; hemoglobin, 30; white blood count, 2,250; differential, band forms, 2; segmented, 21; mononuclears, 1; lymphocytes, 76; red cells show anisocytosis, few microcytes and macrocytes, occasional normoblast. Through the day fluids were given by mouth, by hypodermoclysis, and by rectum.

March 12.—(Fifth day.) Death at 2:50 p. m. Autopsy diagnosis: (1) Acute yellow atrophy of the liver; (2) acute parenchymatous nephritis; (3) marked edema of both lungs, thoracic cavity filled with fluid; (4) parenchymatous degeneration of the heart, spleen, and adrenals.

Discussion.—While neoarsphenamine was probably the immediate cause of death, other factors may be regarded as contributory. In addition to syphilis he had cystitis, a left inguinal hernia, varicose veins of one leg, and arthritis of the right ankle and foot. It was also learned that he had been drinking heavily while under specific treatment.

(87–1933.) This is a doubtful liver damage case. The patient, who was infected in October 1933, began arsenical treatment on November 13, when he received 0.3 gram of neoarsphenamine. On November 18 he was given a second injection of 0.3 gram. On November 25 he

received 0.6 gram of neoarsphenamine dissolved in 10 cubic centimeters of water and given in 2 minutes. About 6 hours after this injection he developed nausea, vomiting, severe headache, a rash over the entire trunk and a temperature of 103.8° F. A urinalysis was negative. The next day he was transferred to a naval hospital for treatment. Symptoms subsequent to admission to the hospital were jaundice and headache. The jaundice persisted for about 2 months. After its disappearance he was given four injections of neoarsphenamine without reaction and without return of jaundice. For this reason the medical officer thought the jaundice was probably coincidental to and not caused by the neoarsphenamine. However, the appearance of symptoms appear to have been directly associated with the treatment and at least aggravated by the arsenic.

VASOMOTOR PHENOMENA

(5–1933.) A patient, who was infected in November 1931 received treatment as follows:

March 17 to April 23, 1932—10 injections of neoarsphenamine (5.55 grams).
March 26 to May 10, 1932—15 injections of bismoid.
June 11 to August 13, 1932—8 injections of neoarsphenamine (4.35 grams).
June 7 to October 4, 1932—16 injections of bismoid.

He began his third course of arsenical treatment on December 17 when he received 0.3 gram. He was given 0.45 gram on December 21 and 0.6 gram on January 4. The 0.6 gram dose was dissolved in 12 cubic centimeters of double distilled sterile water and administered in 1 minute. He immediately became nauseated, vomited, and his skin was intensely red. An injection of 10 minims of adrenalin and 1 dram of aromatic spirits of ammonia by mouth relieved his symptoms. An hour later he complained of headache, pains in his neck and shoulders, and blurring of vision. During the succeeding 5 days he was given sodium thiosulphate in 1 gram doses by mouth and by intravenous injection.

Recovery in 5 days.

(7, 8–1933.) This patient, who was infected early in October 1933 received a 0.3 gram injection of neoarsphenamine on October 11, one of 0.45 gram on October 17, and another of 0.45 gram in the morning of October 24. The last dose was dissolved in 10 cubic centimeters of water and administered in 2½ minutes. About 20 minutes later he felt chilly, with pains in the lower lumbar region and in his eyes, and slight weakness. About 10 hours later he reported to the medical officer because of increasing severity of symptoms. At this time his blood pressure was 115/50, pulse 116, respirations 22. The only objective symptom was moderate edema of the eyelids. He was kept in bed for 3 days during which time his urine showed no abnor-

malities and all symptoms disappeared. As treatment he received 4 minims of adrenalin (1–1000) subcutaneously, and 1 gram of sodium thiosulphate intravenously. Recovery on October 24 after 3 days of illness. Intramuscular injections of bismuth were given on November 2, 9, 16, and 23.

On November 28, 32 days after recovery from the first reaction, arsenical treatment was resumed with the administration of 0.3 gram of neoarsphenamine. About 6 hours later the patient had severe pains in his left chest and some generalized aching especially in the lower part of his back. He was given 1 gram of sodium thiosulphate intravenously which relieved his symptoms.

He was kept in bed for 3 days, during which time there was no return of symptoms.

Recovery in 3 days.

(9–1933.) A patient, who was infected on September 10, 1933, received his first injection of neoarsphenamine, 0.3 gram, on October 7. Treatment was continued, and the patient received 0.45 gram injections of neoarsphenamine on October 12, 14, 19, 21, 26, 28, and November 2. The last dose of 0.45 gram was dissolved in 10 cubic centimeters of sterile distilled water and given in 3½ minutes. He had also received 0.13 gram intramuscular injections of bismuth salicylate on October 9, 16, 23, and 30. About 3 hours after the injection of neoarsphenamine on November 2 the patient complained of headache, shortness of breath, and severe pains in his left chest. There was edema of the lips, a temperature of 100° F, pulse 110, respirations 24. White blood count, 7,200; polymorphonuclears, 67 percent. Dickens' tests, positive at 1 and 2 hours.

As treatment for the reaction he received 10 minims of adrenalin subcutaneously and 1 gram sodium thiosulphate intravenously as soon as symptoms appeared, and in the evening 1 gram of sodium thiosulphate intravenously and a hypodermic of morphine sulphate grain ¼ and atropine sulphate grain $\frac{1}{150}$.

There was marked improvement the next day, with a slight elevation of temperature in the evening. During the day he was given 2 grams of sodium thiosulphate, 1 gram orally and 1 gram intravenously.

Recovery in 2 days.

(14–1933.) A patient, who had a history of chancroids in February 1929, was sent to a hospital on August 24, 1931, because of attacks of sore throat. On August 27 a Kahn blood test was 4+.

There was a history of 3 courses of arsenicals, the amounts of which were not stated: First course, August 14 to October 8, 1931; second course, October 8, 1931, to July 6, 1932; third course, December 18, 1932, to July 5, 1933. On September 13, 1933, a fourth course was started, the patient receiving 0.3 gram of neoarsphenamine on

that date, a 0.6 gram injection on September 20, and 0.45 gram on September 27. This latter dose was dissolved in 7.5 cubic centimeters of sterile triple distilled water and given in 2 minutes. Within 5 minutes he complained of fullness in his head and a few minutes later had a chill, headache, and backache, and then nausea, vomiting, and a rapid, weak pulse.

He was given 1 cubic centimeter of adrenalin intramuscularly, 1 gram of sodium thiosulphate intravenously, and 1 grain of codeine orally.

Recovery in 8 hours.

(16–1933.) A patient, who was infected in 1913 and prior to enlistment in the Navy, received five courses of treatment, amounts not stated, as follows: January 6 to April 7, 1926; May 4 to July 15, 1926; May 6 to June 10, 1929; April 1 to July 5, 1930; January 9 to March 15, 1932.

On October 21, 1933, he received 0.3 gram of neoarsphenamine, which was dissolved in 10 cubic centimeters of water and given in 2½ minutes.

Within 15 minutes he complained of headache, rapid pulse, sense of pressure in his chest, and nausea with vomiting. His temperature was 101.4° F, pulse 130, respirations 26. White blood count, 12,200.

He was given, immediately, 1 gram of sodium thiosulphate intravenously and later in the day 2 doses of 1 gram each by mouth.

Recovery in 3 days.

(18–1933.) A patient, who was exposed in January 1922, reported for treatment on March 9, 1922, at which time he had secondaries. Treatment was instituted immediately and continued until April 26, during which time he received 7 intravenous injections of neoarsphenamine for a total dosage of 4.5 grams. Beginning again on May 2 he received three injections of neoarsphenamine for a total dosage of 1.35 grams.

During a period of over 11 years repeated blood tests were negative. On September 27 and October 9, 1933, blood tests were positive. Treatment was resumed with the administration of 0.3 gram of neoarsphenamine on October 17, 1933. This injection was given at 10 a. m. and at 11:30 a. m. the patient ate a hearty lunch and shortly afterwards went to work. In half an hour he began to perspire profusely, to pass large quantities of urine and had an insatiable thirst. His temperature at 6:30 p. m. was 101° F.

The next morning he complained of headache and backache and it was noted that he had voided only 100 cubic centimeters of urine in the previous 12 hours. His urine was negative immediately prior to the injection and continued so during the succeeding days.

Recovery in 3 days.

(23–1933.) A patient, who was infected in September 1929, began his treatment on February 11, 1930. He received five courses of

treatment, amounts not stated, as follows: February 11 to March 25, 1930; May 27, 1930, to January 22, 1931; March 13 to April 24, 1931; February 25 to May 5, 1932; July 9 to October 20, 1932.

He was started on his sixth course on February 9, 1933, when he received 0.2 gram. This was followed by eight injections of 0.45 gram each at approximately weekly intervals until May 4. The last dose was dissolved in 10 cubic centimeters of water and given in 5 minutes. Within 30 minutes he became nauseated, vomited, and had a slight rise in temperature. He had a slight occipital headache and a full bounding pulse.

He received 20 minims of adrenalin subcutaneously and 1 gram of sodium thiosulphate intravenously.

Recovery in 12 hours.

(24–1933.) A patient, who was exposed December 5, 1931, received arsenical treatment as follows:

> December 17, 1931, to February 9, 1932—8 injections of neoarsphenamine (4.5 grams).
> March 15 to April 14, 1932—5 injections of neoarsphenamine (2.7 grams).
> July 5 to August 16, 1932—8 injections of neoarsphenamine (4.5 grams).
> November 29, 1932, to January 3, 1933—6 injections of neoarsphenamine (3.0 grams).

Slight skin reactions occurred after the injections of December 28, 1932, and of January 3, 1933. On each occasion a few pruritic papules appeared on the body. No further treatment was given until March 3, when a 0.2 gram dose was followed by slight pruritus.

On May 24 another 0.2 gram dose was administered and within a few minutes the patient became ill, complaining of shortness of breath, weakness, and vomiting. All symptoms disappeared within an hour.

(25–1933.) A patient, who was infected in February 1924, received a course of arsenical treatment in 1924, another in 1925, a third in 1926, and the fourth course in 1929 and 1930. The amounts of arsenicals were not stated.

A fifth course was started on May 24, 1933, when he received 0.45 gram of neoarsphenamine. This was followed by injections of 0.45 gram each on May 31, June 7, and June 14. The injection of June 14 consisted of 0.45 gram of neoarsphenamine in 10 cubic centimeters of water and given in 45 seconds. Almost immediately after completion of the injection the patient had a typical epileptic seizure which lasted 15 minutes. He regained consciousness for a few minutes and then fell asleep. At the end of 3 hours he awoke and felt all right except for slight weakness and headache.

A blood count revealed no abnormalities and a neurological examination made the following day was negative.

He gave a history of two similar attacks following injections in 1929.

Recovery in 15 hours.

(26–1933.) A patient, who developed an initial lesion on July 9, 1932, began his first course of arsenical treatment on July 18, when he received 0.3 gram of neoarsphenamine. Following this he received injections of 0.45 gram each on July 21 and 25, and on July 28 a 0.3 gram dose. About 20 hours after his last injection he became nauseated, had a metallic taste, and pain in his back and legs. There was no elevation of temperature, vomiting, or skin manifestations. On August 10, 13 days after the last injection, he developed a slight jaundice which persisted for 3 weeks. The time interval between the injection and the appearance of jaundice caused the reporting medical officer to express doubt whether the jaundice was due to the arsenical.

A second course of arsenical treatment was begun on February 10, 1933, when he received 0.3 gram of neoarsphenamine dissolved in 10 cubic centimeters of water and given in 1 minute. About 2 hours later the patient had headache, backache, nausea, and a mild chill which was followed by elevation of temperature to 101° F.

A Dickens' test showed delayed arsenic elimination. The following day he felt better, but was weak and had a temperature of 100° F.

Laboratory data.—Kahn blood test, negative; Van den Bergh's, normal; red blood count, 4,860,000; hemoglobin, 85 percent; white blood count, 4,950; polymorphonuclears, 70 percent; lymphocytes, 24 percent; mononuclears, 4 percent; eosinophiles, 2 percent.

Recovery in 3 days.

(27–1933.) A patient, who was infected on May 25, 1933, began his first course of treatment on May 31 and continued until July 18, during which time he received seven injections of neoarsphenamine, a total of 3.75 grams.

He commenced his second course of arsenical treatment on September 5, 1933, when he received 0.3 gram of neoarsphenamine. On September 12 he received a 0.45-gram dose in 15 cubic centimeters of water, given in 3 minutes. Two hours later he developed general malaise, dizziness, chilly sensations, and a temperature of 103.4° F. He was given 1 gram of sodium thiosulphate intravenously.

Recovery in 24 hours.

(28–1933.) A patient, whose time and place of syphilitic infection was unknown, received seven injections of neoarsphenamine of 0.6 gram each in June and July 1932. A second course of treatment was started on January 10, 1933, the patient receiving 0.3 gram on that date and 0.6-gram doses on January 17 and 31. A slight reaction, the nature of which was not stated, caused a discontinuance of injections. On March 28, 1933, he received 0.6 gram of neoarsphenamine dissolved in 12 cubic centimeters of water and given in 1 minute. Five hours later he had a chill which was followed by vomiting and an elevation of temperature to 100.8° F. He was given 10 minims

of adrenalin subcutaneously and 1 gram of sodium thiosulphate intravenously.

Recovery in 9 hours.

(30–1933.) A patient, who was infected on January 6, 1933, received seven injections of neoarsphenamine, a total of 3 grams, between January 23 and April 25. On July 11 he received 0.3 gram of neoarsphenamine dissolved in 10 cubic centimeters of water and given in 2 minutes. Within 30 minutes he had a chill which was followed by an elevation of temperature to 101.4° F. He was given 1 gram of sodium thiosulphate intravenously.

Recovery in 24 hours.

(31–1933.) A patient, who was infected on September 20, 1930, received 0.45 gram of neoarsphenamine on September 30, 1930, and on October 10, 1930. He also received four injections of a heavy metal. Between August 11 and October 1, 1931, he received 2.8 grams of neoarsphenamine.

On October 15, 1933, he began a third course of arsenicals, receiving 0.3 gram on that date. This was followed by six weekly injections of 0.45 gram each. The last injection was given on November 16 and consisted of 0.45 gram in 15 cubic centimeters of triple-distilled water and given in 5 minutes. Three hours later the patient developed a slight chill, headache, backache, malaise, and a temperature of 100° F.

All symptoms disappeared in 3 hours. He was retained on the sick list for 3 days, during which time two complete blood counts and daily examinations of the urine showed no abnormalities.

(32–1933.) A patient, who was exposed on May 8, 1932, received arsenical treatment as follows:

> August 3 to September 13, 1932—6 injections of sulpharsphenamine (2.2 grams).
>
> November 15, 1932, to January 15, 1933—9 injections of neoarsphenamine (4.05 grams).

Treatment with arsenicals was interrupted because of a mild attack of cholangitis.

On August 3, 1933, he received 0.3 gram of neoarsphenamine, on August 10, 0.6 gram, and on August 16, 0.6 gram. The last dose was dissolved in 10 cubic centimeters of water and given in 2 minutes.

Two hours after the injection the patient complained of nausea, vomiting, and chilliness.

Blood count: White blood count, 6,500; polymorphonuclears, 65 percent; lymphocytes, 30 percent; monocytes, 4 percent; eosinophiles, 1 percent.

Urinalysis was negative.

Recovery in 6 hours.

(33–1933.) A patient, whose diagnosis of syphilis was established on April 15, 1930, received arsenical treatment as follows:

April 15 to May 27, 1930—7 injections of neoarsphenamine (2.7 grams).
October 22 to October 29, 1931—2 injections of neoarsphenamine (0.85 gram).
April 5, 1932—one injection of neoarsphenamine (0.45 gram).
July 19, 1932—one injection of neoarsphenamine (0.45 gram).
July 27, 1932—one injection of neoarsphenamine (0.45 gram).
October 10, 1933—one injection of neoarsphenamine (0.3 gram).
November 14, 1933—one injection of neoarsphenamine (0.6 gram).
November 21, 1933—one injection of neoarsphenamine (0.6 gram).
December 9, 1933—one injection of neoarsphenamine (0.45 gram).
December 19, 1933—one injection of neoarsphenamine (0.6 gram).

The 0.6-gram injection of December 19 was dissolved in 13 cubic centimeters of distilled water and given in 5 minutes. About 4½ hours later he had a chill and elevation of temperature to 100° F. Recovery in 2 hours.

(34, 35, 1933.) This patient, a Filipino, was given a diagnosis of yaws on November 23, 1933. As preliminary to arsenical treatment he was given three injections of bismuth salicylate. On December 9 he received 0.3 gram of neoarsphenamine and 1 week later 0.45 gram. The 0.45-gram injection of December 16 was dissolved in 15 cubic centimeters of water and given in 45 seconds. Nine hours later he developed a headache and a temperature of 103° F. The next day his temperature rose to 105° F. and then returned to normal. He complained of nothing except a slight headache.

The following day, December 18, a rash resembling secondaries appeared and disappeared within 24 hours. On December 23 it was thought he had recovered so he was given 0.45 gram of neoarsphenamine. About 6 hours later he complained of pain in his knees and thirst. His temperature rose to 103.6° F. but subsided in a few hours.

He was kept under observation until January 12, when he was returned to duty.

(36–1933.) A patient, who was exposed on February 15, 1933, reported for treatment on July 17 at which time he had secondaries and a four plus Kahn. On July 20 he received 0.2 gram of sulpharsphenamine; on July 25, 0.1 gram; on July 29, 0.2 gram and on August 4, 0.4 gram in 1.5 cubic centimeters of water. All the injections were given intramuscularly.

The first symptoms of reaction appeared 1½ hours after the last injection and consisted of chills, fever, headache, and muscle pains. Shortly thereafter the eyelids became edematous and an erythematous rash appeared on the face and chest.

Blood, white blood count, 6,200; polymorphonuclears, 78 percent; lymphocytes, 18 percent; monocytes, 4 percent. Urine was negative.

His temperature rose to 104° F. and returned to 98.6° F. in 12 hours. Recovery in 17 hours.

(37–1933.) This patient, whose diagnosis of syphilis was established on February 16, 1932, received treatment as follows:

February 16 to March 25, 1932—5 injections of sulpharsphenamine (1.8 grams).
May 27 to July 7, 1932—6 injections of sulpharsphenamine (2.2 grams).
September 13 to November 1, 1932—8 injections of neoarsphenamine (4.65 grams).
January 10 to January 17, 1933—2 injections of neoarsphenamine (0.8 gram).

A shift to sulpharsphenamine was made because of poor tolerance for neoarsphenamine.

January 31 to March 21, 1933—7 injections of sulpharsphenamine (2.3 grams).

It was noted that this course was interrupted by frequent colds which seemed to be aggravated by the arsenicals.

On June 29, 1933, a seventh course of arsenical treatment was started and the patient received 0.2 gram of sulpharsphenamine. This injection caused a slight rise in temperature and an exacerbation of the upper respiratory infection.

On July 27, 1933, the respiratory infection having subsided, another injection of 0.2 gram of sulpharsphenamine was given. Within 2 hours he had a chill which was followed by erythema, pains in the legs, and a temperature of 102.8° F.

Urinalysis was negative. White blood count, 4,500; polymorphonuclears, 70 percent; lymphocytes, 25 percent; monocytes, 5 percent.

He was given 2 subcutaneous injections of adrenalin and 1 intravenous injection of sodium thiosulphate.

Recovery in 4 hours.

(95–1933.) A patient, who was infected in November 1930, received treatment as follows:

January 28 to March 17, 1931—8 injections of neoarsphenamine and 4 injections of bismosol.
October 20, 1931, to February 23, 1932—17 injections of bismuth salicylate.
March 22 to June 22, 1932—10 injections of neoarsphenamine.
April 20, 1933—one 0.3-gram injection of neoarsphenamine.
April 27, 1933—one 0.35-gram injection of neoarsphenamine.
May 4, 1933—one 0.4-gram injection of neoarsphenamine.

The dose of May 4 was dissolved in 8 cubic centimeters of water and given in 3 minutes. Symptoms of shock developed an hour later. He was given 2 intravenous injections of sodium thiosulphate of 1 gram each. He had no further symptoms and was sent to duty 5 days later.

(6–1933.) This patient, whose date and place of syphilitic infection was unknown, came under observation because of progressive

arthritis of the large joints. A Kahn blood test was 4 plus on 3 occasions.

Arsenical treatment was instituted on May 16, 1933, at which time he received a 0.6 gram dose of neoarsphenamine. The arthritic condition improved within 24 hours. On May 23 he was given a 0.6-gram dose of neoarsphenamine and in 24 hours complained of a cough, acute coryza, and diarrhea. His temperature rose during the day to reach a maximum of 105° F. at 11 p. m.

Upon arrival at a naval hospital his temperature was 105° F., pulse 120, respirations 35. Blood pressure 118/64. Chief complaints were backache and general malaise.

The following day his temperature remained elevated and he vomited several times.

On the morning of May 27, 3 days after the injection of neoarsphenamine, a diffuse maculo-papular rash appeared on his forearm and extremities and rapidly spread over his entire body. He complained of a sore throat and his tonsils were found to be enlarged and injected. White blood count, 20,200; polymorphonuclears, 83 percent. Urine: Albumin 1 plus, arsenic 3 plus by Dickens' test.

His temperature gradually returned to normal and the rash disappeared in 4 days.

Recovery in 7 days.

(10–1933.) A patient, who was infected on July 2, 1930, received treatment as follows:

July 15 to September 10, 1930—amount not stated.
October 17 to December 9, 1930—amount not stated.
August 23 to November 2, 1932—amount not stated.
July 11, 1933—one 0.3-gram injection of neoarsphenamine.
July 18, 1933—one 0.45-gram injection of neoarsphenamine.
July 25, 1933—one 0.45-gram injection of neoarsphenamine.

The dose of July 25 was dissolved in 15 cubic centimeters of sterile distilled water and given in 1½ minutes. Following this injection, which was given at 10 a. m., he returned to his work and in half an hour complained of severe pains in his hips and chest and had a temperature of 100.2° F. and a pulse rate of 91. He became worse and was transferred to a naval hospital. Upon admission he complained of severe headache and backache with cramp-like pains in the abdomen and legs. He vomited a large quantity of material which was yellow in color and contained undigested food. A physical examination revealed palpable epitrochlear, cervical and inguinal glands, and some tenderness over the liver.

The following day, July 26, he had headache, photophobia, and backache.

July 27, no symptoms except a dull soreness in his head.

July 31, slight stiffness in both knees. The tenderness over the liver had disappeared.

Laboratory reports were as follows:

Blood

	Red blood count	Hemoglobin	White blood count	Juveniles	Band forms	Segmented	Lymphocytes	Eosinophiles	Mononuclears
July 25	4,190,000	80	8,400	3	70	22	1	1	3
July 27	4,690,000	80	5,150	----	12	56	26	----	6
Aug. 7	4,670,000	80	7,300	----	7	62	21	6	4

Urine.—Dickens' test was negative on July 25, 26, 27, 28, 31, August 3, and 8.

Blood chemistry

[Milligrams per 100 cubic centimeters]

	July 26	Aug. 2	Aug. 7
Nonprotein nitrogen	41.0	41.0	30.0
Urea nitrogen	18.0	18.0	12.0
Uric acid	4.2	4.2	2.8
Creatinine	1.9	1.7	1.3
Sugar	107.0	94.0	88.0
Chlorides	500.0	490.0	500.0

July 28. Icterus index, 7; van den Bergh's, direct reaction negative, indirect reaction less than 0.1 milligram of bilirubin per 100 cubic centimeters of serum.

August 7. Bromsulphalein test: 5-minute specimen, 45 percent; 30-minute specimen, negative.

Treatment, July 25: 1½ ounces magnesium sulphate, 1 gram sodium thiosulphate intravenously, 1,000 cubic centimeters of normal saline by proctoclysis; and saturated solution sodium bicarbonate by mouth.

Recovery in 17 days.

(12–1933.) A patient, who was exposed on July 3, 1933, developed multiple ulcers on July 12. On July 25 a darkfield examination of one of the ulcers was positive for *Treponema pallidum*. On that date he received an injection of 0.3 gram of neoarsphenamine and on July 29 one of 0.45 gram. On August 1 the Kahn blood test was 4–4–4. On August 3 he received a 0.45-gram injection of neoarsphenamine in 10 cubic centimeters of sterile distilled water. The injection was administered in 3 minutes. About 6 hours later he complained of intense headache, general malaise, and nausea. His temperature was 101° F., pulse 110 with an occasional premature contraction. The following day, August 4, his headache continued, his temperature was 103° F., pulse 110 with frequent premature contractions. On August 5 he was critically ill with a temperature of 105° F., a pulse of 144, and numerous premature contractions. Physical examination: Patient drowsy; face flushed; pupils equal and respond to light and

accommodation; neck slightly rigid and painful on motion; pulse full, bounding, and irregular; blood pressure 100/45; skin, no rash; abdomen and genitalia, negative; reflexes slightly exaggerated. On August 6 he was much improved, had no complaints, and his maximum temperature was 101° F.

He continued to improve and fully recovered by August 25. As treatment he received daily intravenous injections of sodium thiosulphate for a week and during the first 4 days of his illness was also given sodium thiosulphate by mouth.

Recovery in 22 days.

(15–1933.) A patient, who was infected on December 17, 1931, began treatment on January 25, 1932. Between that date and March 8 he received 5 injections of neoarsphenamine, the total amount of which was not stated. A second course from March 29, 1932, to May 3, 1932, consisted of 6 injections of neoarsphenamine for a total of 3.3 grams. His third course, June 14, 1932, to July 19, 1932, consisted of 8 injections of neoarsphenamine, a total of 4.5 grams. The fourth course, December 27, 1932, to January 27, 1933, also consisted of 8 injections of neoarsphenamine, a total of 4.5 grams. In April 1933 he received 4 injections of neoarsphenamine for a total of 2.1 grams.

His sixth course was begun on November 21, 1933, the patient receiving 0.2 gram on that date; 0.4 gram on November 28, and lastly a 0.2-gram injection on December 12. This injection was dissolved in 3 cubic centimeters of water and given in 1½ minutes.

In about 2 hours he returned to the sick bay complaining of nausea and dizziness. He appeared pale and weak; and had a temperature of 100° F. These symptoms disappeared after an intravenous injection of 1 gram of sodium thiosulphate. The next day, December 13, he complained of blurred vision and an examination revealed that both pupils were dilated and fixed and there was some edema of the eyelids. Temperature, pulse, and respirations were normal.

December 14. Eye examination: Negative. Urine: Negative, except for a trace of albumin. Blood chemistry: Urea nitrogen, 10.2 milligrams per 100 cubic centimeters and creatinine, 1.2 milligrams per 100 cubic centimeters. Icterus index, 5.

Blurring of vision, which was the only symptom complained of, gradually cleared up.

December 22. Recovery in 10 days.

(17–1933.) A patient, who gave a history of an initial infection in May 1933 reported for treatment late in September. At this time late secondary lesions were present and on October 4 his Kahn blood test was 4 plus.

On September 30 he received 0.3 gram of neoarsphenamine, on October 3 a 0.45-gram injection, and on October 10 a 0.6-gram injec-

tion. The last dose was dissolved in 12 cubic centimeters of distilled water and given in 2 minutes. Within 15 minutes his face became flushed, he complained of a severe headache, and his temperature rose to 104° F. Blood: Red blood count, 4,870,000; hemoglobin, 86 percent; white blood count, 10,700; polymorphonuclears, 63; lymphocytes, 35; mononuclears 1, basophiles, 1. The next day, October 11, he had a temperature of 102° F. There was redness of the face and chest but no rash. His urine was acid in reaction, specific gravity of 1.025, contained no albumin and sugar, but did contain many red blood cells. Within 24 hours his temperature returned to normal and the amount of blood in his urine had decreased. In a few days blood had disappeared from his urine. Immediately following the onset of symptoms he received 10 minims of adrenalin intramuscularly. He was given a second injection of sodium thiosulphate the following day.

Recovery in 8 days.

(19–1933.) A patient, who was last exposed on June 1, 1933, began treatment on July 8, 1933, when he received 0.3 gram of neoarsphenamine. On July 12 he was given 0.45 gram and on July 15 a 0.6-gram injection. The latter dose was dissolved in 10 cubic centimeters of water and given in 40 seconds. About 1½ hours later his temperature rose to 104° F. The next day he felt better but his temperature was still above normal. His blood count was: Red blood count, 4,380,000; hemoglobin, 85 percent; white blood count, 8,850; juveniles, 2 percent; band forms, 30 percent; segmented, 54 percent; lymphocytes, 14 percent. Urinalysis was negative.

Two days later, July 18, his urine showed a trace of albumin and occasional fine and coarse granular casts.

July 28. Recovery in 13 days.

(20–1933.) This patient was given neoarsphenamine for a disease other than syphilis (disease not stated).

He received on April 15 a 0.3-gram intravenous injection of neoarsphenamine dissolved in 10 cubic centimeters of water and given in 2 minutes. Two hours after the injection he had a chill, cyanosis, and edema of the ankles. He later developed anuria. Although the acute symptoms disappeared rather promptly he was kept under observation for a week, during which time he was constipated and complained of loss of appetite. The urine showed albumin 2 plus and an occasional white blood cell and red blood cell. Blood: Red blood count, 4,590,000; white blood count, 14,300; hemoglobin, 85 percent.

Recovery in 7 days.

(21–1933.) A patient, who developed a primary lesion on April 12, 1932, began treatment on April 14, and between that date and June 23 received 10 injections of neoarsphenamine for a total of 4.5 grams and

8 injections of bismuth salicylate. A second course from August 26, 1932, to September 29, 1932, consisted of 4 injections of neoarsphenamine, a total of 1.8 grams, and 6 injections of bismuth salicylate. Between February 27, 1933, and March 20, 1933, he received 2 injections of neoarsphenamine, 0.45 gram each, and 2 injections of bismuth salicylate. Between August 22, 1933, and September 29 he received 4 injections of neoarsphenamine, 0.45 gram each, and 6 injections of bismuth salicylate. Between September 6, 1933, and November 17, 1933, he received 30 mercurial inunctions and 3 injections of bismuth salicylate. Neoarsphenamine was temporarily discontinued because of a slight reaction. Following the series of mercury inunctions a small injection of neoarsphenamine, 0.09 gram, was given. It was dissolved in 3 cubic centimeters of water and given in 30 seconds.

In about 2 hours he began to vomit and then became chilly but did not have a definite chill. He was given 20 minims of adrenalin and 1 gram of sodium thiosulphate.

He vomited at intervals for 3 hours at the end of which time he had a slight headache and a temperature of 100.8° F.

Recovery in 3 days.

(29–1933.) A patient, who was infected in December 1932 received 10 injections of neoarsphenamine between January 4, 1933, and April 6, 1933. On July 17, 1933, he received 0.25 gram of neoarsphenamine and on July 21 and 28 injections of 0.3 gram each. His last injection was dissolved in 20 cubic centimeters of water and given slowly. In 30 minutes he had marked prostration, cyanosis, profuse perspiration, a rapid weak pulse, vomiting, and diarrhea. He was given 10 minims of adrenalin subcutaneously and 1 gram of sodium thiosulphate intravenously.

All symptoms, except general weakness, disappeared in 8 hours.

Recovery in 3 days.

(4–1933.) A patient, whose diagnosis of syphilis was established early in August 1933 received his first injection of 0.45 gram neoarsphenamine on August 15. On August 22 he was given a 0.6-gram injection and on the following day complained of chilliness, headache, and cough. His temperature was 102° F. and moist râles were heard at bases of both lungs. This acute respiratory condition cleared up in 4 days and treatment was resumed on August 29 with the administration of 0.6 gram of neoarsphenamine. Treatment was continued, the patient receiving 0.3 gram on September 19, 0.45 gram on October 10, and 0.3 gram on October 24. The last dose was dissolved in 15 cubic centimeters of sterile distilled water and given in 2½ minutes. Within 5 minutes the patient began wretching, collapsed and died almost immediately. No autopsy was performed.

The medical officer reported that 32 injections were given to other patients on the same morning, using the same technic and same lot

number of drug as was used on the patient who died and that there were no other reactions.

HERXHEIMER REACTIONS

(93–1933.) A patient, who was infected in October 1933, reported for treatment in December with a secondary rash, persistent headache, and a 4 plus Kahn blood test. He received his first injection of neoarsphenamine on December 22, the treatment consisting of 0.3 grams of neoarsphenamine in 5 cubic centimeters of water and given in 3 minutes. Four hours later the patient's headache became much worse, and shortly thereafter there was exacerbation of the rash. Within 18 hours following the injection he had an extensive erythematous rash, edema of the face, a severe headache, and a temperature of 101° F.

Recovery in 24 hours.

(94–1933.) A patient, who was infected on December 10, 1932, received nine intravenous injections of neoarsphenamine between that date and January 31, 1933. On April 4, 1933 he received 0.45 gram of neoarsphenamine in 10 cubic centimeters of distilled water. This injection was administered in 5 minutes. Seven hours later the patient developed headache, fever 102.4° F., a macular rash, and tenderness of the left submaxillary glands. Blood: Red blood count, 4,760,000; hemoglobin, 93 percent; white blood count, 8,250; polymorphonuclear, 75; lymphocytes, 19; monocytes, 4; eosinophiles, 2. By the following morning all symptoms had practically disappeared.

Recovery in 2 days.

On April 16 he received a 0.45-gram injection of neoarsphenamine and on April 25 a 0.75-gram injection. There was no reaction after either of these injections.

BLOOD DYSCRASIAS

(13–1933.) This patient was infected in August 1933 and developed an ulcer on the scrotum, on the basis of which the diagnosis of syphilis was established on September 6, 1933. He received 0.45 gram of neoarsphenamine on September 7, and on September 9 and 11 injections of 0.6 each. On September 13 he was given 0.3 gram of neoarsphenamine in 10 cubic centimeters of water. About 4 hours later he reported to the sick bay complaining of weakness. He was found to have a temperature of 102° F. and a pulse of 88. His temperature remained elevated the following day and on September 15 it was 101° F.

Laboratory reports.—Icterus index, 20; red blood count, 5,400,000; white blood count, 6,250; hemoglobin, 95 percent; polymorphonuclears, 11 percent; lymphocytes, 86 percent; basophiles, 1 percent; mononuclears, 2 percent. Urine, negative.

He experienced a slight elevation of temperature for several days and complained of general malaise.

Recovery in 8 days.

(22–1933.) A patient, whose diagnosis of syphilis was established on January 10, 1933, received treatment as follows:

January 10 to March 6, 1933—7.5 grains of neoarsphenamine.
April 6 to May 8, 1933—3.9 grains of neoarsphenamine.
March 9 to April 3, 1933—8 injections bismosol (0.8 gram).
May 11 to July 31, 1933—22 injections bismosol (2.2 grams).

On August 3, 1933, a third course of arsenical treatment was instituted, the patient receiving 0.3 gram of neoarsphenamine. This was followed by 0.45-gram injections on August 7, 10, and 14. The last injection was dissolved in 20 cubic centimeters of distilled water and given in 3 minutes. About 5 hours later the patient noted bleeding from his gums and small hemorrhagic areas in the skin where he had scratched pimples. His left forearm showed a subcutaneous hemorrhage at the site of a bruise and also a hemorrhagic area around the site of injection. His temperature, pulse, and respirations were normal and he did not feel ill.

The following morning, August 15, examination of the blood gave a bleeding time of 21½ minutes; coagulation time, 2¼ minutes; red blood count, 3,980,000; hemoglobin, 75 percent; white blood count, 5,200; platelets, 19,900; band forms, 6 percent; segmented, 61 percent; lymphocytes, 23 percent; mononuclears, 6 percent; eosinophiles, 4 percent.

August 16. Bleeding from the gums had practically stopped. Bleeding time, 7 minutes; red blood count, 3,850,000; platelets, 26,950.

August 18. No bleeding from gums. Bleeding time, 3½ minutes; red blood count, 4,250,000; platelets, 59,500.

August 22. Bleeding time, 3 minutes; red blood count, 4,120,000; platelets, 206,000.

August 27. Bleeding time, 1½ minutes; red blood count, 4,260,000; hemoglobin, 80 percent; white blood count, 8,300; platelets, 293,940; band forms, 3 percent; segmented, 67; lymphocytes, 23; mononuclears 4; eosinophiles, 3.

August 27. Recovery in 13 days.

(11–1933.) This patient was infected in May 1932. Treatment was as follows:

May 20 to July 30, 1932—8 injections of neoarsphenamine.
October 1 to December 10, 1932—8 injections of neoarsphenamine.
April 18, 1933—1 injection of neoarsphenamine (0.45 gram).
April 25, 1933—1 injection of neoarsphenamine (0.45 gram).
May 5, 1933—1 injection of neoarsphenamine (0.6 gram).
May 12, 1933—1 injection of neoarsphenamine (0.6 gram).
May 17, 1933—1 injection of neoarsphenamine (0.75 gram).

The dose given on May 17 was dissolved in 20 cubic centimeters of water and injected in 3 minutes. Five hours later he had a severe chill and headache. The next morning, May 18, there were petechiae on his ankles and wrists. By the morning of the second day similar spots were present on his arms, legs, and mucous membranes of the mouth, pharynx, and nose. His only complaint was a slight headache.

He received 5 daily injections of sodium thiosulphate of 1 gram each.

He gradually improved and recovered on June 29, 43 days after the injection.

(92–1933.) This patient was infected on December 5, 1932. Treatment prior to the reaction was:

January 16, 1933—1 injection of neoarsphenamine (0.6 gram).
January 21, 1933—1 injection of neoarsphenamine (0.3 gram).
January 31, 1933—1 injection of neoarsphenamine (0.5 gram).
February 7, 1933—1 injection of neoarsphenamine (0.4 gram).

The February 7 dose was dissolved in 8 cubic centimeters of water and injected in 2 minutes. Two days later, February 9, he complained of headache, general malaise, temperature 100° F.

February 10. Appearance of throat suggestive of septic sore throat. Temperature, 102.6° F.

February 11. Temperature, 103° F., pulse 100, respirations 20. Backache and small tender area over sacrococcygeal articulation. White blood count, 5,350; hemoglobin, 90 percent; polymorphonuclears, 55 percent; lymphocytes, 34 percent; transitionals, 2 percent; eosinophiles, 1 percent; basophiles, 8 percent. Urinalysis, negative. Specific fluid clear with no apparent increase in pressure; globulin, negative; cell count, 30.

February 12. General condition unchanged. Temperature, 102.2° F.

February 14. Improving. Temperature, 100° F.

February 18. Temperature normal. Patient feels well.

February 21. Intravenous injection of 0.4 gram of neoarsphenamine. No apparent reaction.

February 23. Headache, fever, sore throat, punctate rash on abdomen and chest.

February 25. Chill, followed by temperature of 101.8° F., pulse 120, respirations 18.

February 26. Urine, strongly acid; specific gravity, 1.019; faint trace of albumin.

February 27. Urine, positive for albumin; occasional white blood cells. Blood chemistry: Nonprotein nitrogen, 26 milligrams per 100 cubic centimeters; urea nitrogen, 14; and creatinine, 1.6.

February 28. Urine, negative.

March 3. Urine, negative.

Blood

	Red blood count	Hemoglobin	White blood count	Reticulocytes	Myeloblasts	Platelets	Myelocytes	Segmented	Juveniles	Lymphocytes	Eosinophiles	Basophiles	Mononuclears	Band forms
Feb. 26	4,480,000	75	4,000	----	2	----------	37	------	22	35	1	2	1	----
Feb. 28	4,070,000	75	4,350	9	----	276,000	15	------	30	33	9	1	2	7
Mar. 1	4,920,000	85	5,250	3	----	266,000	4	6	20	55	3	1	2	9
Mar. 3	4,720,000	85	9,800	----	----	----------	3	17	18	22	10	1	3	26
Mar. 7	4,810,000	85	14,100	----	----	----------	1	43	6	24	14	----	2	10
Mar. 13	4,910,000	90	5,850	----	----	----------	------	57	------	33	5	----	2	3
Mar. 20	4,890,000	85	5,450	----	----	----------	------	57	------	34	2	----	2	5
Mar. 21	4,260,000	80	5,450	----	----	----------	------	52	------	31	11	----	3	3

April 7. Recovery in 57 days.

In addition to the reactions which have been presented in some detail there were 11 table reactions. In these instances the individuals experienced untoward symptoms while the injection was being given or shortly thereafter. The majority of them recovered in a few minutes. This group of reactions while usually not considered important should not be regarded too lightly.

DISCUSSION

A review of the reports of Navy reactions in past years indicates that any symptom which occurs after the administration of an arsenical may be a danger signal. In a number of instances it has been noted that individuals who complain of slight discomfort subsequent to treatment eventually suffer a severe and sometimes a fatal reaction. While it is true that some types of reactions, notably hemorrhagic encephalitis, are prone to occur unexpectedly and without warning, other types are almost invariably preceded by mild manifestations.

In discussing arsenical dermatitis in the October number of the NAVAL MEDICAL BULLETIN the observation was made that more than half the cases had slight skin disturbances following the injections which preceded the one causing the more serious type of dermatitis. Evidence is accumulating to substantiate the wisdom of making blood counts on every individual who experiences any type of reaction. In fact this practice should be extended to include all individuals who do poorly under treatment. If this were done it is altogether probable that injury to the hematopoietic system would be much more frequently discovered than is now the case. In this connection Stokes in the latest edition of Modern Clinical Syphilology writes: "Current conceptions as to the rarity of this group (hematopoietic) of reactions would probably require modification if periodic blood examinations were made of patients showing malaise, pallor, pruritus, and purpuric manifestations under arsphenamine treatment."

While there is some confusion as to nomenclature in blood dyscrasias it is agreed that there are three general types of reaction.

In the thrombocytopenic type, symptoms usually appear very soon after treatment and consist of hemorrhages into the skin and mucous membranes. The symptoms vary in degree from a few petechial spots on the skin to massive hemorrhages from the gastrointestinal tract or hemorrhagic infiltration into many or all of the viscera. The striking feature of the blood picture is the marked diminution in platelets. Unless there are severe hemorrhages the other blood elements frequently show little or no change. Recovery is the rule. In the granulocytopenic type, symptoms may appear promptly after an injection but, as a rule, there is a delay of several days or longer. The first symptoms are fever, soreness of the gums, and sore throat. As the condition progresses one may easily mistake the condition for septic sore throat. There is often necrosis and sloughing of the buccal surfaces and painful swelling of the cervical glands. The blood examination will reveal a marked leukopenia with a white cell count that may be less than 1,000. The differential count shows a diminution or disappearance of the granulocytes (neutrophiles, eosinophiles, and basophiles). The prognosis is grave and those cases who recover do so after a rather protracted and severe illness. In aplastic anemia the onset is delayed, sometimes as long as a month. The symptoms may be a combination of those seen in the thrombocytopenic and granulocytopenic types or the outstanding clinical picture may be progressive anemia with sore throat and purpura as terminal events. In this type, as the name implies, there is diminution in all the blood elements due to depression of the blood-forming structures. The prognosis is poor in the aplastic cases. In general, the prevention of reactions requires constant observation of every patient who is taking any arsenical. In addition, the preparation and administration of the drug must be properly done and in accordance with well established rules of therapy. Many syphilographers have adopted a questionnaire which each patient is required to answer before each injection. The list of questions covers a comprehensive list of symptoms all of which constitute contraindications to treatment without full investigation. It is also essential to examine patients before each injection, looking particularly for a skin rash, purpuric spots, jaundice, and evidences of anemia.

A patient who reports any untoward symptoms or who shows evidence of a reaction should have a thorough examination including a complete blood count.

The preparation of the solution is important and should be done according to established methods. The water should be freshly distilled and sterilized, and the drug sifted on the surface of the water and allowed to dissolve without any agitation. The United States Public Health Service recommends that the concentration should not exceed 0.1 gram to 5 cubic centimeters of water and that the rate of injection should not exceed 0.1 gram in 30 seconds.

In conclusion we wish to express our appreciation of the cooperation of the medical officers who have furnished the reports on which these articles are based. These articles which have become an annual fixture are regarded only as summaries of Navy experience with arsenicals. They do not in any sense preclude the reporting of cases by medical officers in separate articles. It is hoped that medical officers who have cases will submit them for publication and in greater detail than is possible in an article of this nature.

We also wish to acknowledge the valuable material which we have been permitted to reproduce from The Modern Treatment of Syphilis, J. E. Moore, and Modern Clinical Syphilology, J. F. Stokes.

TOXOID FOR DIPHTHERIA IMMUNIZATION

The following paragraphs are reprinted from the Quarterly Bulletin of the Department of Health, city of New York. It is of timely interest because alum precipitated toxoid has been recently placed on the supply table:

"Alum-precipitated diphtheria toxoid, as now supplied by the health department's laboratories, is developed from toxin to which 0.4 percent of formalin has been added. The mixture is kept in an incubator at 38° C. for a period of 1 month. The addition of the formalin to the toxoid destroys its toxicity completely without markedly impairing its immunizing power. As a final step 1.5 percent alum is added. This causes a precipitation of the toxoid. Havens of the Alabama State Department of Health found that this precipitated toxoid, when injected into the tissues, because of its slow absorption, had greater immunizing power than when it was in solution. Dr. William H. Park has corroborated these findings. Toxoid is therefore rapidly replacing toxin-antitoxin. It is a more potent immunizing agent and is a more stable modification of diphtheria toxin.

"From what has been said it is clear that the precipitate is the active immunizing agent. For this reason it is important that the hypodermic needle used for the injection be sufficiently large to allow the precipitate to pass through readily. In using clear solutions, such as toxin-antitoxin, physicians have usually made use of a no. 25 needle. Such a needle is too fine for the alum-precipitated toxoid; it will draw up mostly clear fluid and allow only very little precipitate to pass through. In using precipitated toxoid, therefore, physicians should use a needle of approximately no. 23 gage. In this way all of the valuable precipitate will be injected into the tissues. The precipitated toxoid, while it gives little or no reaction in infants and young children, does give more or less severe reaction in a considerable percentage of older children and adults.

"*Dosage and administration.*—In children under 3 years, only 1 dose of 1 cubic centimeter is given. In school children and adults a smaller first dose, namely, 1/10 cubic centimeter is advisable. If no annoying reaction follows this dose a second injection of 0.5 cubic centimeter is given 1 or 2 weeks later. If there is a severe reaction after the first small dose, the amount of the following dose is not increased, and a third dose of the same size is given 2 weeks after the second."

HEALTH OF THE NAVY

Based on statistical returns for diseases and injuries occurring in April, May, and June, 1934, the general admission rate was 452 per 1,000 per annum. The rate for the corresponding quarter of 1933 was 470. The median rate for the second quarter, as indicated by the records of the preceding 5 years, is 497. The admission rate from disease was 385 per 1,000 per annum, as compared with 457, the 5-year median for the corresponding 3 months. An increase occurred in admissions for injuries due to an increase in accidents occurring within command but not connected with work. The rate for the quarter under discussion was 67, as compared with 55, the rate for the previous quarter of 1934, and 62, the rate for the second quarter of 1933. The median or expected rate for the corresponding quarter of the previous 5 years is 53.

There was a continuation of the mild epidemics of the common infections of the respiratory type and of measles and German measles during April, May, and June. A total of 1,702 cases of acute respiratory diseases and 408 cases of measles and German measles were reported by shore stations in the United States for this quarter, as compared with 1,409 cases of acute respiratory infections and 301 cases of measles and German measles for the first quarter of the year. As usual, catarrhal fever predominated.

The United States naval training station, Norfolk, Va., reported that mild epidemics of catarrhal fever, German measles, and measles have been existent at that station. A total of 1,166 cases of the common infections of the respiratory type were recorded for the quarter, which was 68.5 percent of all cases of these diseases notified by shore stations in the United States. Admissions for German measles and measles increased from 88 during the first quarter to 313 for the second quarter. The cases of the above diseases were mild with practically no complications, but a check-up revealed that pneumonia; otitis, media, acute; empyema; and cerebrospinal meningitis had complicated cases transferred to hospital.

The United States Naval Training Station, San Diego, Calif., reported 168 cases of acute respiratory diseases or a reduction of 41.2 percent over the first quarter of the year.

The United States Naval Air Station, Norfolk, Va., recorded 46 cases of the common infections of the respiratory type and 14 cases of

measles and German measles for the quarter, as compared with 23 cases and 2 cases, respectively, for the previous quarter.

Shore stations outside of the continental limits of the United States reported 209 cases of the acute respiratory diseases for the quarter, as follows: Hawaii, 42; Panama Canal Zone, 47; Philippine Islands, 17; Guam, 3; Guantanamo Bay, Cuba, 6; Haiti, 21; Samoa, 1; and China, 72.

Four cases of chickenpox were reported, 2 from the Naval Training Station, San Diego, Calif., in June; 1 from the Navy Yard, Washington, D. C., in April; and 1 from the Submarine Base, New London, Conn., in May.

One case of scarlet fever occurred in April in a recruit on leave from the Naval Training Station, San Diego, Calif., and one in June at the Navy Yard, New York, N. Y.

A recruit with 5 weeks' service was transferred from the Naval Training Station, Norfolk, Va., to the Norfolk Naval Hospital with "diagnosis undetermined (cerebrospinal fever)." Diagnosis was definitely established as cerebrospinal fever and the patient died the day after admission to the hospital.

The admission rate, all causes, for forces afloat was 327 per 1,000, as compared with 396 per 1,000, the rate for the first quarter of the year, and 444, the corresponding median rate for the preceding 5 years. A total of 781 cases of catarrhal fever was notified from all ships for the quarter.

Ships recorded admissions for acute respiratory infections, distributed over the quarter as follows:

Ship	April	May	June	Ship	April	May	June
U. S. S. Argonne	18	17	5	U. S. S. Medusa	14	28	8
U. S. S. California	10	4	25	U. S. S. Mississippi	17	28	10
U. S. S. Chaumont	17	7	9	U. S. S. New Orleans	27	13	5
U. S. S. Idaho	39	14	16	U. S. S. Saratoga	16	14	13
U. S. S. Lexington	37	48	27	U. S. S. Tennessee	11	11	19
U. S. S. Maryland	21	17	5	U. S. S. Wyoming	15	45	10

One hundred and forty-six cases of German measles were reported during the quarter. The U. S. S. *Pennsylvania* reported 12 cases in April and 17 in May; the U. S. S. *Mississippi*, 28 cases in May; the U. S. S. *Chaumont*, 30 cases in June; the U. S. S. *Astoria*, 28 cases in June; and the U. S. S. *Oklahoma*, 12 cases in June.

Three cases of scarlet fever were notified by forces afloat during the quarter—one from the U. S. S. *Augusta* in May, one from the U. S. S. Oklahoma in April, and one from the U. S. S. *Lexington* in June.

A total of 58 cases of mumps were reported for the quarter, the majority occurring on board the U. S. S. *Oklahoma* and the U. S. S. *Idaho* in April.

Ten cases of chickenpox among forces afloat were reported by eight ships, 4 cases occurring in April, 4 in May, and 2 in June.

TABLE 1.—*Summary of morbidity in the United States Navy for the quarter ended June 30, 1934*

Average strength	Forces afloat, 72,225		Forces ashore, 34,775		Entire Navy, 107,000	
	Admissions	Rate per 1,000	Admissions	Rate per 1,000	Admissions	Rate per 1,000
All causes	5,915	327.59	6,167	709.36	12,082	451.66
Diseases only	4,889	270.76	5,398	620.91	10,287	384.56
Injuries	1,024	56.71	759	87.30	1,783	66.65
Poisonings	2	.11	10	1.15	12	.45
Communicable diseases transmissible by oral and nasal discharges (class VIII):						
(A)	272	15.06	657	75.57	927	34.65
(B)	1,115	61.75	2,001	230.17	3,116	116.49
Venereal diseases	1,214	67.23	501	57.63	1,715	64.11

TABLE 2.—*Deaths reported, entire Navy, during the quarter ended June 30, 1934*

Cause—disease		Navy			Marine Corps		Nurse Corps	Total
Primary	Secondary or contributory	Officers	Midshipmen	Men	Officers	Men		
Average strength		9,343	1,492	79,496	1,174	15,133	362	107,000
Abscess, scalp	Septicemia					1		1
Adrenalitis	None				1			1
Appendicitis, acute	Abscess, liver			1				1
Do	Poisoning, anesthesia (novocain, spinal and ether), drop.			1				1
Carcinoma:								
Adrenal cortex	None			1				1
Generalized	do			1				1
Stomach	Obstruction, intestinal, from external causes.			1				1
Cellulitis:								
Face	Thrombosis, cavernous sinus.	1						1
Shoulder and neck	Septicemia					1		1
Cerebrospinal fever	None			1				1
Gastrick ulcer	Hemorrhage			1				1
Gonococcus infection, endocardium.	do			1		1		2
Glioma, brain	do			1				1
Hemorrhage, subdural	Dementia, praecox			1				1
Leukemia	None			1				1
Do	Pneumonia, broncho			1				1
Myocarditis, chronic	Arterial hypertension			1				1
Do	Nephritis, acute			1				1
Do	Thrombosis, coronary artery.	1						1
Nephritis, chronic	None	1						1
Obstruction, intestinal from spastic or paralytic causes.	Calculus, renal	1						1
Obstruction, intestinal, from external causes.	None			1				1
Pancreatitis, acute	do			1				1
Pansinusitis	Meningitis, cerebral			2				2
Pneumonia, broncho	Embolism			1				1
Do	Pleurisy, suppurative			1				1
Pneumonia, lobar	Meningitis, cerebrospinal, acute.			1				1
Do	Thrombosis, cerebral			1				1
Pleurisy, suppurative	Meningitis, cerebrospinal			1				1
Syphilis	Poisoning, neoarsphenamine, acute.			1				1
Do	Encephalitis, acute			1				1
Sarcoma, pancreas	Hemorrhage, intestinal			1				1
Thrombosis, coronary artery	None			1				1
Tuberculosis, pulmonary, chronic.	do			4				4
Tuberculosis, general miliary.	do			1				1
Ulcer, stomach	Hemorrhage, stomach			1				1
Total for disease		4		33	1	3		41

TABLE 2.—*Deaths reported, entire Navy, during the quarter ended June 30, 1934*—Continued

Cause—disease		Navy			Marine Corps		Nurse Corps	Total
Primary	Secondary or contributory	Officers	Midshipmen	Men	Officers	Men		
INJURIES AND POISONINGS								
Asphyxiation, illuminating gas.	None			1				1
Avulsion, skull, cerebrum, temporoparietal.	do					1		1
Drowning	do			6	1			7
Electric shock, injury from	do			1				1
Fracture, compound, skull	do			2		1		3
Fracture, compound, cervical vertebra.	do			1				1
Fracture, simple	do			3				3
Fracture near joint with dislocation, cervical vertebra.	Intraspinal injury					1		1
Heat exhaustion	None	1						1
Injuries, multiple, extreme	do	1		4				5
Intracranial injury	do	1						1
Strangulation, neck	Psychoneurosis unclassified.			1				1
Wound, gunshot:								
Chest	None					1		1
Head	do					2		2
Heart	do			1				1
Wound, punctured, heart	do					1		1
Poisoning, acute:								
Bichloride of mercury	do			1				1
Cyanide, sodium	do	1						1
Do	Psychosis, manic depressive	1						1
Heroin	None					1		1
Total for injuries and poisonings.		5		21	1	8		35
Grand total		9		54	2	11		76
Annual death rate per 1,000:								
All causes		3.85		2.72	6.81	2.91		2.84
Disease only		1.71		1.66	3.41	.79		1.53
Drowning				.30	3.41			.26
Poisoning		.86		.05		.26		.15
Other injuries		1.28		.70		1.85		.90

ADMISSIONS FOR INJURIES AND POISONINGS, SECOND QUARTER, 1934

The following table, indicating the frequency of occurrence of accidental injuries and poisonings in the Navy during the second quarter, 1934, is based upon all form F cards covering admission in those months which have reached the Bureau:

	Admissions, April, May, and June 1934	Admission rate per 100,000 per annum	Admission rate per 100,000, year 1933
INJURIES			
Connected with work or drill	679	2,538	2,237
Occurring within command but not associated with work	633	2,366	1,692
Incurred on leave or liberty or while absent without leave	471	1,761	1,757
All injuries	1,783	6,665	5,686
POISONINGS			
Industrial poisoning	5	19	26
Occurring within command but not connected with work	3	11	191
Associated with leave, liberty, or absence without leave	4	15	19
Poisonings, all forms	12	45	236
Total injuries and poisonings	1,795	6,710	5,922

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.	
	April, May, and June 1934	Year 1933	April, May, and June 1934	Year 1933	April, May, and June 1934	Year 1933
Percent of all injuries	38.1	39.3	35.5	29.8	26.4	30.9
Percent of all poisonings	41.7	10.9	25.0	80.9	33.3	8.2
Percent of total admissions, injury and poisoning titles	38.1	38.2	35.4	31.8	26.5	30.0

Poisoning by a narcotic drug or by ethyl alcohol is recorded under the title "Drug addiction" or "Alcoholism", as the case may be. Such cases are not included in the above figures.

There were no cases during the second quarter of 1934 worthy of notice from the standpoint of accident prevention.

STATISTICS RELATIVE TO MENTAL AND PHYSICAL QUALIFICATIONS OF RECRUITS

The following statistics were taken from monthly sanitary reports submitted by naval training stations:

April, May, and June 1934	U. S. Naval Training Station	
	Norfolk, Va.	San Diego, Calif.
Recruits received during the period	2,372	1,550
Recruits appearing before Board of Medical Survey	55	0
Recruits recommended for discharge from the Service	54	0
Recruits discharged by reason of medical survey	47	0
Recruits held over pending further observation	5	0
Recruits transferred to the hospital for treatment, operation, or further observation for conditions existing prior to enlistment	38	31

The following table was prepared from reports of medical surveys in which disabilities or disease causing the surveys were noted as existing prior to enlistment. With certain diseases, survey followed enlistment so rapidly that it would seem that many might have been eliminated in the recruiting office.

Cause of survey	Number of surveys	Cause of survey	Number of surveys
Absence, acquired, teeth	2	Flat foot	2
Ankylosis, bony. Proximal interphalangeal joint, right middle finger	1	Gastritis, chronic	1
Arterial hypertension	2	Gonococcus infection	4
Arthritis, chronic	2	Hernia, inguinal	3
Asthma	1	Hypertrophy, bone	1
Bronchitis, chronic	2	Malocclusion, teeth	1
Cicatrix, skin, left forearm	1	Myopia	1
Color blindness	1	Nephritis, chronic	1
Constitutional psychopathic inferiority, without psychosis	3	No disease (under height)	1
Constitutional psychopathic state, emotional instability	2	Nostalgia	2
Constitutional psychopathic state, inadequate personality	2	Otitis, media, chronic	2
Deformity, acquired, right clavicle	1	Psychoneurosis, hysteria	2
Dementia praecox	4	Psychoneurosis, neurasthenia	4
Dislocation, articular cartilage, knee	2	Psychosis, unclassified	1
Dyspituitarism	1	Pterygium (right eye)	1
Eczema, chronic, scrotum	1	Seasickness	1
Effort syndrome	1	Syphilis	4
Enuresis	12	Tachycardia	1
Epilepsy	5	Tuberculosis, pulmonary, chronic, active, incipient	1
		Union of fracture, faulty	2
		Valvular heart disease	4

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UNITED STATES NAVAL MEDICAL BULLETIN

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NAVY DEPARTMENT,
Washington, March 20, 1907.

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

TRUMAN H. NEWBERRY,
Acting Secretary.

Owing to exhaustion of certain numbers of the BULLETIN and the frequent demands from libraries, etc., for copies to complete their files, the return of any of the following issues will be greatly appreciated:

Volume IX, no. 1, January 1915.
Volume X, no. 2, April 1916.
Volume XI, no. 3, July 1917.
Volume XII, no. 1, January 1918.
Volume XII, no. 3, July 1918.

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PREFACE

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

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

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

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

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

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

NOTICE TO SERVICE CONTRIBUTORS

Contributions to the BULLETIN should be typewritten, *double spaced*, on plain paper, and should have wide margins. Fasteners which will not tear the paper when removed should be used. Nothing should be written in the manuscript which is not intended for publication. For example, addresses, dates, etc., not a part of the article, require deletion by the editor. The BULLETIN endeavors to follow a uniform style in heading and captions, and the editor can be spared much time and trouble, and unnnecessary changes in manuscript can be obviated if authors will follow in these particulars the practice of recent issues.

The greatest accuracy and fullness should be employed in all citations, as it has sometimes been necessary to decline articles otherwise desirable because it was impossible for the editor to understand or verify references, quotations, etc. The frequency of gross errors in orthography in many contributions is conclusive evidence that authors often fail to read over their manuscripts after they have been typewritten.

Contributions must be received at least 3 months prior to the date of the issue for which they are intended.

The editor is not responsible for the safe return of manuscripts and pictures. All materials supplied for illustrations, if not original, should be accompanied by refernce to the source and a statement as to whether or not reproduction has been authorized.

The BULLETIN intends to print *only original articles, translations, in whole or in part, reviews, and reports and notices of Government or departmental activities, official announcements, etc. All original contributions are accepted on the assumption that they have not appeared previously and are not to be reprinted elsewhere without an understanding to that effect.*

U.S. NAVAL MEDICAL BULLETIN

VOL. XXXIII APRIL 1935 No. 2

SPECIAL ARTICLES

THE EFFICACY OF TYPHOID PROPHYLAXIS IN THE UNITED STATES NAVY [1] [2]

By S. S. COOK, Lieutenant Commander, Medical Corps, United States Navy

Typhoid fever was for many years a serious problem in the United States Navy and epidemics were frequent aboard ship and on shore stations. That much time and thought was expended in efforts to solve this problem is evidenced by frequent references to this disease in the reports of medical officers of the late years of the nineteenth century and in the early years of the twentieth century.

For purposes of this study the 44-year period 1890–1933 has been chosen. The initiation of compulsory typhoid prophylaxis in 1912 serves as the dividing line thus separating the 44 years into 2 periods of 22 years each. In this study will be given a review of certain features of the disease, namely the annual prevalence, deaths, case fatality, and geographical distribution during the period when vaccine was not administered and for comparison during the years when vaccine was administered. There will also be given the effects of inoculations with relation to severe reactions and deaths and for a portion of the period an account of the incidence of typhoid fever in individuals previously inoculated.

Reports of encouraging results following the use of antityphoid prophylaxis prompted naval authorities early in 1910 to make typhoid vaccine available to those who wished to avail themselves of it. In 1911 it was decided to require inoculation of all naval personnel and this was done in General Order No. 133 dated December 1, 1911. This order reads:

> 1. As soon as practicable after the receipt of this order, typhoid prophylactic will be administered to all officers and enlisted men of the Navy and Marine Corps, under the age of 45 years, who have not already received it or who have not already had a well-defined case of typhoid fever. Officers and enlisted men on leave, or on duty where no medical officer is available, will receive the typhoid prophylactic upon their arrival at the first station where this measure is practicable.

[1] Read before southern branch, American Public Health Association, San Antonio, Tex., Nov. 14, 1934.

[2] Printed in American Journal of Public Health, March 1935.

2. Typhoid prophylactic will be administered to all recruits under 45 years of age immediately upon their arrival at a training station, receiving ship, or marine recruit depot. This applies to all men reenlisting who have not received the prophylactic treatment within 2 years previously or had a well-defined case of typhoid fever; in case of doubt the prophylactic will be administered. Every applicant for enlistment in the Navy or Marine Corps will be informed that he must submit to typhoid prophylaxis, and unless he agrees to acquiesce in this procedure be will not be considered eligible for enlistment.

3. Typhoid phophylaxis must be regarded as a supplemental safeguard, and under no circumstances will the usual sanitary precautions be modified or set aside.

4. The antityphoid serum should be obtained from the Bureau of Medicine and Surgery by telegraphic or written request.

This order was promulgated to the service and immediate steps taken to comply with its provisions. Inoculations were begun early in 1912 and by the end of that year practically every person in the Navy to whom the order applied had received three injections of typhoid vaccine.

While there have been some minor modifications of the original instructions it has not been found necessary to alter materially the original method. At present, two courses of three injections each are generally regarded as adequate protection. The vaccine has been obtained from the Army Medical School.

In table I are shown the admissions and deaths from typhoid and paratyphoid fevers among officers and enlisted men of the United States Navy for the 44-year period, 1890–1933. In this period there were 3,409 admissions and 296 deaths with a case fatality of 8.68 percent. In the 22-year period, 1890–1911, in which typhoid vaccine was not administered, there were 2,837 admissions and 252 deaths, and in the 22 years, 1912–33, there were 572 admissions and 44 deaths. The case fatality rate for 1890–1911 was 8.88 percent and for 1912–33 it was 7.69 percent.

TABLE I.—*Typhoid fever and paratyphoid fevers, admissions and deaths, by years, United States Navy, 1890–1933*

Year	Mean strength	Admissions	Admission rate per 100,000	Deaths	Death rate per 100,000	Case fatality rate per 100
1890	11,768	41	348.40	1	8.50	2.44
1891	11,501	37	321.71	5	43.47	13.51
1892	11,775	59	501.06	7	59.45	11.86
1893	12,109	47	388.14	4	33.03	8.51
1894	12,520	53	423.32	4	31.95	7.55
1895	13,191	31	235.01	6	45.49	19.35
1896	14,196	56	394.48	10	70.44	17.86
1897	15,734	52	330.49	4	25.42	7.69
1898	23,986	109	454.43	12	50.03	11.01
1899	20,819	134	643.64	11	52.84	8.21
1900	23,756	175	736.66	25	105.24	14.29
1901	26,873	105	390.73	14	52.10	13.33
1902	31,240	125	400.13	14	44.81	11.20
1903	37,248	188	504.73	20	53.69	10.64
1904	40,555	194	478.36	21	51.78	10.82
1905	41,313	172	416.33	11	26.63	6.40
1906	42,529	230	540.81	14	32.92	6.09
1907	46,336	249	537.38	17	36.69	6.83

TABLE I.—*Typhoid fever and paratyphoid fevers, admissions and deaths, by years, United States Navy, 1890–1933*—Continued.

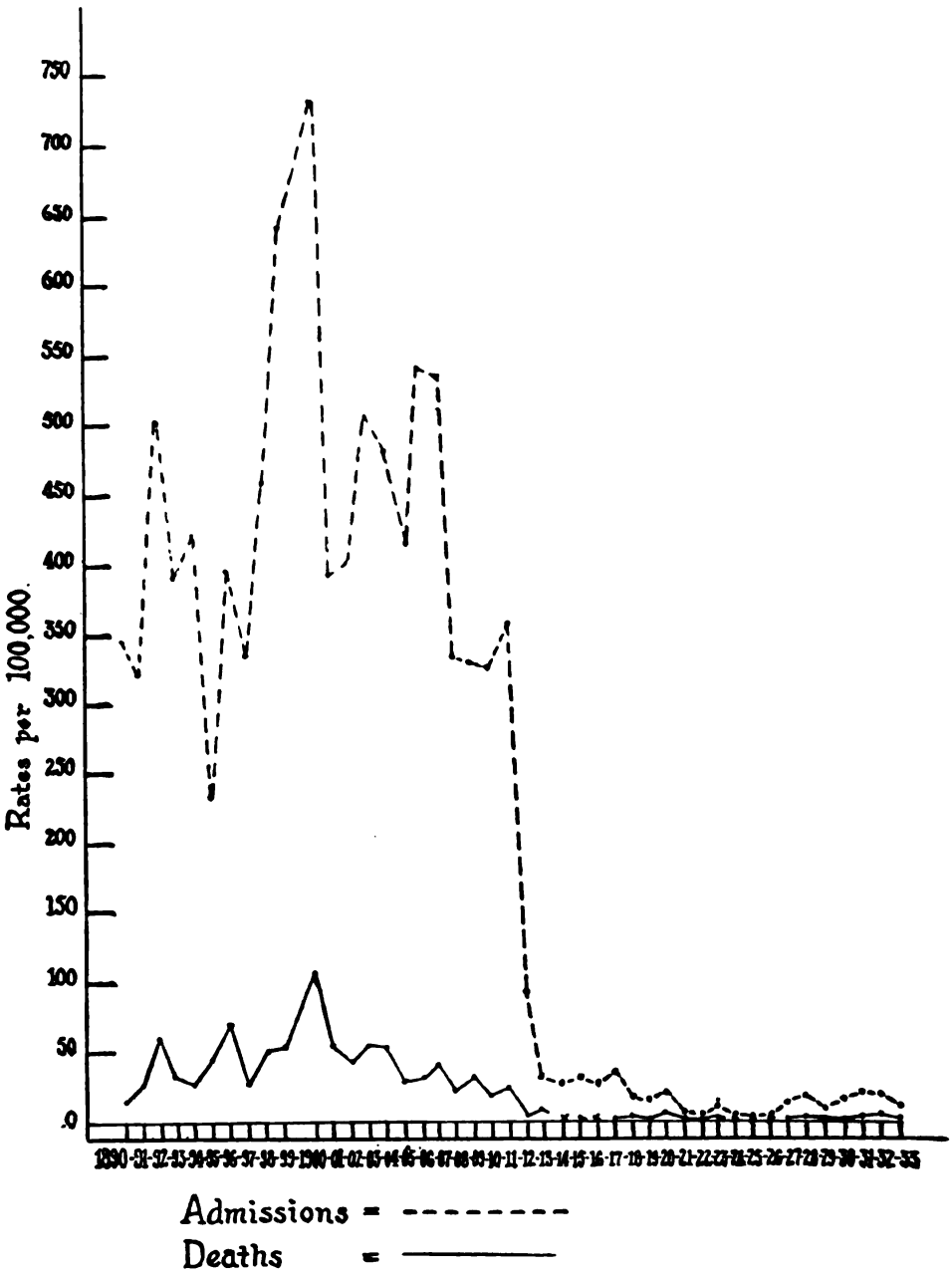
Year	Mean strength	Admissions	Admission rate per 100,000	Deaths	Death rate per 100,000	Case fatality rate per 100
1908	52,913	176	332.62	10	18.90	5.68
1909	57,172	189	330.58	17	29.73	8.99
1910	58,691	193	328.84	10	17.04	5.18
1911	61,399	222	361.57	15	24.43	6.76
1912	61,897	57	92.09	2	3.23	3.51
1913	65,926	23	34.89	4	6.07	17.39
1914	67,141	22	32.77	0	0	0
1915	68,075	23	33.79	1	1.47	4.35
1916	69,294	21	30.31	0	0	0
1917	245,580	86	35.02	1	.41	1.16
1918	503,792	83	16.48	9	1.79	10.84
1919	298,774	49	16.40	2	.67	4.08
1920	140,773	35	24.86	7	4.97	20.00
1921	148,861	16	10.75	1	.67	6.25
1922	122,126	12	9.83	2	1.64	16.67
1923	116,565	14	12.01	2	1.72	14.29
1924	119,280	7	5.87	0	0	0
1925	115,381	4	3.47	0	0	0
1926	113,756	4	3.52	0	0	0
1927	115,316	15	13.01	1	.87	6.67
1928	116,047	20	17.23	2	1.72	10.00
1929	117,388	12	10.22	2	1.70	16.67
1930	117,453	18	15.33	1	.85	5.56
1931	112,767	22	19.51	2	1.77	9.09
1932	110,717	20	18.06	3	2.71	15.00
1933	108,183	9	8.32	2	1.85	22.22

There is a striking difference in admission rates and death rates in the two periods. From 1890 to 1911, in only 1 year was the admission rate lower than 300 per 100,000. In 6 of the 21 years the rate exceeded 500, and in 1899 and 1900 the rate was 643 and 736 respectively. There was an abrupt drop from a rate of 361 in 1911 to 92 in 1912. Antityphoid inoculation of the entire Navy was not completed until the end of 1912 which may account for the higher rate in 1912 than that of succeeding years. Except for the rate of 92 in 1912 the admission rate in the period 1912–33 has never exceeded 35 per 100,000. In the 5 years, 1913–17, immediately following the inauguration of compulsory antityphoid prophylaxis, the rate fluctuated between 30 and 35. In the 16-year period, 1918–33, there was only 1 year when the rate exceeded 20 and in 4 of these years the rate was less than 10 per 100,000.

The decline in death rates which occurred after the introduction of compulsory antityphoid inoculation is remarkable. In the 22 years 1890–1911 there were only 3 occasions when the death rate was less than 20 per 100,000. In 8 of these years the rate exceeded 50 and in 15 of the 22 years it exceeded 30. In the 22-year period, 1912–33, the death rate per 100,000 was less than 3 in each year except 1912, 1913, and 1920, when it was 3, 6, and 5, respectively. In the 19 years when the death rate was less than 3 there were no deaths in 5 of the years, and in 5 others the rate was less than 1. The death rate declined abruptly from 24 in 1911 to 3 in 1912.

Prior to 1913, typhoid fever and paratyphoid fevers were not separated in morbidity reports. In tables II and III admissions and

Typhoid Fever and Paratyphoid Fevers, Admissions and Deaths, by Years, United States Navy, 1890-1933.



deaths for typhoid fever and for paratyphoid fevers from 1913–33 are shown.

TABLE II.—*Typhoid fever—admissions and deaths, by years, United States Navy, 1913–33*

Year	Admissions	Admission rate per 100,000	Deaths	Death rate per 100,000	Year	Admissions	Admission rate per 100,000	Deaths	Death rate per 100,000
1913	22	33.37	4	6.07	1924	5	4.19	0	0
1914	13	19.36	0	0	1925	3	2.60	0	0
1915	18	26.44	1	1.47	1926	3	2.64	0	0
1916	17	24.53	0	0	1927	9	7.81	1	.87
1917	66	26.88	1	.41	1928	9	7.76	1	.86
1918	65	12.90	9	1.79	1929	7	5.96	1	.85
1919	36	12.05	2	.67	1930	11	9.37	1	.85
1920	28	19.89	6	4.26	1931	16	14.19	2	1.77
1921	13	8.73	1	.67	1932	12	10.84	3	2.71
1922	8	6.55	2	1.64	1933	6	5.55	2	1.85
1923	11	9.44	2	1.72					

TABLE III.—*Paratyphoid fevers—admissions and deaths, by years, United States Navy, 1913–33*

Year	Admissions	Admission rate per 100,000	Deaths	Death rate per 100,000	Year	Admissions	Admission rate per 100,000	Deaths	Death rate per 100,000
1913	1	1.52	0	0	1924	2	1.68	0	0
1914	9	13.40	0	0	1925	1	.87	0	0
1915	5	7.35	0	0	1926	1	.88	0	0
1916	4	5.77	0	0	1927	6	5.20	0	0
1917	20	8.14	0	0	1928	11	9.48	1	.86
1918	18	3.57	0	0	1929	5	4.26	1	.85
1919	13	4.35	0	0	1930	7	5.96	0	0
1920	7	4.97	1	.71	1931	6	5.32	0	0
1921	3	2.02	0	0	1932	8	7.23	0	0
1922	4	3.28	0	0	1933	3	2.77	0	0
1923	3	2.57	0	0					

It may be seen in table II that typhoid fever caused 378 admissions and 39 deaths in the 21-year period, 1913–33. This gives an average of about 18 cases and about 2 deaths per year in a population of approximately 140,000. If the war years of 1917 and 1918 are excluded a better idea of the usual prevalence is obtained. Thus, in 19 of the years since antityphoid inoculation has been in use there has been an average of about 13 cases and 1 death in a population of approximately 118,000.

In table III are shown admissions and deaths, by years, for paratyphoid fevers for the period 1913–33. During these years there were 137 admissions and 3 deaths. From 1912 to 1917 straight typhoid vaccine was used. From October 1917 until the summer of 1924 a triple vaccine, which contained typhoid, paratyphoid A and paratyphoid B organisms, was given. Since 1924 straight typhoid vaccine has been used. There does not appear to have been a marked decrease in the prevalence of paratyphoid fevers as a result of the use of the triple vaccine. The numbers of cases are too small to warrant definite statements one way or the other.

TABLE IV.—*Typhoid fever and paratyphoid fevers—annual death rates per 100,000 strength, U. S. Navy, and per 100,000 population, registration area of the United States, 1901–29* [1]

Year	U. S. Navy	Registration area of the United States	Year	U. S. Navy	Registration area of the United States
1900	105.24	35.9	1915	1.47	12.4
1901	52.10	32.4	1916	0	13.3
1902	44.81	34.5	1917	.41	13.5
1903	53.69	34.4	1918	1.79	12.6
1904	51.78	32.0	1919	.67	9.2
1905	26.63	27.8	1920	4.97	7.8
1906	32.92	32.1	1921	.67	9.0
1907	36.69	30.3	1922	1.64	7.4
1908	18.90	24.3	1923	1.72	6.8
1909	29.73	21.1	1924	0	6.7
1910	17.04	23.5	1925	0	8.0
1911	24.43	21.0	1926	0	6.5
1912	3.23	16.5	1927	.87	5.5
1913	6.07	17.9	1928	1.72	4.9
1914	0	15.5	1929	1.70	4.2

[1] The death rates for the registration area of the United States for the period 1900–1919 are those shown in mortality statistics under title no. 1, international nomenclature for typhoid fever and paratyphoid fevers, and for 1920–29 under title no. 1a for typhoid fever and no. 1b for paratyphoid fevers.

In table IV are shown annual death rates per 100,000 strength, United States Navy and per 100,000 population, registration area of the United States, 1900–1929.

During the first 5 years of this period Navy death rates were consistently higher than those of the registration area of the United States. Beginning with 1905 and continuing through 1911 Navy rates paralleled quite closely those of the registration area. From 1912 on there is no similarity. The death rate in the Navy dropped from 24 in 1911 to 3 in 1912 while in the registration area it dropped from 21 to 16. The decline in the Navy was precipitous whereas in the registration area the decline which had been in progress since 1900 continued in an orderly but gradual fashion. So far as can be determined the only change which was made in the Navy in 1912 that was not made in the registration area was the inauguration of antityphoid inoculation. Such improvements as were effected in sanitation including water purification doubtless affected the Navy as well as the civilian population. If it were found that the principal sources of infection for the Navy were localities where notable sanitary improvements had been made in 1912 this factor would of necessity require consideration.

In the paragraphs which follow the geographic distribution of typhoid fever in the Navy, 1900–1912, is given in sufficient detail to substantiate the statement that the disease was prevalent in the Navy in a number of localities both in the United States and elsewhere.

GEOGRAPHIC DISTRIBUTION

Abstracts are given from official records in chronological order:

1900.—Island of Guam, 18 cases; a ship, Atlantic Fleet, 4 cases; Cavite, P. I., 26 cases; a ship, Asiatic Fleet, 11 cases.

1901.—Asiatic station, 19 cases; a ship, Atlantic Fleet, 9 cases.

1902.—Training station, San Francisco, 7 cases.

1903.—San Juan, P. R., 7 cases in Atlantic Fleet; Naval Academy, Annapolis, Md., 7 cases; Navy Yard, Norfolk, Va., 6 cases; Navy Yard, Philadelphia, Pa., 9 cases; a ship, Atlantic Fleet, 7 cases.

1904.—Portsmouth, N. H., 22 cases; receiving ship (U. S. S. *Franklin*), Norfolk, Va., 11 cases; a ship, Atlantic Fleet, 13 cases.

1905.—Training station, Newport, R. I., 12 cases; receiving ship (U. S. S. *Franklin*), Norfolk, Va., 16 cases; Naval Academy, Annapolis, Md., 11 cases; Asiatic Fleet, 16 cases.

1906.—A ship, Atlantic Fleet, 44 cases; Naval Academy, Annapolis, Md., 9 cases; training station, Newport, R. I., 16 cases; Asiatic Fleet, 8 cases; foreign stations, 13 cases.

1907.—A ship, Atlantic Fleet, 8 cases; foreign stations, 11 cases; Asiatic Fleet, 9 cases.

1908.—Norfolk, Va., 31 cases; Las Animas, Colo., 28 cases.

1909.—Atlantic Fleet, 98 cases; Chelsea, Mass., 24 cases.

1910.—Receiving ship (U. S. S. *Franklin*), Norfolk, Va., 17 cases; a ship, Atlantic Fleet, 12 cases; foreign stations, 5 cases.

1911.—Marines, Guantanamo Bay, Cuba, 11 cases; foreign stations, 16 cases.

1912.—Fourteen ships and eight shore stations reported one case each.

TYPHOID PROPHYLAXIS

Data are available for the 9-year period, 1925–33, with regard to the number of injections given in these years and the reactions and deaths resulting therefrom. These are shown in table V. There were 291,796 complete courses of 3 inoculations each, January 1, 1925, to December 31, 1933.

TABLE V.—*Typhoid prophylaxis, U. S. Navy, 1925–33*

	First-course inoculations	Second-course inoculations	Total
Number administered	620,135	275,545	895,680
Moderately severe reactions	7,751	1,022	8,773
Percent	1.25	0.37	0.98
Reactions requiring admission to sick list	1,825	300	2,125
Percent	0.29	0.11	0.24

Deaths resulted from antityphoid inoculations as follows: 1918, 2; 1923, 1; and 1926, 1.

During the 9-year period 895,680 inoculations were given. These inoculations caused 8,773 moderately severe reactions and in 2,125 instances the reaction was of sufficient severity to require admission to the sick list. There was one death during the period. There was one moderately severe reaction to each 102 injections and one admission to the sick list to each 421 injections. In other words, some type of reaction followed slightly more than 1 percent of the injections.

During the 10-year period, 1924–33, there were 81 cases of typhoid fever and of this number 73 have records of previous inoculations with typhoid vaccine. Of the remaining 8, one had received 1 injection; 4 had not been inoculated; and in 3 instances there is no record.

The time interval between inoculation and admission with typhoid fever is shown in table VI.

TABLE VI.—*Typhoid fever in persons previously inoculated, United States Navy, 1924–33. Interval between inoculation and admission*

Year	Up to 6 months	6 months to 1 year	1 to 2 years	2 to 3 years	3 to 4 years	4 years and up	Total
1924	2			1	1		4
1925		1		2		1	4
1926				1		1	2
1927		2	2	1	1	2	8
1928	1	1	3	2	1		8
1929	1	2		1	1		5
1930	1	3	1	3	2	1	11
1931		1	11	2	1	1	16
1932		1	2	3	3	1	10
1933		1	1	3			5
Total	5	12	20	19	10	7	73

It may be seen from this table that 5 individuals developed typhoid fever within 6 months of the completion of inoculation and slightly more than one-half the total cases occurred between 1 and 3 years after receiving 3 injections of typhoid vaccine. It is noted that there was an increase in the number of cases in 1930, 1931, and 1932 as compared to the years immediately preceding. The 11 cases in 1930 were reported from widely separated places under varying conditions of exposure. Of the 16 cases in 1931, 12 were reported in Marines in Nicaragua. An earthquake in March 1931 caused extensive damage to the waterworks in Managua, and for several months thereafter the water was grossly polluted. The water supply of the Marine camp was chlorinated, but there were numerous opportunities for exposure in the city. In 1932 there were 10 cases and of these 8 occurred in China.

SUMMARY

Typhoid prophylaxis was instituted in the Navy in 1912 and has been consistently practiced since that date. Specific prophylaxis has

consisted of the administration of typhoid vaccine to all persons in the naval service.

Coincident with the prophylaxis a marked reduction in the incidence of typhoid fever occurred. This reduction is believed to have been due in large measure to the immunization of the personnel. The incidence of typhoid fever has gradually declined in the registration area of the United States. Since 1900 this decline has been more gradual than that observed in the Navy and has at no time shown the marked drop comparable to that seen in the Navy in 1912.

In the 9-year period, 1925–33, 895,680 injections of typhoid vaccine were given. As a result of these injections there was 1 death and 10,898 reactions. Some type of reaction followed slightly more than 1 percent of the injections.

In the 10-year period, 1924–33, there were 73 cases of typhoid fever among persons who had received 3 or more injections of typhoid vaccine.

MODIFIED STOKES STRETCHER

By W. L. Mann, Captain, Medical Corps, United States Navy

Some years ago, Blackwood in a communication dated October 8, 1917 (Bureau of Medicine and Surgery file no. 124,704–40) recommended the substitution of tubular steel for solid metal rods in the construction of the Stokes stretcher. He also called attention to the fact that in riveting the cross rods, a hole about three-sixteenths of an inch in diameter was drilled in each rod, which destroyed two-thirds of its strength, and for this reason recommended that welding be substituted for riveting.

A few years later, the Medical Department of the United States Army, independently, developed a modification of the Stokes stretcher by using hollow steel pipes, and securing these in place by welding. The accompanying illustration, with description, is essentially the Army modification with a few changes, as worked up by the undersigned while in the planning section of the Bureau of Medicine and Surgery, Washington, D. C. At that time specifications were prepared for the Federal Specification Board, and an experimental model (see illustrations) was constructed by the Bureau of Construction and Repair.

The experimental model has the following advantages over the present Stokes stretcher.

Lighter in weight.—The Stokes litter, without fittings, is 34½ percent heavier in weight than the experimental type, without fittings. The wooden braces of latter weigh 3 pounds, and webbing straps 2 pounds.

	Experimental	Stokes
	Pounds	*Pounds*
Stripped	17¾	23¾
Fittings	5	1½
Complete	22¾	25¼

Increased rigidity is gained by.—(1) Welding transverse ribs to longitudinal supports instead of rivets, which weaken the supports 60 to 70 percent. (Actual tests on dynamotor show it to be 100 percent more rigid in a four-point suspension.)

(2) The substitution of tubular steel metal for solid construction.

(3) Rearrangement of transverse ribs so as to approximate maximum strain in a four-point suspension.

(4) Diagonal supports between ribs.

Promotes comfort of patients.—(1) Rounded perineal ridge.

(2) Rides more securely in ambulance; less tendency to rock, due to distance between skids being greater, 13½ inches compared to 8 inches.

(3) Supporting slats.

(4) More commodious, particularly in trunk compartment and section about knee.

Handling of litter facilitated.—(1) Litter can be grasped at almost any point along upper support, a very important feature in handling loaded litter down gangways, loading ambulances, etc. This point is particularly emphasized by Hospital Corps men.

(2) The configuration of the experimental type allows the stretcher to be secured, fore and aft, or aft to fore, to permanent fittings in motor or airplane ambulance, and litter carriers.

(3) It is, or should be, designed to nest more compactly.

(4) Better balanced.

Summarizing, the experimental type is.—(1) Lighter (34½ percent).

(2) More rigid (100 percent).

(3) More durable (300 percent, estimated).

(4) More easily handled.

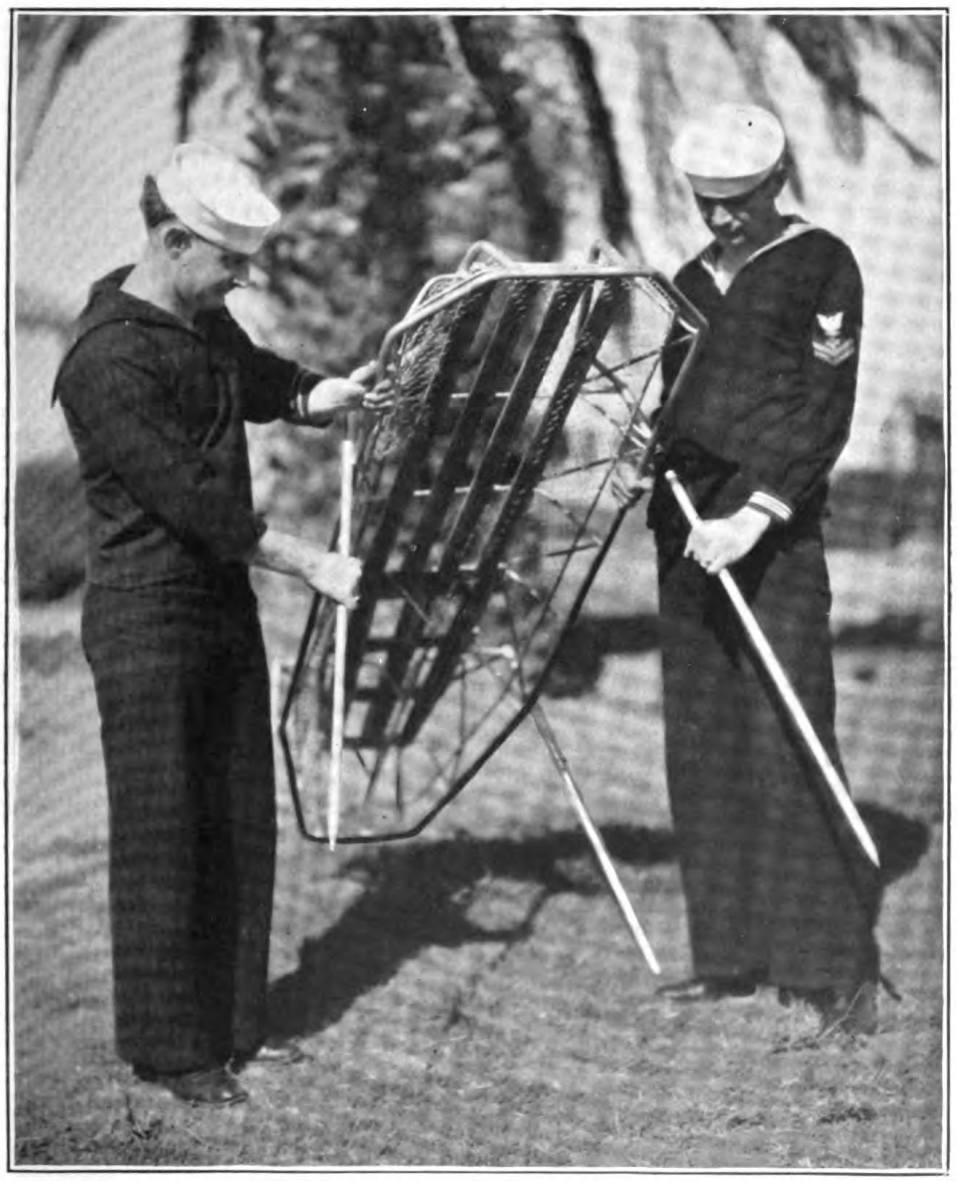
(5) More comfortable.

(6) More adaptable for securing to permanent fittings.

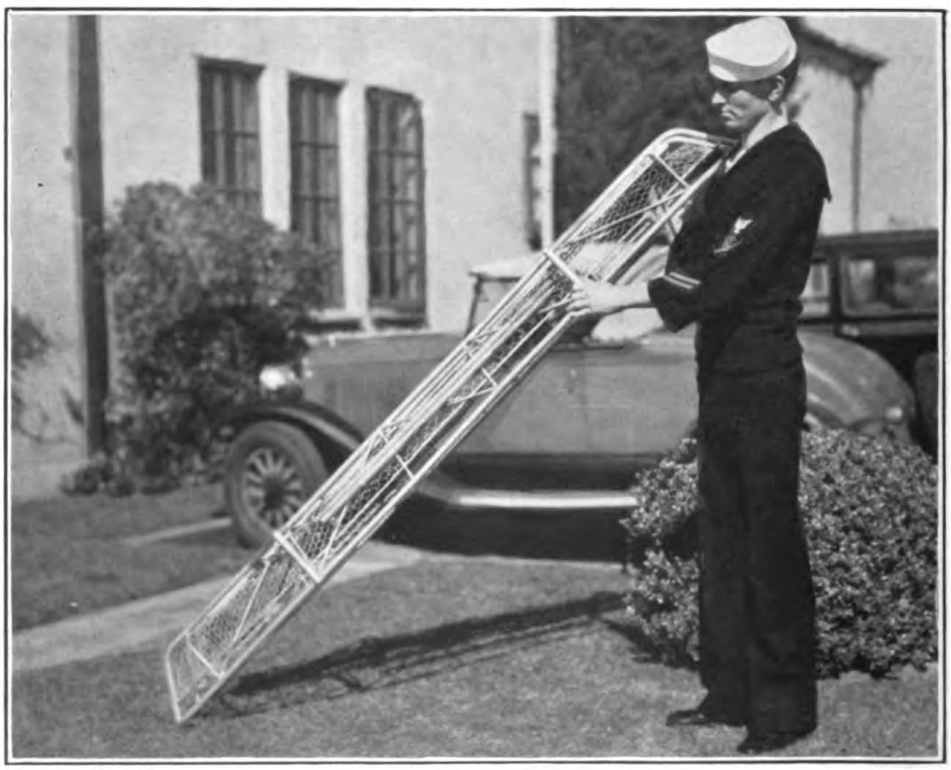
(7) Nests more readily.

(8) More economical (?).

Used as an improvised stretcher-operating table.—Following an aviation crash, and other accidents, there are occasions where a field operating table is urgently needed for such cases as ligation of arteries, immediate surgery, treatment of nontransportable patients, and other conditions.



INSERTING THE SUPPORTS USED TO CONVERT STRETCHER INTO A FIELD DRESSING TABLE.



SHOWING TWO SUPPORTS SECURED TO SIDES OF LITTER, FROM WHICH THEY CAN BE READILY DETACHED AND USED AS SUPPORTS FOR AN IMPROVISED LITTER-DRESSING TABLE.

U. S. Navy Stretcher. Experimental Model. Converted into an Improvised Field Dressing Table.

ILLUSTRATING POSSIBLE USE OF ONE OF THE SUPPORTS TO SERVE AS AN AXLE FOR A TWO WHEELED LITTER CARRIER—A VERY USEFUL FIELD EXPEDIENT.

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To serve this purpose the author has utilized the modified Stokes stretcher, experimental type, and has designed an improvised "stretcher-operating table", which consists of the inverted stretcher supported at a convenient height by four supports of tubular steel.

By the construction of two devices on each side of the stretcher (see illustrations) to receive the insertion of the pipe "legs", it is possible to use the inverted litter as a field dressing or operating table.

These fittings, weighing about 4 pounds, are devised as attachments so that they will be immediately available at all times.

GONOCOCCIC AND MENINGOCOCCIC ENDOCARDITIS, WITH REPORT OF THREE CASES

By C. W. Ross, Commander, and F. C. Greaves, Lieutenant, Medical Corps, United States Navy

Bacterial endocarditis, due to infection of the endocardium by organisms other than those causing acute rheumatic fever and syphilis, occurs as a grave complication to infection elsewhere in the body. An academic classification divides them into the acute and subacute varieties on a basis of their clinical manifestations. While it is not always possible to classify them according to etiology, such a classification is very desirable because the treatment and prognosis may be profoundly influenced by knowing the causative organism.

The etiological agent is usually one of the cocci. White quotes Thayer's table of 199 cases, with 138 autopsies in which the following organisms were found (1):

	Percent
Streptococcus	57
Pneumococcus	14
Staphylococcus aureus	13
Gonococcus	11
Influenza bacillus	4
Staphylococcus albus	1

White also reports occasional cases occurring in which the organisms were found to be the meningococcus, typhoid bacillus, enterococcus, para influenza bacillus, plague bacillus, and Brucella melitensis (2).

Two factors apparently are operative in the production of the endocardial lesions. First of all, there must be an active infectious process elsewhere in the body as the result of which a bacteremia occurs. The infection may be a pneumonia, a meningitis, a gonorrhea, a furunculosis, a cellulitis or any other infection that is able to overcome the resistance and pour viable organisms into the blood stream. The second factor is a previously damaged endocardium or one that has a congenital defect. A striking fact in these cases is the high percentage that gives a history of an earlier

acute rheumatic fever or the signs of a previously diagnosed organic cardiac lesion. On the other hand, the literature abounds in reports of proved cases of bacteremia with recovery, which gave no histories of previous cardiac damage. Cabot's case had arthritis and blood cultures positive for gonococcus, and recovered with no evidence of a cardiac lesion (3). Bruunsgaard and Thjötta reported a case which presented arthritis, purpura, and petechiae. The gonococcus was cultured from the blood, the spinal fluid and the petechiae with recovery of the patient and with no evidence of cardiac involvement (4). It is probably the exceptional case of bacteremia that develops an endocarditis on a normal endocardium.

Gonococcus endocarditis is a relatively rare form of the disease and, fortunately, it is an exceedingly rare complication of a gonorrheal urethritis. Hoffman and Taggart reviewed the literature for the 10-year period between 1922 and 1932 and concluded that during that time only eight cases could be considered authentic. They added one case of their own (5). Peters and horn reported two more in both of which they demonstrated gram negative diplococci in smears taken from the aortic valve vegetations (6). Meningococcus endocarditis is very rare. Rhodes reviewed the literature up to 1927, and found 11 cases, all of which were proved by bacteriological studies, and all of which resulted in death. He reported one additional case (7).

Three cases are reported below. In the first two, gonococcus endocarditis was diagnosed ante-mortem by positive blood cultures and confirmed post-mortem by bacteriological studies of the vegetations. In the third case, there is evidence, more or less presumptive, of a meningococcus endocarditis which recovered, but with a permanently damaged endocardium.

A 22-year-old seaman first-class was admitted to the sick list on board ship on January 13, with the diagnosis of gonococcus infection of the urethra. The diagnosis was changed, on January 19, to gonococcus infection, joint, because of an arthritis of the left shoulder. He was transferred to the hospital with the latter diagnosis, on February 3, at which time, he had a temperature of 103.6°, pulse 92, respiration 18. He complained of pain in the left shoulder and back, but there was no evidence of inflammation in those areas. There was no urethral discharge, but the meatus was found to be minute and the urine to be loaded with pus cells. A meatotomy was done to facilitate drainage.

The heart was somewhat enlarged, with an aortic murmur, which obliterated the second sound. The blood pressure was 120/40. The spleen was markedly enlarged. R. b. c. 4,110,000, with 90 percent hemoglobin. W. b. c. 12,000, polymorphonuclears 73 percent, lymphocytes 24 percent, monocytes 2 percent, eosinophiles 1 percent. The blood Kahn was negative.

His condition remained stationary during the next week, with daily blood cultures being reported negative. Circulatory embarrassment was noticeable on February 13. The next day, the blood culture was reported positive for diplococci resembling the gonococcus in morphology and staining characteristics. Signs of pneumonia appeared in the right lung, and on the 18th, the char-

acter of the cardiac murmur changed. The blowing quality of the murmur previously noted became a muffled, roaring sound. Circulatory failure increased in severity and he died on February 20. As soon as the positive blood culture was reported, he was given a transfusion of whole blood from a donor recently recovered from acute gonorrhea. He received three such transfusions.

At autopsy, the heart was enormously dilated. The posterior aortic cusp was destroyed and presented a cauliflower vegetation measuring 1½ cm in diameter. A few small nodular vegetations were present on the mitral valve. Smears made from these vegetations revealed gram negative diplococci resembling gonococci. A pneumonic process was present in the right middle and lower lobes. The only evidence of embolism was the presence of one small, cortical infarct in the right kidney.

In this man's history, no mention was made of the presence or absence of a possible acute rheumatic fever in childhood.

A 29-year-old fireman second class was admitted to the hospital April 6, 1934, with the diagnosis of catarrhal fever, acute, complaining of headache, fever, malaise, and backache, which had begun the day before admission. Two days before admission, a profuse urethral discharge appeared and he admitted a venereal exposure within the week. He had 9 years' service, his health had always been excellent, and he denied all previous venereal disease. He described an illness in childhood which consisted of high fever and swollen joints, and which lasted 2 or 3 weeks.

His temperature was 102, pulse 114, r. b. c. 4,350,000, hemoglobin 85 percent, w. b. c. 9,100. Mature forms 58 percent, band forms 6 percent, lymphocytes 34 percent, monocytes 1 percent, and eosinophiles 1 percent. The urethral smears were positive for gram negative, intracellular diplococci. He improved under routine treatment and the discharge lessened and subsided under conservative treatment. He did not maintain improvement as he should, but at the end of 2 weeks, he was running an afternoon temperature of about 2° elevation and, while he had no complaints, it was evident that he was not doing well. On May 9, there was a slight chill, a temperature of 101, pulse 100, and a feeling of discomfort in the right lower quadrant. W. b. c. numbered 12,000 and r. b. c. were found in urine. These symptoms continued for the next few days and some tenderness and rigidity developed over the appendix. He submitted to an appendectomy, but the appendix was found to be normal.

The symptoms continued and, for the first time on June 6, a soft, blowing, systolic murmur was heard at the apex. Fleeting joint pains appeared. Several blood cultures were negative using ordinary blood culture media, but on June 8, using a modified Swartz media, gram negative diplococci were found. Stained blood smears showed an occasional large endothelial cell. The diagnosis was changed to endocarditis and the abdominal pain was interpreted as being embolic in origin. Blood transfusions from immunized donors were given. The course of his illness was not influenced in the least by this treatment. He steadily declined and circulatory distress and failure appeared. The cardiac murmur changed in character and quality several times, pulmonary edema supervened and he died June 15.

At autopsy, no petechiae were present in the skin. Furthermore, during life, repeated inspections of the skin had failed to reveal petechiae. The heart weighed 470 grams. The cusps of the mitral valve were partially destroyed and covered by friable, yellowish, fungoid vegetations. There was fibrosis and distortion of the portions of the cusps not involved in the fresh vegetations. Both lungs were edematous. The spleen weighed 340 grams and contained a recent infarct involving a quarter of the organ. The kidneys showed both recent and healed infarcts. The prostate appeared normal grossly.

Gram negative diplococci were demonstrated both in direct smears of the vegetation and in differential histological sections of the valves. Microscopic studies of the prostate, particularly in the tissues about the prostatic urethra, showed an extensive subacute inflammation, although no gram negative diplococci could be demonstrated.

In these two cases it was proved that the gonoccus was the causative agent. The medium used in obtaining the blood cultures was Swartz's medium, modified by omitting the agar. 15 cc of blood was used for inoculation, care being taken to have the medium at body temperature when the blood was introduced, after which the flask was tightly stoppered with a rubber cork. Twenty-four hours later there was a scanty growth of gram negative diplococci. The growth increased during the next 24 hours, after which it rapidly died out. All attempts to obtain subcultures met with failure.

The last case to be reported is one in which the etiology is open to question, but it is believed that there are certain factors that warrant its being reported with these other cases of known etiology.

A 19-year-old seaman second class went on the sick list April 1, with symptoms resembling catarrhal fever. Several days later "red spots" appeared on the palms, soles, and legs, and about the same time, dyspnoea was noted. There was no arthritis. He was transferred to the hospital on April 23. He gave a history of swollen joints, associated with fever and sore throat, in childhood, from which he recovered completely, so far as he knew.

On admission to the hospital, it was noted that there were numerous small pigmented, crusted lesions on the palms, soles, and in the skin of the legs, as well as several deeper areas of tender induration in the palms and soles. The joints were normal. The heart was normal in size. There was a high pitched, systolic murmur heard best in the 4th left interspace. The genitourinary system was negative, he denied all venereal disease, and the blood Kahn was negative. Temperature 100, w. b. c. 12,600. During the next week his temperature went to 100 every day, and on the 29th, the cardiac murmur changed, becoming rougher and lower in pitch, and in addition, a soft, blowing, diastolic murmur appeared. He complained of dyspnoea and precordial pain. Repeated blood cultures remained sterile. On May 12, a severe headache developed and definite signs of meningeal irritation appeared. The spinal fluid was cloudy and contained gram negative diplococci, which were identified as meningococci. Specific serum therapy caused immediate improvement in the meningeal symptoms, the temperature returned to normal and remained so, but the cardiac symptoms continued for several days. Compensation was gradually established, and at the end of 2 months, he was able to be up and about without dyspnoea, but the slightest exertion out of the ordinary made him breathless. He was given a medical discharge and left the hospital on September 8 at his own request, with a moderately enlarged heart, a systolic murmur, and very little cardiac reserve.

The meningococcus was not definitely shown to be the cause of this endocarditis, but there is rather strong presumptive evidence that it was. There was a fairly definite history of rheumatic fever in childhood. It is a recognized fact that a meningococcic bacteremia may exist for weeks or months before meningeal signs ap-

pear. Herrick has maintained for years that cerebrospinal fever is primarily a bacteremia and many case reports bear this out. Before the appearance of the meningeal signs in this patient, he was running a daily temperature of 100 with a cardiac disability that was gradually increasing in severity. As soon as the specific therapy was begun, the temperature became normal, and the cardiac condition became stationary, and then slowly improved as compensation occurred. It is not unreasonable to assume that the antimeningococcic serum exerted a specific action on any meningococci localized in the endocardium and halted the progress of the endocardial lesion and permitted recovery within the limits of the damaged heart to compensate.

CONCLUSIONS

Three cases of bacterial endocarditis are reported. In two, the gonococcus was found to be the etiological agent, being demonstrated in the blood streams before death and in the endocardial vegetations at autopsy. The third case is one of a possible meningococcic endocarditis that recovered with a badly damaged endocardium.

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IVY POISONING (Rhus Dermatitis)

By W. R. Manlove, Jr., Lieutenant, Medical Corps, United States Navy

Ivy poisoning is a form of dermatitis venenata due to exposure to poison ivy (rhus toxicodendron).

The excitant is an oily compound called toxicodendrol belonging to the phenol group. This substance is believed to be the same as that in other plants causing a similar dermatitis, such as poison-oak and poison sumac. As little as 0.001 mgm of toxicodendrol may produce dermatitis.

The poison-ivy plant.—The recognition of the plant is the important means of the prevention of ivy poisoning. The following descriptions of the plant and its related shrubs are abstracted from United States Department of Agriculture, Farmers' Bulletin No. 1166, by C. V. Grant and A. A. Hansen, revised by Coville and Talbot.

Poison-ivy grows as a woody vine, trailing shrub, or erect bush. It grows anywhere; in the woods or in the open, in moist or dry soil. It climbs trees and posts. It may be entwined so closely around a dead sapling that the poison-ivy leaves are mistaken for those of the tree.

The plant is recognized by its leaves which are divided into three-leaflets; and by its white mistletoe berries, known as drupes; the latter remain on the plants into winter after the leaves have fallen. All of these plants do not bear the drupes and it must be recognized by the leaves.

The leaves are from 1 to 4 inches long. The end leaflet of the triad has a longer stem than the two opposite leaflets, which, as the leaves are broad, though pointed, preserves the symmetry of the arrangement.

The mature leaves are dark green on the upper surface and lighter underneath. The crinkly young leaves are red when they first unfold, becoming green later. In autumn they turn to beautiful shades of scarlet and orange. The innocent sometimes pluck these leaves and bear them off to their sorrow. Years ago the leaf pressing fad occasioned innumerable attacks of dermatitis.

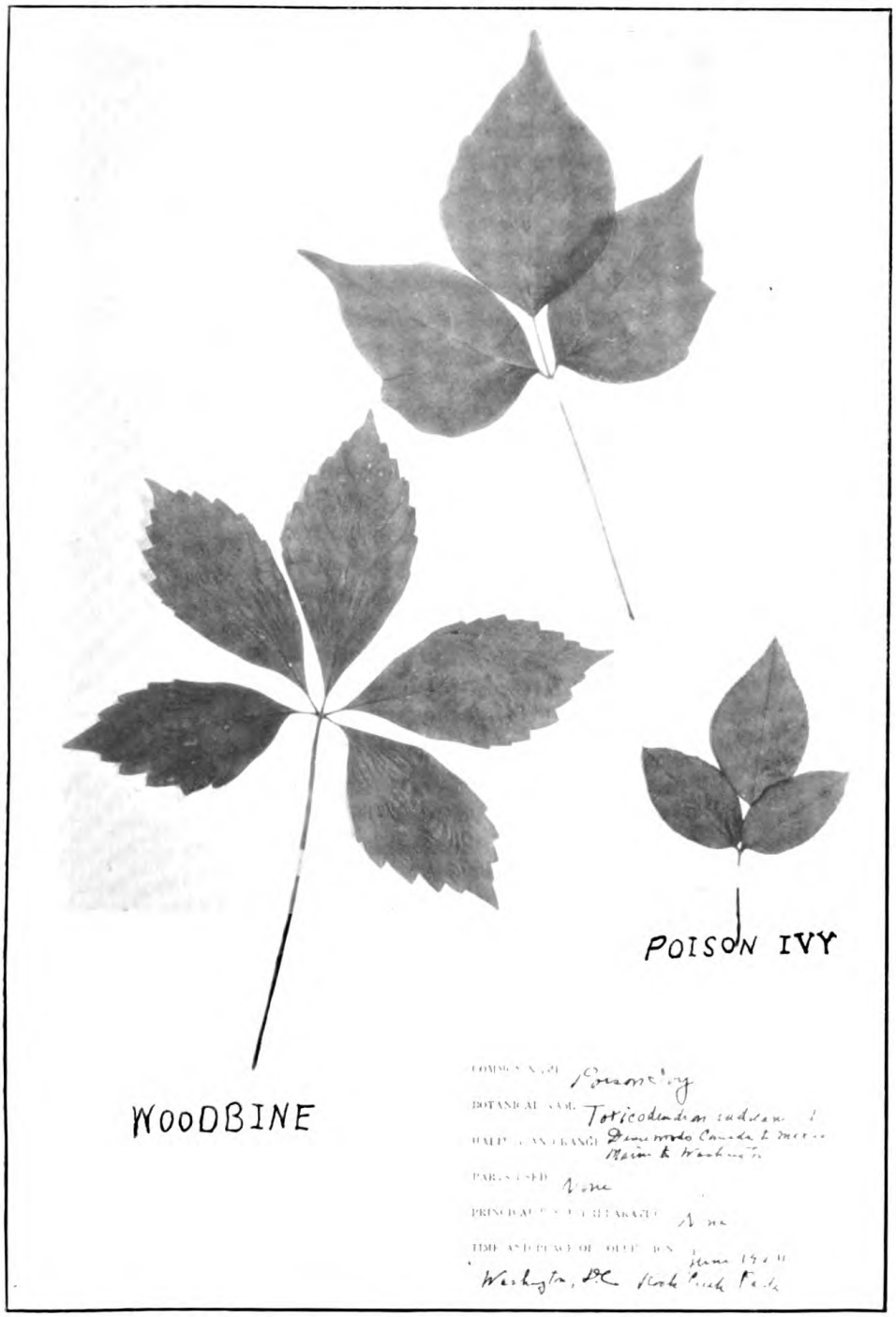
Some varieties show a deeply notched or indented leaf margin, causing the plant to be called poison-oak. The term is applied especially to those types that grow as independent bushes.

More properly the poison-oak is the erect bush of the Pacific coast region whose leaf is more oak like. However, all of these plants show the three-leafed form.

The old adage runs—"Leaflets three, let it be"—makes a safe rule for those of us who perhaps have neglected "How to know the wild flowers."

Poison sumac is a low tree which grows only on wet land, usually in swampy thickets, that are hard to get through, and the contact is therefore less frequent. However, it may be recognized by its leaves which are 7 to 14 inches long consisting of a stem with a row of 7 to 13 leaflets opposite each other in pairs and a single long-stalked leaflet at the end. The seasonal color changes are the same as those of poison-ivy. It also bears clustered white berries in season.

Etiology.—It is well known that there is a great individual variation in susceptibility to poison-ivy dermatitis. This phenomenon brings up the question of its relationship to allergy. In the looser



COMPARISON BETWEEN WOODBINE AND POISON IVY.

application of the term allergy to include all hypersensitive reactions, contact dermatitis must be included. But as sensitivity to rhus can be induced in about 70 percent of all people, a hereditary influence cannot be ascribed, according to Coca. And he states that this dermatitis is not an atopy—such as hay fever, asthma, eczema—which, according to Cook, shows hereditary influence and is present in about 7 percent of the population.

At present time the known excitants of atopic phenomena are proteins, and toxicodendrol and other excitants of contact dermatitis are of different groups, including oils and simpler compounds.

Babies are not susceptible to poison ivy until they are sensitized by sufficient contact. Heinbecker on the Putnam expedition of 1927 found no instances of hypersensitiveness to rhus among the Eskimos. However, the American Indian was found by Diebert to be as sensitive proportionally as the whites.

All parts of the poison ivy contain the irritating principle, the leaf especially.

The attack of dermatitis is usually caused by actual contact with the plant. Older writings on the subject speak of attacks being precipitated by passing near or to the leeward of the vines.

Since the discovery of the oily nonvolatile principle, this has been denied or doubted. However, in highly susceptible or sensitized individuals it occurs and has been attested to by reliable observers not excepting the writer.

Coca states that contact with the oleoresin of poison ivy is had by all people living in counties where it grows, on account of the presence of the substance in the air during the summer and that its concentration in the air then is known to be sufficient to precipitate an attack in highly susceptible persons when they pass near the vines, but without touching them.

Symptoms and diagnosis.—The period of incubation, after contact in the susceptible, depends on the degree of sensitivity and the amount and character of the contact. It is usually a few hours but may be delayed for days. Redness and erythema appears, usually first on the face and hands. Shortly, fine clear vesicles, which are closely grouped, are observed in the thinner areas, such as the inner surfaces of the fingers. Other areas of the skin become affected, even those where there has been no direct contact. The eyelids and surrounding tissues are frequently greatly swollen. Probably by indirect contact the genitals become edematous and the rash has a predilection for the looser and thinner skin.

New lesions appear by extension and in independent areas. Mucous membranes are affected. Large vesicles may develop, even bullae.

The attack may be very severe and deaths have been reported. There is no question of its disabling effect. As the dermatitis subsides the skin is left dry and finely crusted. Usually, even in severe cases, a few days after the subsidence of the acute inflammation, there is a complete clearing of the skin.

The diagnosis is made by the history of contact and it is usually readily obtained.

Prophylaxis.—1. Recognition and avoidance of the plant.

2. Protection of exposed skin by gloves, high shoes, etc.

3. Prompt bathing preferably by shower after an excursion in the woods.

4. Desensitization by injection of the various commercial preparations of the extract. Conflicting and doubtful reports are published as to the efficacy of this method. In new and nonofficial remedies these extracts are listed, only as recommended in the treatment of the dermatitis itself. As the active principle is probably a nonantigen, the response to injection is not so dependable.

The extract or tincture by mouth in increasing dosage is probably useless as a desensitization method. Goodman states that the colonists tried chewing the leaves as a preventative and it is probably still practiced in some communities.

Rhus extract was formerly used in the treatment of certain forms of paralysis and was so listed and described in the pharmacopeias during the nineteenth century and its availability has probably occasioned its later use.

5. Eradication of the plants. Preferably by nonsensitive workers or those well protected by costume.

Treatment.—As the active agent is fata and alcohol soluble, the irritant may be largely removed if immediately after exposure the areas are washed with soap and water. Alcohol or ether, used on swabs, being careful to clean the irritated areas from the periphery toward the center, is a more effective method.

Two readily available agents are recommended for the treatment of the lesions, their usefulness predicated by experimental work of J. B. McNair.

1. Potassium permanganate in a 5 percent solution in water applied locally by cotton swabs or cloths; frequent applications should be made, or if a dressing is used, frequent change of dressings.

2. Chloride of iron, a 5 percent solution in equal parts of alcohol and water can be used in the same way.

This is also recommended to be applied to exposed areas just before entering places where the poison bearing shrubs are prevalent.

It is important to remove the precipitate and repeat the application when using any of the metallic salts as an antidote to the poison, as the formed precipitate may decompose releasing the poison again.

In the event that the chloride of iron salt cannot be obtained locally, tincture of ferric chloride, which is a 13 percent alcoholic solution mixed with 1½ parts of water would give an equivalent mixture for application.

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A COMPARATIVE STUDY OF THE MEASUREMENT OF THE SPEED OF ADJUSTMENT OF THE EYE FOR NEAR AND FAR VISION [1]

By C. J. Robertson, Lieutenant Commander, Medical Corps, United States Navy

In the July 1934 issue of the Naval Bulletin we had an article entitled "Measurement of the Speed of Adjustment of the Eye to Near and Far Vision", which dealt with a few tests which we had obtained on the multiple exposure tachistoscope, an apparatus developed by Ferree and Rand of the Wilmer Ophthalmological Institute of Johns Hopkins University, Baltimore, Md.

The article was only considered as a preliminary study and was written to stimulate thought and criticism along that line which would be of value to future work.

We realized at the time of writing the article that the tests made and available were far too few to allow us to draw conclusions of any great moment, but we felt entitled to make the statement that more work should be done in order to make a positive or negative report as to the value of such measurements of speed of adjustment of the eye for near and far vision.

Our conclusions at that time were:

1. That speed in the adjustment of the eye for different distances is essential in aviation, and that a measure of such speed can be made.

2. That a standard of measurement of such speed can be established if sufficient tests are made with men of various ages and various ocular conditions.

[1] Received for publication, Dec. 15, 1934.

3. That such a standard, when established, should be incorporated in the visual requirements of aviation.

4. That such tests disclose the pathological eye and may be of use in the preliminary examinations for the Naval Academy.

Since that time, through the courtesy and aid of Rear Admiral P. S. Rossiter (M. C.), United States Navy, Surgeon General of the Navy, and the splendid encouragement and cooperation of Capt. C. M. Oman (M. C.), United States Navy, commanding officer, United States naval hospital, Annapolis, Md., as well as many others too numerous to enumerate and give well merited credit, we have been able to perform 353 tests, 247 on pilots, ages 22 to 44, and 106 on nonpilots, ages 18 to 35.

Pharmacist's Mate T. O. Roberts was ordered to the naval hospital at Annapolis for training in handling the tachistoscope. After he was sufficiently well versed in the work, he was transferred to the air station at Hampton Roads, Va., where, with the very efficient cooperation of the flight surgeons of the fleet and the air station, the tests were continued.

The work was done under circumstances which were trying for the operator as well as those tested. It is keenly appreciated that the time which the pilots took from their work to be tested was an added labor to an already well-filled calendar.

All testing was done under what was considered well controlled conditions. The lighting was continuously checked with the Macbeth illuminometer so that all tests were done under an illumination of 5-foot candles. Also, a tachometer was used to check the motor; thus, at all times the lighting and the speed were controlled, and all tests were done under known circumstances.

A printed form was evolved and used with each test containing the data considered essential for future evaluation. The particular factors of value being, the time of day, the age, the number of flight hours, years in aviation, the number of crashes, refractive errors, visual acuity, accommodation, depth perception, and phorometer readings. For convenience this chart is called "Form A". Upon the completion of the test the form A has been forwarded to me at the Annapolis hospital, where the findings are transferred to form B. On this form the findings of the test are placed in two main divisions, pilot and nonpilot, and then subdivided into the various ages.

FORM A

TACHISTOSCOPIC EXAMINATION

Place _____ Date _____ Time _____
Name _____ Rank _____ Age _____
Occupation _____
Flight hours _____ Years in aviation _____ No. of crashes _____
Previous visual waivers _____
Refraction _____
Visual acuity, far: O. D. _____ O. S. _____ Near: O. D. _____ O. S. _____
Accommodation: O. D. _____ O. S. _____ P. D. _____ Pc B. _____ Angle _____
Depth perception _____
Phorometer: P. D. _____ Hypo _____
6 M. Eso. _____ Exo. _____ 33 C. M. Eso. _____ Exo. _____
Degree values:
N. (33 C. M.) _____ N. to F. (6 M.) _____ F. to N. (30 C. M.) _____ Complete _____
Time value:
N. (33 C. M.) _____ N. to F. (6 M.) _____ F. to N. (30 C. M.) _____ Complete_____
Illumination: Foot candles _____
Remarks: _____

FORM B

Number	Observer	Age	Occupation	Flight hours	Number of crashes	Years in aviation	Visual acuity		Refraction	Accommodation	Angle	Phorometer at 6 M	Phorometer at 33 CM	Depth perception	Time on tachistoscope				Illumination	Time of day	Length of time for test	Remarks
							Far	Near							Near	Near to far	Far to near	Complete				
1																						
2																						
3																						
4																						
5																						
6																						

The division of the two main groups is a division of what could be called selected and nonselected people. Pilots are supposedly carefully selected physically upon entry into aviation, with particular emphasis as to all eye conditions such as visual acuity, accommodation, depth perception, refractive error, muscle imbalance, etc. The nonpilot group are a nonselected group and do not have the annual check-up on their visual conditions.

The data submitted on each test are such that a finer evaluation can be made than just a comparison of selected or nonselected groups, but comparison as to age groups in a particular classification can be made. For example, with pilots we can compare the ages between 20 to 30 with 30 to 40, those from 20 to 30 with 40 to 50, or 30 to 40 and 40 to 50. Then, again, comparison can be made with men with a known refractive error and those with the so-called " normal " eye regardless of age. Consideration can be given to people with various muscle imbalances, variation in depth perception, etc.

It is considered that the exophorics should have more trouble in converging, and then take a longer time in passing from far to near; while those with an esophoria should have more trouble in diverging, and hence take longer to pass from near to far.

At the beginning of this work we had felt the evaluation of the results would be so simple that with a multiplicity of tests we could immediately prove the worth of the work, but we now find that a simple evaluation of age alone is insufficient for an intimate knowledge of the tachistoscope in its relationship to aviation.

It is not felt in this article that a description of the machine or method of use is again necessary, as a complete description was given in the July 1934 Naval Medical Bulletin. Suffice it to say that the tachistoscope is a machine so mechanically adjusted that the separately rotatable letter " E ", at 33 centimeters, 6 meters and 33 centimeters, is cut by rotating disks which make one full revolution in 4 seconds. The degree values of speed thus found is transposed into seconds or fractions of seconds, for near seeing, for changing from near to far, and from far back again to near. This gives the speed of adjustment of the eye for different distances.

In our original article we stated that the tests were far too few at that time to form any definite conclusions. We still feel that we have too few tests to base evidence for standardization. However, we do feel our original premise, that speed of vision, or better of adjustment, plays an important part in the flying ability of an aviator, is upheld and in this article we hope to demonstrate the truth of that premise.

It should be understood that the pilots are a selected group. All pilots upon admission to aviation are given a rigid, physical examination and an annual examination just as rigid, excluding refraction. In some cases the examination is semiannual. The visual requirements are briefly as follows:

1. Minumal vision 20/20 each eye.

2. An average depth perception of more than 25 millimeters in five readings disqualifies.

3. *Phorometer findings at 6 meters.*—Esophoria of more than 4 diopters is a disqualifying factor if associated with less than 4

diopters of prism divergence, or if associated with diplopia on the tangent curtain, or associated with the amount of accommodation near the lower limits, or if associated with an amount of hyperphoria near the disqualifying limit.

Exophoria of more than 10 diopters is a disqualifying factor.

Exophoria of more than 2 diopters is a disqualifying factor if associated with an angle of convergence near the disqualifying limit, or if associated with diplopia in the lateral position on the tangent curtain.

Exophoria of more than 5 diopters is a disqualifying factor.

Hypnerophoria of more than 0.75 diopters disqualifies without further evidence except in cases of qualified pilots when 1 diopter will be allowed.

4. *Phorometer at 33 centimeters.*—Exophoria of 4 diopters may be considered normal. Any considerable variation from this should be considered in connection with other associated tests.

Exophoria of 12$^{\triangle}$ disqualified.

5. *Prism divergence.*—Prism divergence of more than 9 diopters disqualifies if associated with an angle of convergence near the disqualifying limit. If less than 4 diopters of prism divergence is found associated with 4 diopters of exophoria at 6 meters. Prism divergence of more than 15 or less than 2 diopters is disqualifying.

6. *Associated parallel movements.*—Disqualify if under or over action of any of the extrinsic ocular muscles produce diplopia except in extreme positions, where a small separation of the images may be disregarded.

7. Accommodation is normal if it lies between limits 3 diopters above and below the mean allowed for the examinee's age.

8. *Angle of convergence.*—An angle of convergence less than 40° disqualifies.

9. *Refraction.*—The examinee is disqualified if he cannot read 20/20 in each eye without more than 1 diopter of correction, either hyperopic, myopic, or astigmatic. A reasonable allowance may be made in the case of qualified fliers.

These requirements are given to demonstrate the selectivity of the aviators when used in comparison to the nonaviators whose requirements are in brief 20/20 vision in each eye with no gross errors and pathological conditions upon admission to the service.

It is felt that a short word should be said about the use and the time element in the testing on the tachistoscope. Although the present machine is built for experimental purposes only, and as such does not have the refinements which a perfected machine would have, it can be easily learned and is not difficult to run or understand. The time element is noted as follows:

The average time consumed for all of the 353 tests, which includes the time used in explaining the work of the examinee to the final end of the test with the result, was 26 minutes. The average time for pilots from age 22 to 29 was 23 minutes. The average time for pilots from age 30 to 39 was 24 minutes, and the average time for nonpilots from age 18 to 35 was 31 minutes. It is believed that 25 minutes will be the average time per test. As was noted in the original article to some extent this is a test for immediate memory but this is remedied by repeated trials where the examinee is slow. It has been noted in the tests that those found to have visual errors of any type and amount are much slower in their performance on the tachistoscope.

There would appear to be some significance in the above findings. The aviators as a selected group (age 22 to 30 and 30 to 44) have a time of 7 and 6 minutes less than the nonselected men, age 18 to 35.

TABLE I.—*Showing the distribution of complete times: Near to far and back to near, for 249 pilots, separated into decade groups of age*

Age	Complete time in seconds. Near to far and far to near							
	0.095 to 1.149	1.15 to 1.349	1.35 to 1.549	1.55 to 1.749	1.75 to 1.949	Above 2	Average	Total
15–19.9								
20–24.9	2	12	3				1.244	17
25–29.9	20	58	14	1			1.218	93
30–34.9	6	49	38	4	2		1.348	99
35–39.9	2	12	14	6			1.396	34
40–44.9			1	4	1		1.639	6
Total	30	131	70	15	3			249

TABLE II.—*Showing the distribution of complete time: Near to far and back to near for 105 nonpilots, separated into decade groups of age*

Age	Complete time in seconds: Near to far and return to near							
	0.095 to 1.149	1.15 to 1.349	1.35 to 1.549	1.55 to 1.749	1.75 to 1.949	Above 2	Average	Total
15–19.9	2	8	3	2	1		1.357	16
20–24.9	3	33	10	3			1.288	49
25–29.9	4	10	10		1		1.326	25
30–34.9	2	4	6	2			1.341	14
35–39.9						1	2.13	1
40–44.9								
Total	11	55	29	7	2	1		105

The results from 249 pilots, ages 22 to 44, and 105 nonpilots, ages 18 to 35, are summarized in tables I and II. In these tables the subjects were separated into decade groups and the number of cases whose speed for the complete time near to far and back to near recorded for time ranges of 1.5 seconds; from 0.095 to 1.95 seconds. Cases following above 2 seconds were grouped together. In these

tables, the rows from left to right show the distribution of time value for each decade group; the columns from top to bottom show the distribution of ages for each group of time values. The next to the last column gives the average time for each age group and the final column, the total number of cases.

The results show a shift of the distributions from low to high time values with age above 30 years. They also show that there are very few experienced pilots whose time to perform the test is greater than 1.55 seconds. In comparing the results shown in the two tables it should be borne in mind that the pilots are as a class an older group than the nonpilots.

In table I there are a number of test cases of interest. In the group of pilots, age 20 to 24.9, there are three cases with a complete time, near to far and return to near, between 1.35 and 1.549 seconds. The data in each case is as follows:

CASE I.—Age 22 years, 470 hours in the air, 4 years in aviation. Visual acuity: v. o. d. 20/20, v. o. s. 20/15. Refraction: v. o. d. plus 0.50 plus 0.25 axis 90°, v. o. s. plus 0.50 plus 0.50 axis 90°. Time on the tachistoscope: Near 0.033 second, near to far 0.957 second, far to near 0.407 second, far to near and return to near 1.364. It is considered that in that age group that over 0.9 second for near to far and over 1.35 seconds for the complete near to far and return is too long. The inequality of visual acuity and the astigmatic error should be noted. It took 30 minutes to complete the test.

CASE II.—Age 24, 925 hours in the air, 6 years in aviation. Visual acuity: 20/20 in both eyes. Refraction: Plus 0.75 in both eyes. Phorometer findings at 6 meters: Prism divergence, 4 diopters; left hyperphoria, 0.7 diopters; esophoria, 3 diopters. At 33 centimeters: Exophoria, 13 diopters. Depth perception, 18. Tachistoscope findings: Near 0.033 second, near to far 0.847 second, far to near 0.550 second, and near to far and return to near 1.397 seconds. It took 30 minutes to complete the test. It is considered that over 1.35 seconds at that age is too long. Exophoria of 13 diopters at 33 cm and left hyperphoria 0.7 diopters is present.

CASE III.—Age 24, 600 hours in the air, 4 years in aviation. One crash. Visual acuity: V. o. d. 20/20 plus 2, v. o. s. 20/15. Refraction: V. o. d. plus 0.75, v. o. s. plus 0.50. Accommodation, 8.5, both eyes. Angles of convergence 59. Phorometer findings at 6 meters: Prism divergence 4 diopters, right hyperphoria 0.2 diopters, esophoria 3 diopters; at 33 cm, exophoria 4 diopters. The tachistoscope findings are: Near 0.044 second, near to far, 0.836 second, and near to far and return to near 1.386 seconds. Over 1.35 seconds is considered too long for this age group. Inequality of visual acuity and hyperphoria should be noted.

In the pilots, age 25 to 29.9, we find one pilot with speed of adjustments as follows: Near 0.044 second, near to far 1.166 seconds, far to near 0.44 seconds, and near to far and return 1.606 seconds. It is considered that in this group the speed of near to far and in near to far and return to near is considered far too long. Case findings are as follows: 27 years old, 1,000 hours in the air. Three years in aviation. One crash. Visual acuity: V. o. d. 20/15, v. o. s. 20/20. Ac-

commodation, 6.5 diopters. Angle of convergence 47. Phorometer findings at 6 meters: Prism divergence 2 diopters; at 33 cm, exophoria 6 diopters. Forty-five minutes to accomplish the test. Note the inequality of visual acuity. Accommodation at the low limit for the age of the pilot, and angle of convergence in the low limits.

It should be noted here that there were fourteen cases, ages 25 to 29.9, with a complete time, near to far and return to near, between 1.35 and 1.549 seconds. Space is too limited to consider all these cases, but they are considered within a doubtful period of proper speed of adjustment.

In the age groups of 30 to 34.9 we find 4 pilots with a complete speed, near to far and return, between 1.55 and 1.749 seconds, and 2 between 1.75 and 1.949 seconds.

Case I.—Age 32, 1,700 hours in the air and 8 years in aviation. Showed no anomalous eye condition other than the slowness in speed of adjustment, near 0.044 second, near to far 1.059 seconds, far to near 0.077 second and near to far and return 1.829 seconds.

Case II.—Age 33, 1,700 air hours, 15 years in aviation. Visual acuity: V. o. d. 20/30 plus three, v. o. s. 20/15. Refraction: V. o. d. plus 25 axis 90°, v. o. s. plus 25 axis 90°. Accommodation 7.5 in both eyes. Angle of convergence 70. Phorometer findings at 6 meters: Prism divergence 9 diopters; at 33 cm, exophoria 3 diopters. Tachistoscope findings: Near 0.055 second, near to far 1.155 seconds, far to near 0.550 second, complete, near to far and return, 1.705 seconds. Thirty minutes was taken to accomplish the test. The visual acuity inequality, refractive findings, and prism divergence should be noted.

Case III.—Age 33, 2,500 air hours, 7 years in aviation. Visual acuity, 20/15 in each eye. Accommodation, 5.5Δ in each eye. Angle of convergence, 76. Phorometer findings: Prism divergence, 9 diopters; right hyperphoria, 0.1 diopter; at 33 cm, exophoria, 16 diopters. Depth perception, 16. Tachistoscope findings: Near, 0.044 second; near to far, 1.059 seconds; far to near, 0.405 second; and near to far and return, 1.554 seconds.

It should be noted here that there is an exophoria of 16 diopters at 33 cm.

Case IV.—Age 33. One crash. Visual acuity, 20/15 in each eye. Accommodation, 5 diopters in each eye. Angle of convergence, 71. Phorometer findings at 6 meters: Prism divergence, 4 diopters; exophoria, 1 diopter; at 33 cm, exophoria, 8 diopters. Depth perception, 17. Phorometer findings: Near, 0.077 second; near to far, 1.188 seconds; far to near, 0.495 second; and near to far and return, 1.683 seconds. It took 25 minutes to complete the test. The accommodation at the low limit of 5 diopters and an exophoria of 8 diopters at 33 cm should be noted in this case.

Case V.—Age 32, 1,700 air hours, 8 years' aviation service. Visual acuity, 20/15 in each eye. Accommodation, 7 diopters in each eye. Angle of convergence, 73. Phorometer readings at 6 meters: Prism divergence, 6 diopters; left hyperphoria, 0.4 diopter; at 33 cm, exophoria, 3 diopters. Depth perception, 15. Tachistoscope findings: Near, 0.044 second; near to far, 1.059 seconds; far to near, 0.770; near to far and return, 1.829 seconds. No outstanding condition noted other than slow speed of adjustment.

CASE VI.—Age 32, 2,700 air hours, 11 years in aviation. Visual acuity, 20/20 each eye. Refraction, astigmatic error in each eye. Accommodation, 6.5 diopters. Angle of convergence, 56. Phorometer readings at 6 meters: Esophoria, 1 diopter; at 33 cm, exophoria, 2 diopters. Depth perception, 10. Tachistoscope readings: Near, 0.044 second; near to far, 0.671 second; far to near, 1.265 seconds; near to far and return, 1.936 seconds. Forty-five minutes to complete the test. The astigmatic error should be noted in this case.

In the age group 35 to 39.9 we find 6 cases in the time between 1.55 and 1.749 seconds.

CASE I.—Age 35, 2,800 air hours, 8 years in aviation. Visual acuity; 20/20 in each eye. Accommodation, v. o. d. 5.5 diopters and v. o. s. 6.0 diopters. Angle of convergence 54. Phorometer findings at 6 meters: Prism divergence 8 diopters, exophoria 1 diopter. At 33 cm, exophoria of 16 diopters. Depth perception 15. Tachistoscope findings; near 0.033 second, near to far 0.792 second, far to near 0.770 second and near to far and return 1.562 seconds. Thirty minutes to complete the test. The 16 diopters of exophoria is notable here.

CASE II.—Age 36, 1,400 air hours, 8 years in aviation. Visual acuity 20/20 in both eyes. Accommodation 7 diopters both eyes. Angle of convergence 60. Phorometer findings at 6 meters; Prism divergence 6 diopters, exophoria 1 diopter; at 33 cm, exophoria of 9 diopters. Depth perception 10. Tachistoscope findings; near 0.033 second, near to far 1.177 seconds, far to near 0.440 second, and near to far and return 1.617 seconds. Forty-five minutes to complete the test. Nothing of note here except exophoria of 9 diopters at 33 cm.

CASE III.—Age 37, 1,400 air hours, 11 years in aviation. One crash. Defective color perception. Visual acuity 20/20 both eyes. Accommodation, 7.5 diopters both eyes. Phorometer readings at 6 meters: Prism divergence 7 diopters, left hyperphoria 0.2 diopters; at 33 cm, exophoria 4 diopters. Depth perception 8. Tachistoscope readings; near 0.044 second, near to far 1.166 seconds, far to near 0.440 second and near to far and return 1.606 seconds. Thirty minutes to complete the test. The defective color perception, the prism divergence and the left hyperphoria should be noted.

CASE IV.—Age 37, 2,800 air hours, one crash, 15 years in aviation service. Visual acuity: V. o. d. 20/15 minus one, v. o. s. 20/20. Refraction: V. o. d. plus 0.25 axis 75°, v. o. s. plus 0.25 axis 90. Accommodation 6.5 diopters in both eyes. Angle of convergence 65. Phorometer findings at 6 meters: Prism divergence 6 diopters, left hyperphoria 0.3 diopter; at 33 cm, exophoria 3 diopters. Tachistoscope readings: Near 0.044 second, near to far 0.726 second, far to near 0.880 second, and near to far and return 1.606 seconds. Note here the inequality of the two eyes in visual acuity, the astigmatic error, and the left hyperphoria. It took 30 minutes to complete the test.

CASE V.—Age 38, 2,100 air hours, 11 years in the aviation service. Visual acuity: 20/20 both eyes. Refraction: Astigmatic error in both eyes. Accommodation, 5.5 diopters both eyes. Angle of convergence 62. Phorometer findings negative. Depth perception 13. Tachistoscope findings: Near 0.033 second, near to far 0.957 second, far to near 0.660 second, and near to far and return 1.617 seconds. The test was completed in 30 minutes. The astigmatic error should be noted here.

CASE VI.—Age 39, 1,500 air hours, 6 years in aviation, 2 crashes. Visual acuity: 20/15 in each eye. Accommodation, 5.5 diopters. Angle of convergence 56. Phorometer readings at 6 meters: Prism divergence 4 diopters, right hyperphoria 0.2 diopter; at 33 cm, exophoria 3 diopters. Depth perception 10.

Tachistoscope readings: Near 0.044 second, near to far 1.056 seconds, far to near 0.484 second, and near to far and return 1.540 seconds.

In the age group of pilots, 40 to 44.9, we find one pilot with complete time, near to far and return, between 1.75 and 1.949 seconds. Age 44. 2,600 air hours. 12 years in aviation service. 4 crashes. Visual acuity 20/20 each eye. Refraction: V. o. d. plus 0.25, v. o. s. plus 0.25. Accommodation, 5.5 diopters. Angle of convergence 62. Phorometer findings at 6 meters; prism divergence 5 diopters, exophoria 1 diopter; at 33 cm, exophoria 3 diopters. Depth perception 8. Tachistoscope findings; near 0.198 seconds, near to far 0.902 second, far to near 0.880 second, and near to far and return 1.782 seconds. Thirty minutes for the entire test.

In the above 16 cases which are all considered slower in speed of adjustment for their age than is deemed safe the following conditions seem to stand out; inequality of visual acuity, astigmatic errors, high exophoria at 33 centimeters, hyperphoria, and slowness in accomplishing the test. Of the 16 men involved 8 are known to have crashes, and 2 have had more than one crash.

In the attempt to analyze the data obtained to date, the following graphs have been constructed:

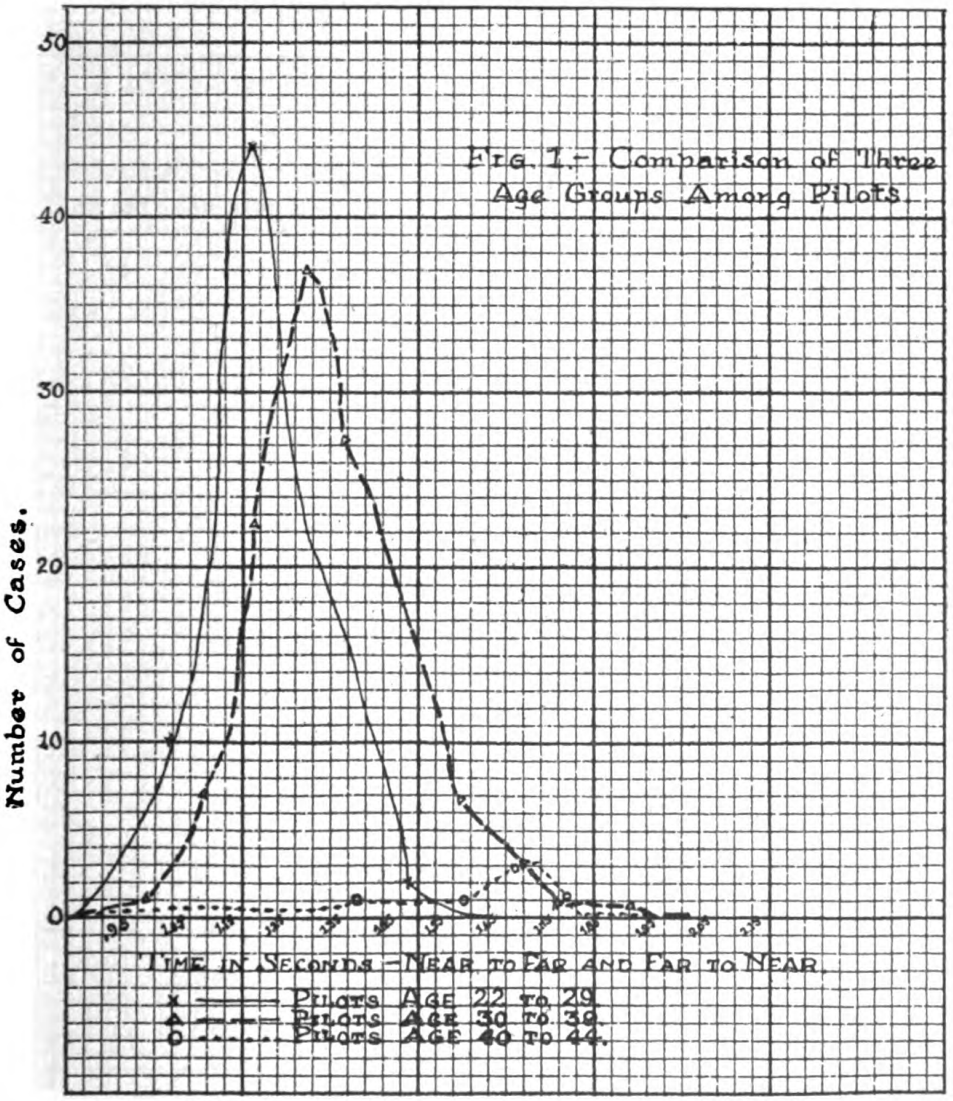
Figure I gives a comparison of various age groups among the pilots age 22 to 29, 30 to 39, and 40 to 44. The modal time of the first group as noted before is between 1.15 and 1.25 seconds; of the second group, between 1.25 and 1.35 seconds, and of the third group, between 1.65 and 1.75 seconds. The average time for each of these groups is respectively 1.24, 1.38, and 1.64 seconds. This demonstrates the age variance in a selected group.

Figure II shows the distribution of time values: Near to far and back to near for pilots and nonpilots approximately the same age, pilots from age 22 to 29, inclusive, and nonpilots from 18 to 29, inclusive. The modal value occurs between 1.15 and 1.25 seconds for both groups. For the aviators, however, more cases have higher speed than the modal value as compared with the nonselected group, and conversely, more nonpilots have slower than the modal speed as compared with the group of aviators. For example, with the aviators or the selected cases we have one case faster than 9.5 seconds and 10 between 9.5 and 1.05 seconds. While with the nonpilots or nonselected group we have no cases faster than 1.05 and 1.15 seconds; then the number of aviators drops down rapidly to a time at the extreme of 1.55 and 1.65 seconds while the curve of the nonpilots holds until between 1.75 and 1.95 seconds. The average value of this group of pilots is 1.24 seconds and of the nonpilots is 1.32 seconds. This would seem to be an excellent example of selectivity of the pilots in comparison to the nonpilots.

Figure III demonstrates a comparison of the time element of near to far and far to near in the same pilots, of age 30 to 39. The

modal time of near to far being between 0.70 and 0.80 seconds, the same as of pilots between ages 22 and 29; while far to near being between 0.50 and 0.60 seconds, an increase over pilots between ages 22 to 29 which was between 0.40 and 0.50 seconds.

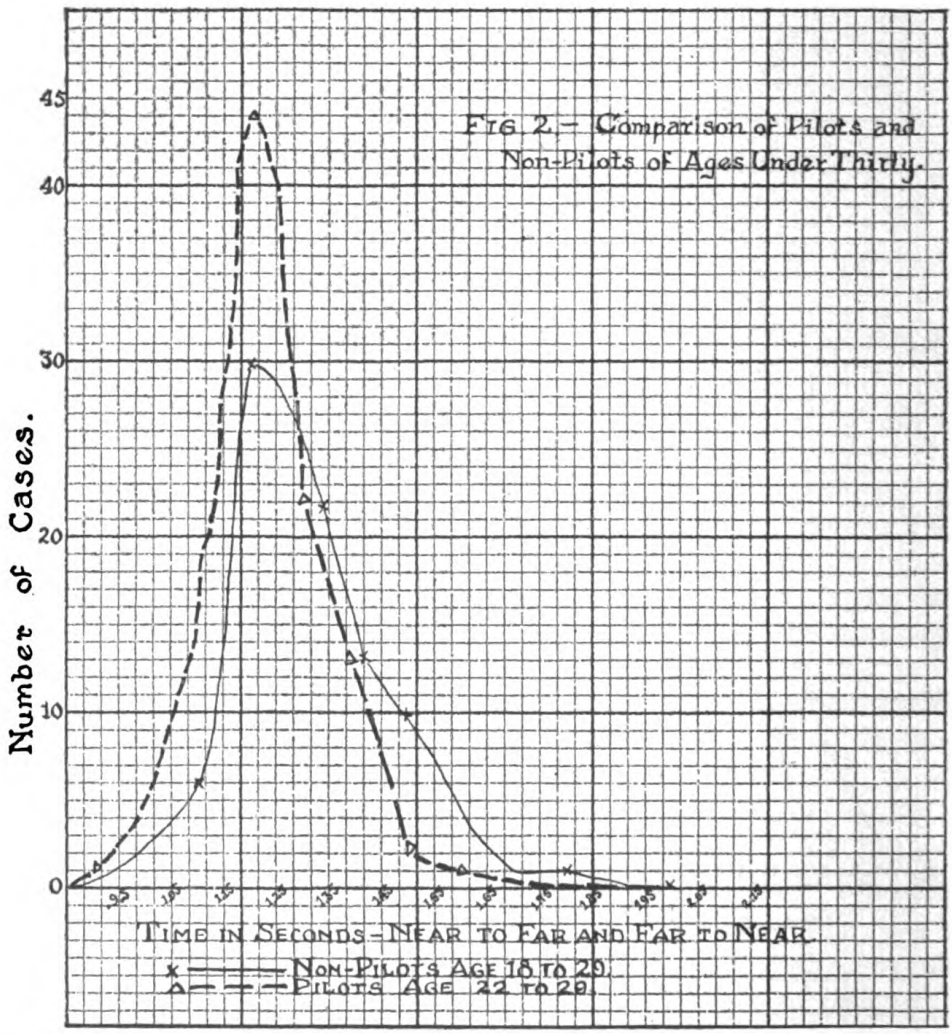
An analysis of the data obtained for the pilots was further made with the hope of determining what ocular conditions play a part in the speed of adjustment of the eye for different distances. The



following ocular conditions were considered as possible factors: Prism divergence of different amounts; degree of exophoria at 33 centimeters; degree of esophoria at 33 centimeters; depth perception etc.

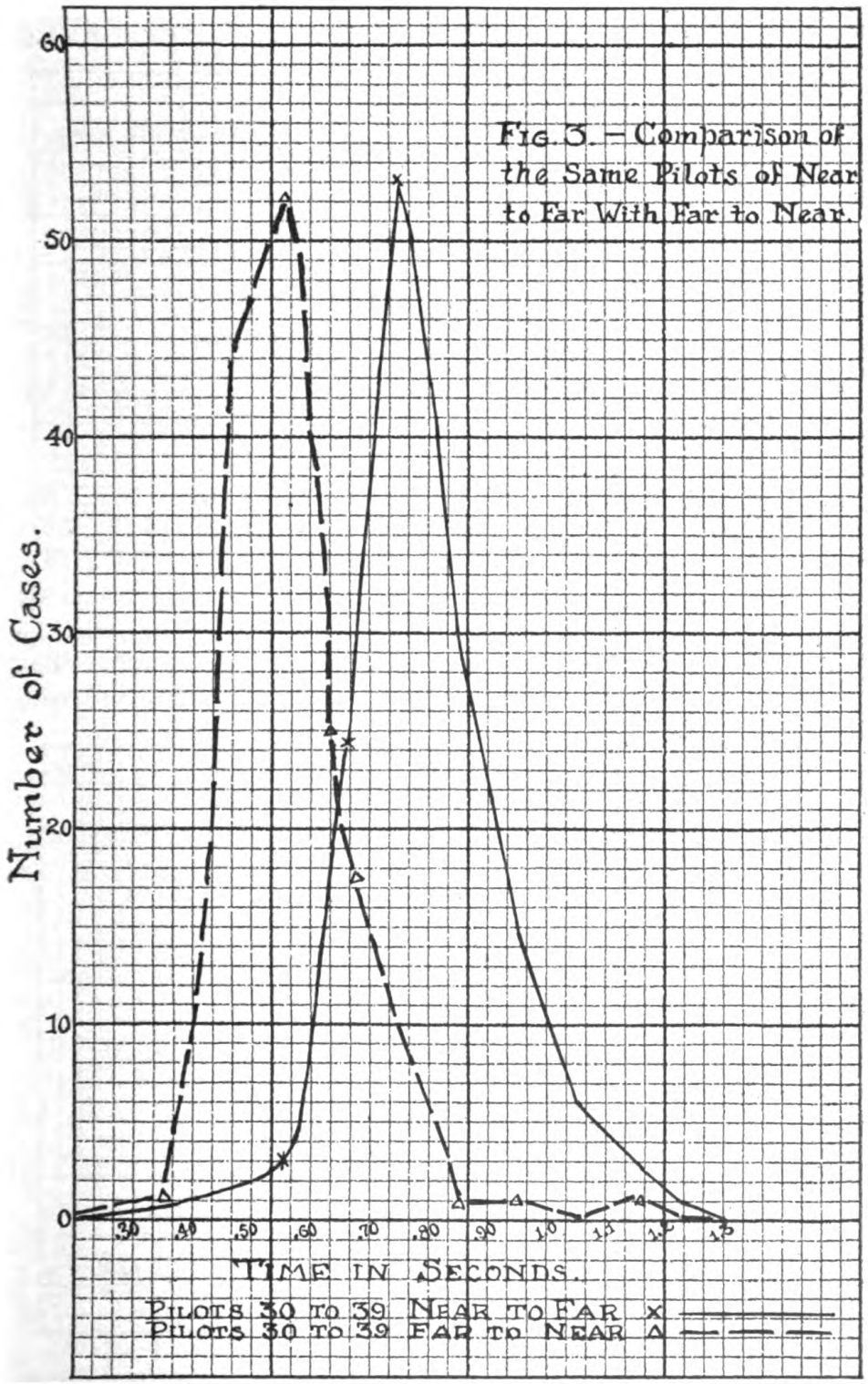
Figure IV shows the effect of prism divergence on speed of adjustment.

Twenty men, with prism divergence between 2 diopters and 7 diopters and twenty with prism divergence greater than 8 diopters, of the age group 22 to 39, were selected for the comparison which was made for the complete time near to far and back to near. The graphs show that the men with the greater amounts of prism divergence tend to have slower speed of adjustment than those with lesser amounts. The slowing up of speed due to this ocular condition is more pronounced in the adjustment for near to far than in the



adjustment for far to near. A similar comparison was made for seven men in the age group 30 to 39. The results were similar to those for the younger age group. Because of lack of space, they will not be reproduced here.

Figure V shows the effect of degree of exophoria at 33 centimeters. For this comparison also, the age group 22–39 years was selected and a comparison was made for 33 men with 5 diopters or less



exophoria at 33 centimeters and 33 men with 10 diopters or greater. The comparison was made for the adjustment of the eyes with the completed time, near to far and back to near.

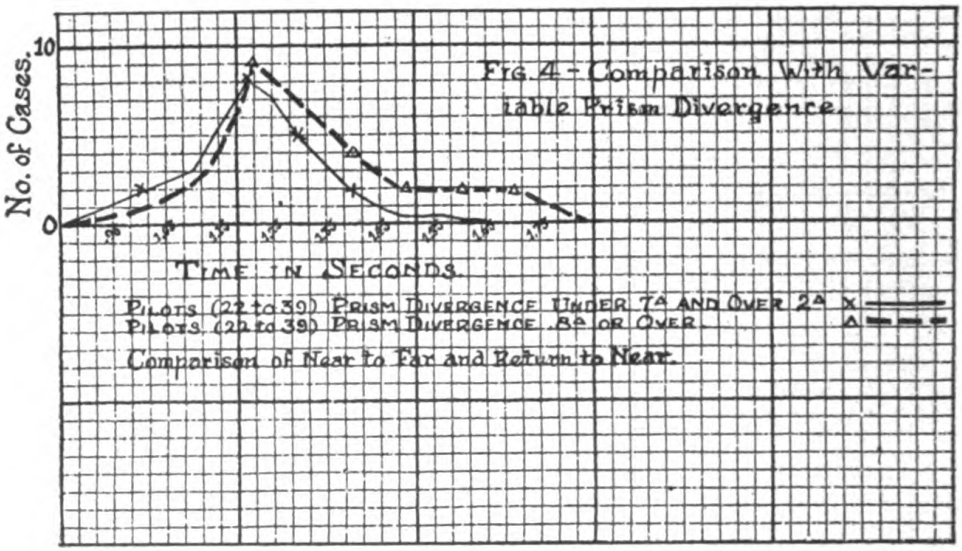
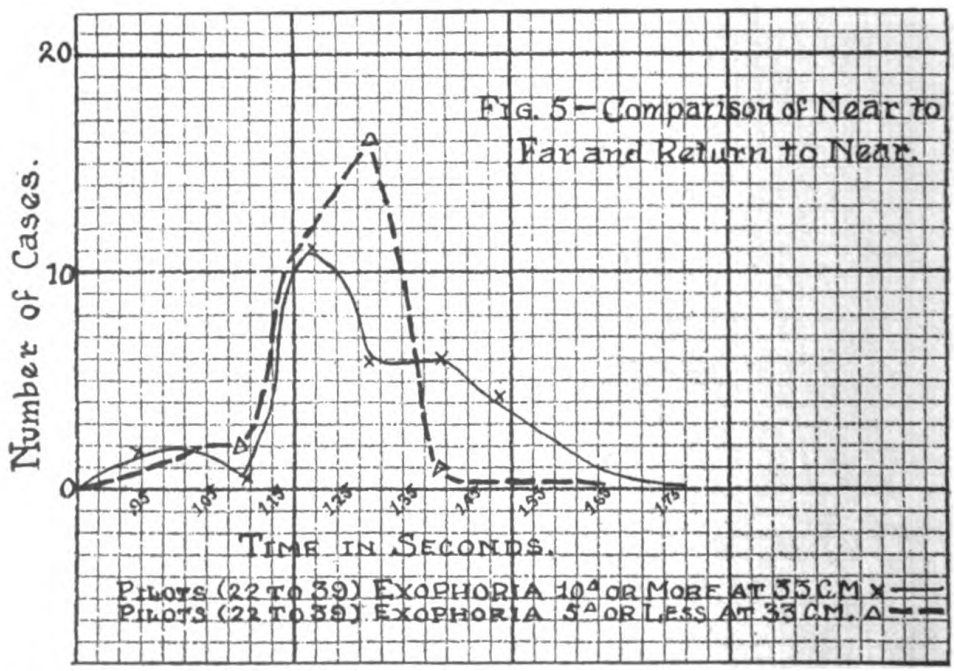
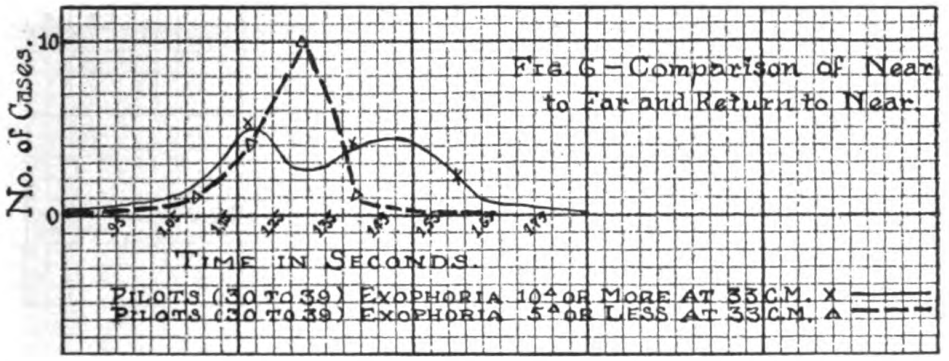


Figure VI shows the effect of an exophoria of 10 diopters or more at 33 centimeters in comparison to exophoria of 5 diopters or less at 33 centimeters. The age groups being between 30 to 39. The comparison was made for the adjustment near to far and return to



near. It is noted that the pilots with an exophoria of 10 diopters or more at 33 centimeters demonstrate a much slower time in all phases.

Figure VII, as in figure VI, considers men with an exophoria of 10 diopters or more at 33 centimeters in comparison to men with an exophoria of 5 diopters or less but in the age group of 22 to 29. Again is it demonstrated that those with an exophoria of 10 diopters or more are slower than those with an exophoria of 5 diopters or



less. However, the slowness in speed is not as marked as is the age group between 30 to 39, due to the youth of the individuals.

Figure VIII is a comparison of pilots, ages 22 to 29, with an exophoria of 10 diopters or more at 33 centimeters with pilots, ages 30 to 39, with an exophoria of the same amount. The age factor is here demonstrated in the slowing of the speed of those in the age group of 30 to 39.

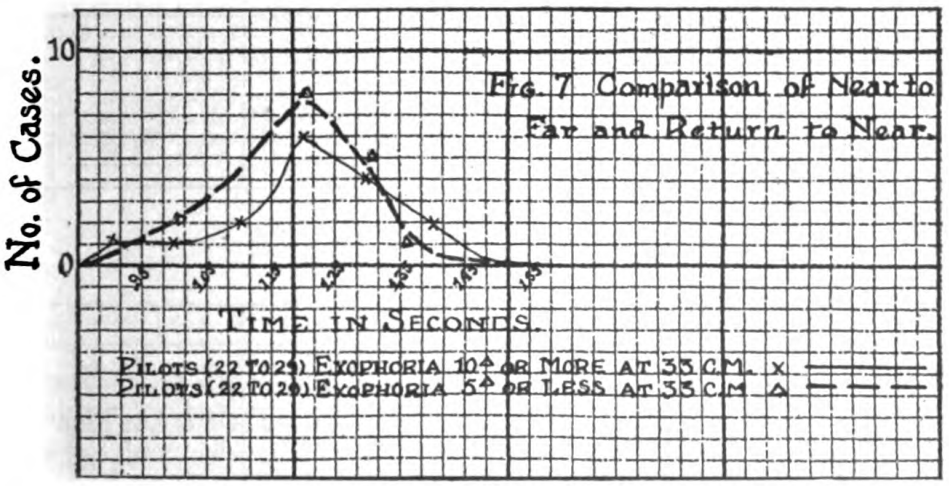
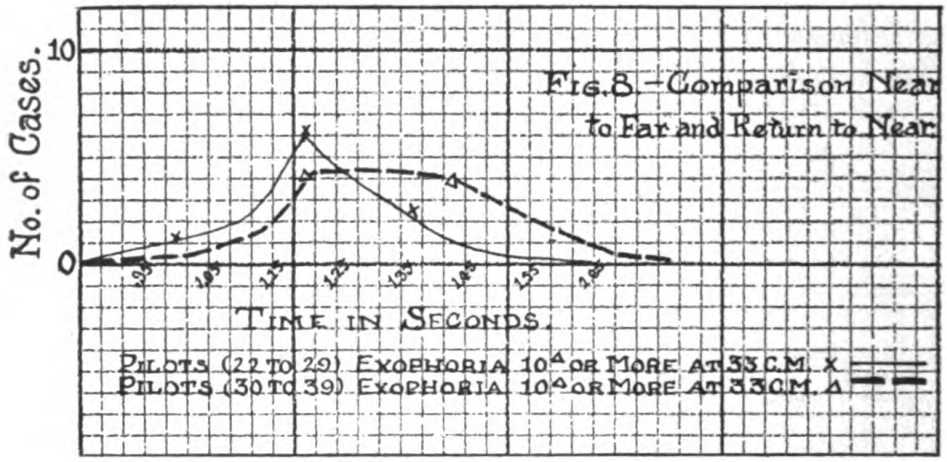
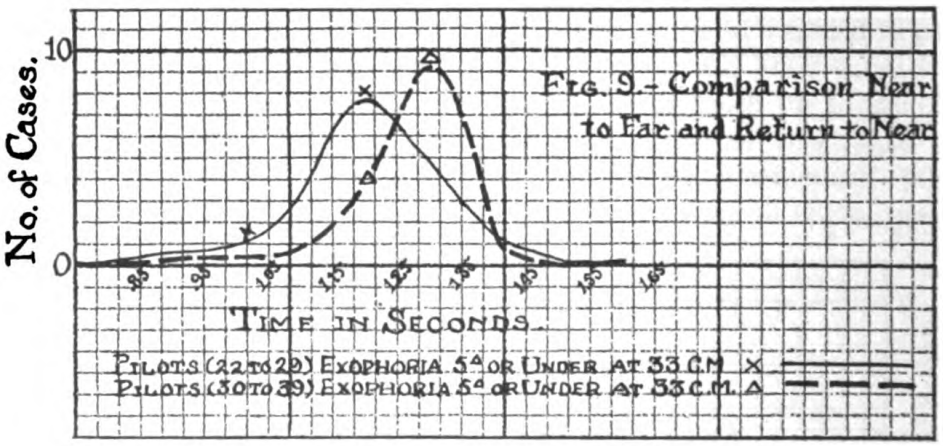
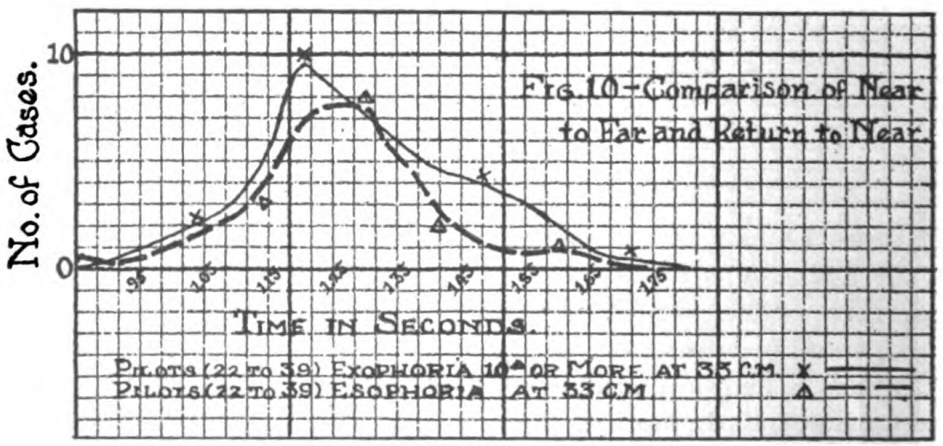


Figure IX is a comparison of pilots in the age groups, 22 to 29, to the age groups of 30 to 39, each group having an exophoria of 5 diopters or less at 33 centimeters. The age factor is again noted in the speed of adjustment of the eyes.

Figure X shows a comparison of pilots, age 22 to 39, with an exophoria of 10 diopters or more at 33 centimeters, with pilots, age 22 to 39, with an exophoria at 33 centimeters. Due to the youth of these groups there is no marked slowing in the speed but it is notable

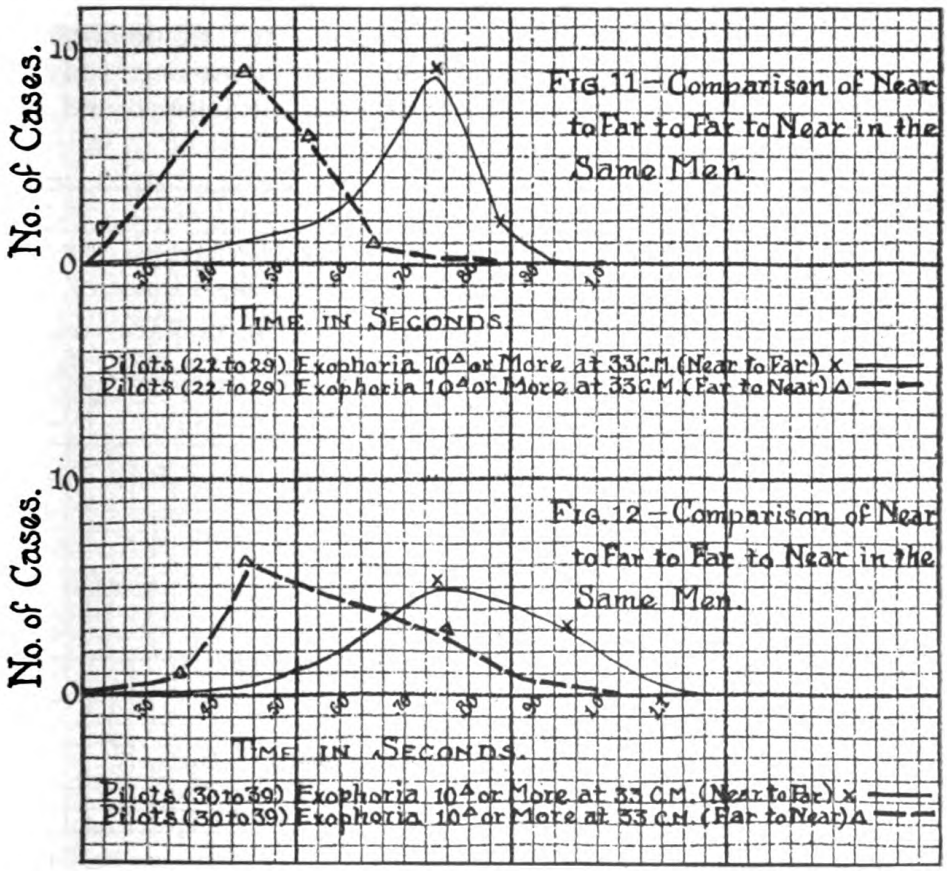






that those with 10 diopters or more of exophoria at 33 centimeters are slower, which is particularly noted near to far and return to near.

Figure XI, graph A is a comparison of speed of adjustment of near to far, to far to near in the same individuals, pilots, age 22 to 29, and with an exophoria of 10 diopters or more at 33 centimeters. The time for near to far is markedly slower than for far to near.



Graph B compares the same factors and conditions in pilots of 30 to 39 with the same findings.

In a comparison of the two graphs it should be noted that there is a marked slowness of the age groups 30 to 39 in comparison to the age groups 22 to 29.

Figure XII with its graph considers pilots, age groups 22 to 39, one group having an exophoria of 5 diopters or less at 33 centimeters and the other group an esophoria at 33 centimeters. The comparison was made for only the speed of adjustment of the eyes for near to far and return to near. As would be expected there is no difference in the speed of the two groups.

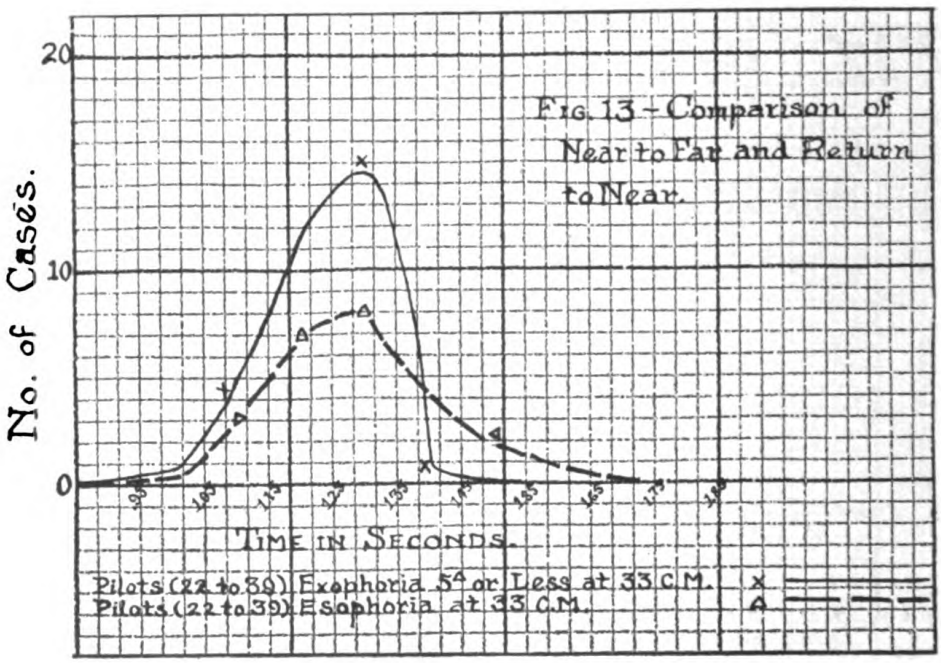
It has been impossible to use all the graphs considered necessary to make the complete picture in the findings which we have attempted

to demonstrate, so we have deleted most of the comparisons of near to far and far to near.

A further study with the tachistoscope is being made at the air station at Pensacola, Fla., in order to increase our data. Also work is being done under conditions of fatigue.

SUMMARY

In attempting to summarize our findings it must be fully understood that we do not consider we have had a sufficient number of tests to completely prove our original premises; however, we do consider that we have had enough to make an intensely interesting study of the selected cases and the comparison between the selected and the nonselected cases.



It would seem that conclusive evidence has been set forth to show that age has a definite bearing on speed of adjustment of the eye, that muscle imbalances, inequality of vision, refractive errors, etc., tend to slow the speed of adjustment and that selected men such as the pilots, tend to have a greater speed of adjustment throughout all ages.

CONCLUSIONS

1. That age has a marked bearing on the speed of adjustment of the eye to clear seeing for different distances.
2. That with any anomalous condition of the eye, the age factor becomes of greater importance in the speed of adjustment.
3. That after the age of 30 the changes become rapidly more marked.

4. That, at the age below 30, anyone with a speed over 1.35 seconds near to far and return to near should be disqualified unless causative factor can be found and eradicated.

5. That, at the age of 40 to 30, anyone above 1.55 seconds near to far and return should be disqualified unless the causative factors can be found and eradicated.

6. That insufficient data is available at present to judge of the speed at the ages over 40 but it is considered that anyone over 1.75 seconds, near to far and return, is unsafe as a pilot.

7. That the limit considered for near to far below age 30 is 0.90 second, near to far age 40 to 30 is 1.0 second, near to far above 40 is 1.0 second, far to near below age 30 is 0.60 second, far to near age 40 to 30 is 0.70 second, far to near above 40 is 0.70 second.

8. That the speed of adjustment of far to near is markedly faster in all ages than near to far.

9. That an exophoria of 10 diopters or more at 33 centimeters has a definite bearing on the slowing of the speed of adjustment of the eyes. This slowness is even more marked as age increases.

10. That an inequality of visual acuity of the eyes, not necessarily below the required 20/20 vision has a definite bearing on the speed of adjustment of the eyes.

11. That there is as yet insufficient data on depth perception to make a statement.

12. That our original premises have been upheld and that speed of adjustment is essential in aviation and that a measurement of such speed can be made; that a standard should be accomplished, and that the pathological eye is disclosed with the tests on the tachistoscope.

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RESUSCITATION OF THE STOPPED HEART BY INTRACARDIAL THERAPY IV. FURTHER USE OF THE ARTIFICIAL PACEMAKER[1][2]

By ALBERT S. HYMAN, Lieutenant Commander, Medical Corps, United States Naval Reserve

INTRODUCTION

Sudden death from cardiac arrest or ventricular standstill is not an uncommon problem in civilian hospitals and general emergency

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[1] From the Witkin Foundation for the Study and Prevention of Heart Disease, Beth David Hospital, New York.

[2] Published by permission of and in accordance with the provisions of the Carolina Greenwood Fund for Cardiovascular Research, Grant No. 11; 1934.

stations equipped with ambulance service; such sudden deaths are naturally far more common in active military service and will become increasingly of greater importance with the advent of gas warfare. Recent experiments (1) have shown that many types of "sub-asphyxiating" gases leave irreparable heart damage which subsequently may lead to myocardial dissolution and sudden death.

Death from cardiac arrest during battle action was frequently seen during the World War; all medical military services reported many nontraumatic sudden deaths. Some of these were listed as "shock", others as "acute dilatation of the heart", and still others as "acute indigestion."

In previous reports (2) we have attempted to classify the clinical causes of sudden death from ventricular standstill. A brief review of these causes may not be amiss here; a more complete study of this problem will be found in the original communications made in 1930.

From a clinical point of view there are three general types of the dying heart syndrome. The first is cardiac arrest caused by sinus nodal paralysis; in this instance the pacemaker of the heart fails to release the excitatory impulse necessary for the orderly sequence of the cardiac cycle. The heart ceases to beat because the stimulus for myocardial contraction is not forthcoming. Such paralysis of the pacemaker mechanism may either be due to extracardiac causes or to intracardiac pathology. Among the extracardiac causes may be mentioned the neurogenic syndromes, the effect of excessive endocrine stimulation like hyperthyroidism, hyperadrenalism, and hyperinsulism, and finally disturbances in the major neurologic control of the sino-auricular nodal area. Of the intracardiac causes, changes in the blood supply to the pacemaker and abnormal physiological reactions in the nodal cells may be responsible for adynamic failure of impulse release.

The second type of the dying heart syndrome is seen in most all cases of death from acute or chronic infectious disease, metabolic disturbances like diabetes and uremia, cachexia, and hemorrhage. Cardiac arrest, here, is due to progressive conduction changes in the specific systems of the heart. The pacemaker continues to function more or less regularly but the stimulus for myocardial contraction may be blocked at any of 6 different points, any or all of which may be responsible for ventricular standstill. Without going too deeply into the pathologic physiology of this interesting aspect of the dying heart, it may be said that such conduction depression is responsible for about 60 percent of deaths from all causes.

Finally, the third type of the dying heart syndrome is due to electro-dynamic dissolution of the cardiac cycle; this condition occurs in certain severe forms of heart disease like coronary thrombosis and

occlusion and angina pectoris and is responsible for many such sudden deaths (3).

We have here only to consider the first type of the dying heart syndrome; this report is thus concerned with the problem of pacemaker paralysis and the possible treatment thereof. Sudden death from ventricular standstill as the result of extracardiac causes and the role of the "subasphyxiating gases" will be discussed; the use and rational of intracardial therapy will be studied, and finally the employment of the artificial pacemaker will be described.

THE ANOXEMIC PHENOMENA OF VENTRICULAR STANDSTILL

The physiologic changes which take place in the myocardium and the mechanism of the cardiac cycle when the ventricles stop beating have been previously described (4). We have shown that simultaneously with ventricular arrest there is prompt secession of coronary blood flow with increasing venous stasis. Oxygen, glycogen, and other basic metabolic substances are rapidly used up and a developing oxygen debt occurs. In addition, there is an increasing accumulation of the waste products of metabolism like CO_2, lactic acid, and A-urea. This oxygen debt on the one side and the accumulating acid products on the other cause marked changes in H-ion concentration of the heart muscle.

Synchronously with these chemical changes there develops in the myocardium differences in irritability and electrical conductivity which run almost parallel. The curves of H-ion concentration and electrical conductivity are graphically presented in figure 1A.

It will be noted that the anoxemic period of the dying heart syndrome may be divided into four phases; each of these periods exhibit separate and distinct physiologic reactions. Depending upon which phase the dying heart is passing, will depend the success or failure of attempted resuscitation by intracardial injection measures. The entire theory of such resuscitation procedures depends upon a clear understanding of the physiologic background of the various phases of myocardial anoxemia through which every dying heart must pass.

The first phase of anoxemia lasts for about 90 seconds after ventricular standstill. The curves of H-ion concentration and electric conductivity start to rise rapidly. Any irritable stimulus may be sufficient to develop an ectopic focus from which an extrasystole can arise. The mechanism of such stimulus production will be subsequently described; suffice it here to state that the extrasystole thus produced is sufficient to give maximal contraction to the ventricles. Blood is forced into the circulation and on the next diastolic interval the coronaries are filled. During cardiac standstill the ventricles have been greatly dilated so that the extrasystolic contraction has

permitted a maximal volume output. The sinus nodal area as well as all other parts of the heart again receive their blood supply and prompt restoration of automatic pacemaker function may occur.

Figure 1B shows the series of events which take place in the experimental animal during the first phase of anoxemia; this and all subsequent records are taken from electrocardiographic studies made in the position of Lead II. The tracing illustrates an initial period of cardiac arrest; following an ectopic stimulus, an extrasystole arises and there promptly occurs a normal sinus rhythm with return of automatic pacemaker function.

The second phase of anoxemia develops after 90 seconds and continues for about 3 minutes. Referring to figure 1A it will be noted that the two curves are rising very rapidly and gradually approaching each other. Physiologically, this would mean that both acid concentration and electrical activity are markedly enhanced. Experimentally, it can be shown that now any stimulus will not only cause an ectopic contraction of the heart, but the stimulus will persist and many extrasystoles will develop. In figure 1C, for example is shown the electrocardiographic tracings made from an experimental animal about 3 minutes after ventricular standstill. The primary extrasystole is followed by a series of right ventricular ectopic beats; this rhythm becomes a paroxysmal extrasystolic tachycardia which sooner or later degenerates into ventricular fibrillation and complete breakdown of the cardiac cycle. Concerning this physiological phenomenon, more will be said subsequently.

The third phase of anoxemia extends from the fifth minute after ventricular standstill to an indefinite maximum of 10 minutes. During this period, the two curves shown in figure 1A have reached their maximum height and are now running parallel to the time abscissa of the graph. Coordinated muscular response on the part of the heart as a whole, is now rapidly lost. Stimuli of increasing intensity are necessary to produce a single ectopic beat. Following such an extrasystole, the myocardium returns to an adynamic state. In figure 1D is shown the electrocardiographic tracing made from an experimental animal 9 minutes after ventricular standstill. It will be noted that the primary extrasystole is not followed by any form of myocardial reaction.

The fourth or terminal phase of myocardial anoxemia is one of complete electrodynamic and muscular dissolution. No coordinated response of any kind is possible at this time regardless of the type and strength of stimulus used.

In summary, therefore, it may be said that immediately following ventricular standstill, there develops a physiological state in the heart muscle described as anoxemia; this anoxemic phase of the

dying heart may be divided into four well-recognized phases, each with its own distinctive reaction to the reception of external stimuli and the propagation thereof throughout the various systems of the heart as a whole. Resuscitation of the stopped heart is possible in any of the three first phases; no resuscitation is possible in the fourth phase.

THE MECHANISM OF INTRACARDIAL STIMULATION

Resuscitation of the stopped heart by the intracardial injection of many substances has been known and practiced for about 20 years (2). Twelve drugs have been used in addition to epinephrine (5). The most common of these have been ether, strychnine, strophanthin, and alcohol; epinephrine has, however, during the past decade been administered more frequently than any other substance. We have previously shown that resuscitation is apparently equally well accomplished by any substance sufficiently irritable to produce an ectopic focus in the heart muscle.

Close examination of our experimental data disclosed that the resuscitation procedure was apparently but little influenced by the drug or chemical injected into the anoxemic heart. In fact, it was found that the needle thrust or prick of the heart alone was sufficient to produce a restoration of automatic cardiac activity after standstill had occurred. Furthermore, it was shown in piercing the heart muscle, a certain number of fibers were injured and from this injured point there developed a specific electrodynamic reaction known to physiologists as the "action current of injury." This action current of injury is a familiar experimental reaction in striated muscle laboratory preparations. When generated in the anoxemic heart muscle, this electric current as shown by electrocardiographic studies is of much greater intensity than the excitatory process liberated from the sinus nodal area itself.

We may now return to a consideration of what happens when a needle is thrust into the heart during ventricular standstill and what may be anticipated during the various phases of anoxemia. If, for example, the needle thrust has been made during the first phase of anoxemia when the two curves of H-ion concentration and electrical irritability are increasing, the action current of injury generated by puncture of the heart will spread throughout the myocardium and an extrasystole develops. Restoration of the normal pacemaker function is possible and the dominant rhythm of the heart is maintained from the sinus node.

During the second phase of anoxemia, however, the same needle thrust will produce an action current of injury of much greater magnitude and intensity; moreover, the factors of electric irritabil-

ity have also been enhanced. Instead of the injured heart muscle fiber focus producing a single action current, many such action currents are developed. Electric stimuli are released at a very rapid rate said to be almost 1,000 per second. Due to the refractory period phenomenon in heart muscle, however, only as many of these impulses are effective as can be conditioned by the recovery phase of myocardial contraction. This rate is about 200 to 240 contractions per minute. Inasmuch as the stimuli for such cardiac contraction arises outside of the normal pacemaker mechanism and since this ectopic focus dominates the rhythm of the heart, such a rhythm may be called a " paroxysmal extrasystolic tachycardia." It will be shown later that such rhythms can be maintained only over short periods of time, as ventricular break-down sooner or later occurs with flutter and fibrillation and circulatory arrest.

Finally, if the needle thrust has been made into the heart during the third phase of anoxemia, the action current which is developed is only sufficiently intense to develop but a single ectopic beat. The heart muscle, on the other hand, will respond to such single stimuli for several minutes; the importance of this physiologic phenomenon will be considered later in relation to the use of the artificial pacemaker.

Resuscitation of the stopped heart by intracardial injection is, therefore, due almost entirely to the electrodynamic reactions developed by the needle thrust into the anoxemic myocardium rather than to any specific pharmodynamic action of the chemical or drug which is used. Recent experiments (6) tend to show that certain phases of electrodynamic activity are enhanced by epinephrine, ether, and alcohol. Primarily, however, it is the action current of injury which is responsible for the development of the primary extrasystole and for the sequence of events which follow in the various phases of anoxemia.

THE SITE FOR INTRACARDIAL INJECTION

We may now approach the clinical use of intracardial therapy in cardiac resuscitation. In all of the available literature up to 1928, the site for intracardial injection has been into the left ventricle. This apparently has been due to the then current impression that the most accessible part of the heart lies to the left of the sternum and may be reached through the third, fourth, or fifth interspaces on that side. The hazards of such ventricular puncture were not well understood until the experiments of the late Dr. G. A. Friedman disclosed the fundamental differences in the physiological response to auricular and ventricular puncture.

These differences may be readily shown in the following series of graphic charts which have been previously used for post-graduate medical instruction (7).

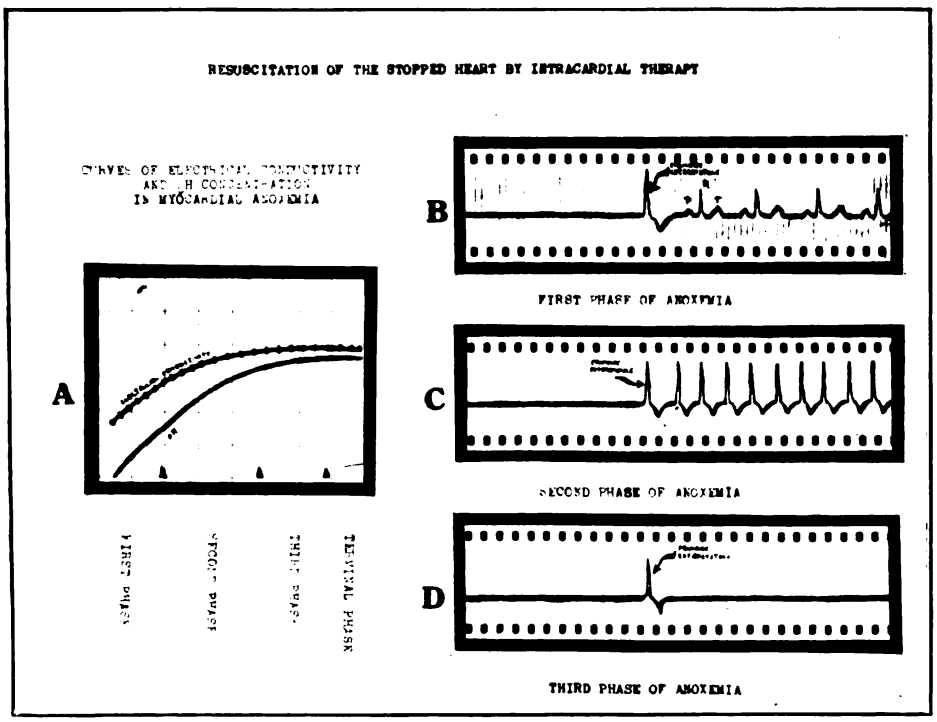

FIGURE 1.—INVESTIGATION OF THE DYING HEART.

Demonstration of the curves of electrical conductivity and hydrogen ion concentration of the heart muscle during cardiac standstill. During the first phase of anoxemia the myocardium is receptive to external stimuli with the development of ectopic primary beats which are quickly converted into a normal sinus rhythm. During the second phase of anoxemia the ectopic stimulus persists and develops into a paroxysmal extra-systolic tachycardia which may rapidly be converted into a ventricular flutter fibrillation and death. The third phase of anoxemia shows only one response to the ectopic stimulus, a single primary extrasystole being developed. See text for further explanation of this phenomenon.

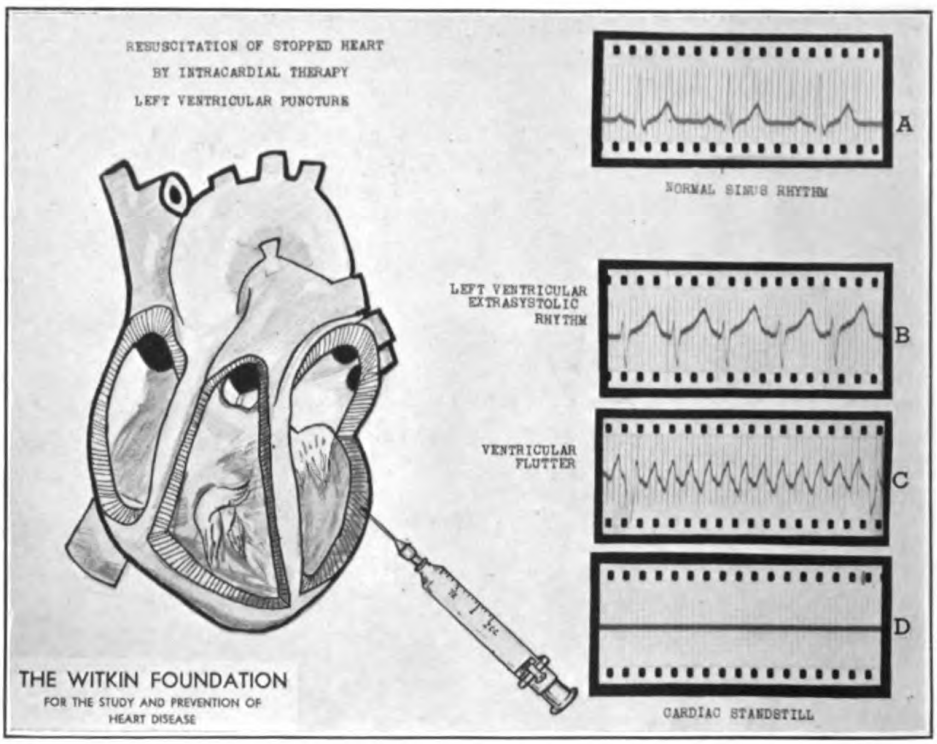

FIGURE 2.—INVESTIGATION OF THE DYING HEART.

Records illustrating the sequence of events which take place when intracardial injection is made into the left ventricle for resuscitation of the stopped heart. If the left ventricular puncture is made during the second or third phases of anoxemia (see fig. 1) there is developed a left ventricular extrasystolic tachycardia which subsequently changes to a ventricular flutter and fibrillation and finally to complete cardiac standstill and vascular collapse with death. Left ventricular puncture should not be attempted in resuscitating the stopped heart.

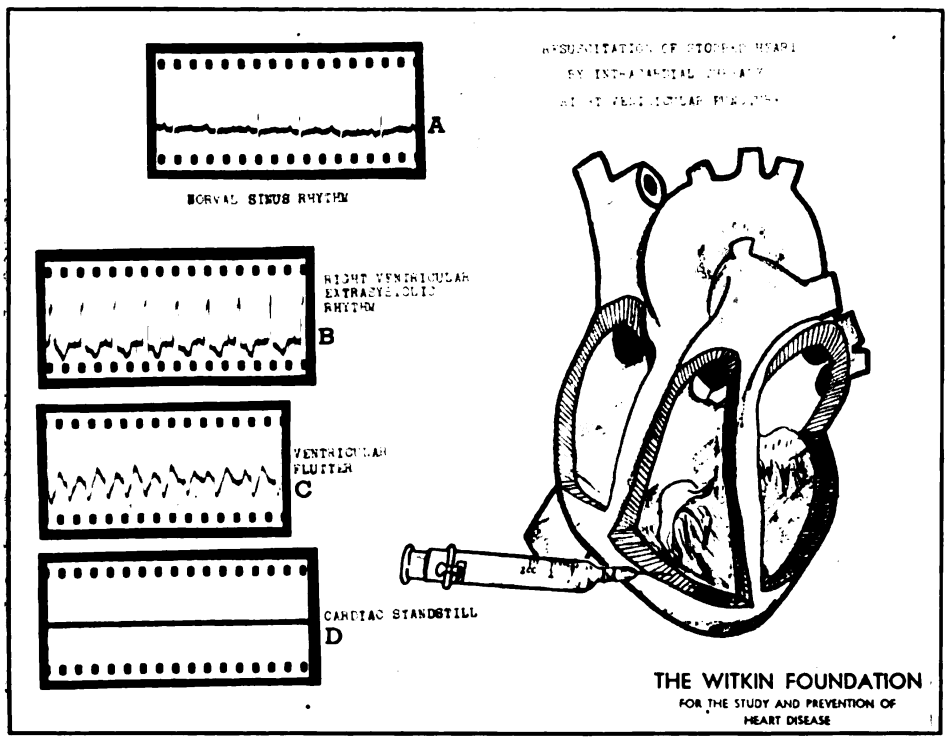

FIGURE 3.—INVESTIGATION OF THE DYING HEART.

Records illustrating the sequence of events which take place when intracardial injection is made into the right ventricle for resuscitation of the stopped heart. If the right ventricular puncture is made during the second or third phases of anoxemia (see fig. 1) there is developed a right ventricular extrasystolic tachycardia which subsequently changes to a ventricular flutter and fibrillation and finally to complete cardiac standstill and vascular collapse with death. Right ventricular puncture should not be attempted in resuscitating the stopped heart.

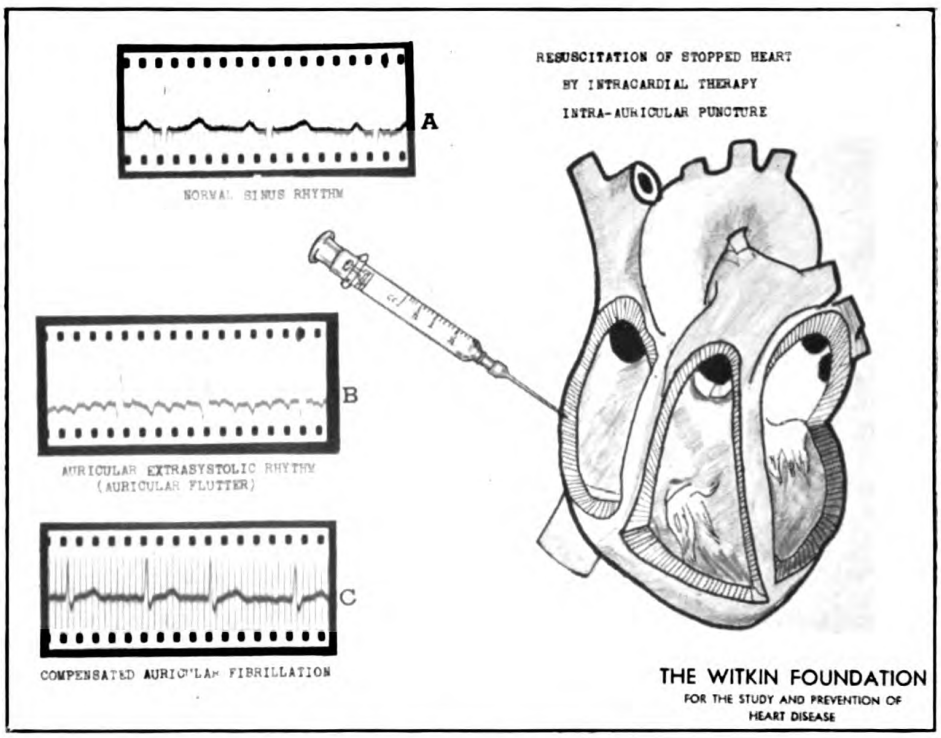

FIGURE 4.—INVESTIGATION OF THE DYING HEART.

Records illustrating the sequence of events which take place when intracardial injection is made into the right auricle for resuscitation of the stopped heart. In this instance, the puncture is likewise followed by a paroxysmal extrasystolic rhythm the difference being that whereas flutter and fibrillation of the auricles is a well known and easily handled clinical entity not incompatible with life, ventricular fibrillation is always associated with vascular collapse and death. Intracardial injection treatment for resuscitation of the stopped heart should, therefore, always be made into the right auricle.

Figure 2 illustrates the series of events which take place when resuscitation is attempted by injection into the left ventricle. Figure 2A shows the electrocardiographic tracing of an individual with a normal sinus rhythm. After ventricular standstill has occurred, injection has been made into the left ventricle during the second phase of anoxemia. Figure 2B shows the prompt development of many extrasystoles arising from the left ventricle where the puncture wound has been made. This rhythm may be called a paroxysmal left ventricular extrasystolic tachycardia with a ventricular contracting rate of about 160 beats per minute. As previously indicated, such rhythms degenerate into ventricular flutter and fibrillation; such a rhythm is seen in figure 2C where the ventricular rate now averages 300 beats per minute. Breakdown of the heart quickly follows such a contracting strain and there is once more cardiac standstill shown by the tracing figure 2D.

Let us turn now to a consideration of the series of events following intracardial injection into the right ventricle. In figure 3 is graphically shown the changes which take place. Figure 3A are the electrocardiograms of the individual prior to ventricular standstill. Injection is now made into the right ventricle during the second phase of anoxemia. Figure 3B shows the prompt development of a paroxysmal right ventricular extrasystolic tachycardia which is comparable in many respects to the extrasystolic arrhythmia seen in figure 2B. In this instance also there is a change from this rapid rhythm to one of ventricular flutter (fig. 3C) and finally to complete cardiac standstill as shown in figure 3D.

Contrast now the hazards of ventricular puncture with the series of events which take place when the injection is made into the right auricle; these are shown in figure 4. Here again figure 4A shows a normal sinus rhythm prior to cardiac standstill. Injection is now made into the right auricle during the second phase of anoxemia. Just as in figures 2B and 3B there is a development of a paroxysmal extrasystolic tachycardia but in this instance (fig. 4B) the rhythm is auricular in origin and as such is identical with well-known clinical forms of auricular flutter. Auricular flutter and fibrillation are easily handled disturbances of rhythm because of the protecting mechanism which blocks most of the impulses from passing through to the ventricles. In other words, although the auricular rate may be very rapid, the ventricular rate may be well within normal limits. This is shown in figure 4C.

The site for intracardial injecton regardless of the substance used and also regardless of the phase of anoxemia through which the heart may be passing should be in the right auricular area. Clinically, this area is just as easy to approach as either ventricle; any

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point in the third and fourth interspaces just to the right of the sternum will reach the right auricle. Intra-auricular puncture is a simple procedure and may be life-saving in selected cases of sudden death from cardiac standstill (8).

CARDIAC RESUSCITATION WITH THE ARTIFICIAL PACEMAKER

Inasmuch as the theory of resuscitation of the stopped heart by intracardial injection is apparently dependent upon the physiologic development of an ectopic electrodynamic system which temporarily assumes the role of pacemaker of the heart, it seemed to us in 1928 while this experimental work was going on, that a simulated or artificial action current might be generated which could take the place of the natural or idiogenic electric stimuli. We had accordingly constructed a small mechanical device which imitated the electric functions of the normal sinus pacemaker in the development and release of a tiny monophasic curent of one millivolt about sixty times per minute. This current was led to the point of a regular intracardial needle through an insulated core. When injected into the heart muscle, an irritable focus is developed each time an impulse is released from the apparatus. This device was originally described in 1932 (4) and subsequently demonstrated at many scientific assemblies. Since it was conceived as a substitute for a nonfunctioning normal sinus nodal pacemaker it has been called the "artificial pacemaker."

Experiments with the artificial pacemaker have shown that the hazards associated with ventricular puncture in the later phases of anoxemia are largely eliminated with this apparatus inasmuch as complete control of stimulus release is provided in the resuscitation process. In addition, the so-called "resuscitation period" can be lengthened to about 14 minutes after ventricular standstill.

In actual clinical use, the artificial pacemaker needle is inserted into the right auricle; here the ectopic stimuli which are developed in the auricular muscle are carried in a normal manner to the auriculo-ventricular node of Tawara from whence it is speeded through the junctional tissues and the common bundle of His to the right and left main branches and finally to the Perkinje terminals in the ventricular walls. In this way a normal cardiac cycle is produced and electrocardiograms of such rhythms show a rapid restoration of automatic sinus nodal activity (9).

It should be needless to say that all intracardial injections as well as that of the artificial pacemaker should be made as aseptic as possible (10). At the same time, however, where such sterility can be maintained only at the sacrifice of precious minutes during the second phase of anoxemia, it is well to remember the adage of

Wolffe that "it availeth not to provide precautions for him who canst not wait; the man dieth ere the preparations art complete."

INTRACARDIAL RESUSCITATION IN THE NEXT WAR

From the foregoing it will be noted that of the three general types of the dying-heart syndrome, the first or pacemaker paralysis is fortunately the type for which most can be accomplished. The normal healthy heart which stops from the extracardiac causes previously discussed is the type which is most susceptible to resuscitation by intracardial injection or by the use of the artificial pacemaker needle (11). Such hearts are found most often in young individuals and are especially prone to occur under the terrific mental and physical stress of warfare. Prompt recognition of the condition and the immediate utilization of appropriate resuscitation measures may do much to reduce the toll of such preventable deaths.

The advent of gas and chemical warfare will unquestionably increase the number and incidence of sudden deaths from pacemaker paralysis. This is especially true of the so-called subasphyxiating gases which through bulbar pathology may cause cardiac arrest long before respiratory paralysis occurs. Respiratory resuscitation measures in such cases are futile if the heart has stopped beating. Experiments have shown that under artificial pacemaker control laboratory animals have been resuscitated after maximal exposure to the common gases of this group. The importance of these studies cannot be overemphasized in the future preparation of our military medical services.

In addition to cardiac deaths from gas warfare, must also be mentioned the hazards of accidental and intential electrocution. Fisher, of the Chicago Rapid Transit Co., believes most such deaths are due to cardiac arrest provided there is no destruction of the central nervous system. H. B. Williams, of Columbia University, has stated that even low-voltage currents may cause death from cardiac standstill. In all of these instances, intracardial injections or the artificial pacemaker needle may be life-saving and should be adopted as a standard resuscitation procedure.

SUMMARY

1. Studies of the dying heart syndrome show three types, the first of which is concerned with pacemaker paralysis and cardiac arrest. Such cases are common in civilian life but more frequent in warfare.

2. Cardiac arrest is accompanied by four phases of anoxemia, each of which displays specific physiologic phenomena. Resuscitation is possible under certain conditions in the first three phases.

3. The mechanism of intracardial stimulation is due to the development of an ectopic focus of electrodynamic activity; the various phases of the anoxemic heart respond by different reactions.

4. The site of intracardial injection for resuscitation is of the utmost importance; injections into the ventricles may be hazardous. Intraauricular puncture is physiologically correct.

5. The artificial pacemaker may substitute for the sinus nodal pacemaker when the latter is not functioning; control of the dominant electrodynamic system may be life saving.

6. Intracardial resuscitation will be of increasing importance in military medical service with the advent of gas and chemical warfare.

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ILLUMINATION FOR DENTAL OPERATIONS

By HARRY E. HARVEY, Commander, Dental Corps, United States Navy, and CLEMENS V. RAULT, Lieutenant Commander, Dental Corps, United States Navy

In operations of delicacy and precision, as those performed in dentistry, it is essential that the field be properly illuminated. Securing suitable artificial illumination is of considerable importance in the Naval Service where the amount of available daylight in operating rooms aboard ship is usually limited. Formerly, when suffi-

cient illumination was provided, glare, injurious to the eyes of the operator, often existed, due to direct or reflected light in the immediate range of vision. That such a condition was permitted to continue must have been due to a lack of appreciation of the presence of glare and its deleterious effect on good seeing.

One of us (1) has been actively engaged for a number of years in a search for the ideal illumination for dental use. Improvements in dental lighting units have been investigated and lighting engineers have been consulted, but the problem of dental illumination is confined to such a limited field of application it apparently has not received serious thought by investigators in the science of illumination. No claim is made that an ideal system of illumination for the dental operating field has been found by the authors, but the results of our investigations are believed to be in advance of anything we have heretofore observed.

Several factors are encountered in dental operating, an appreciation of which offers a problem of some complexity. They are:

1. Operations are performed in a cavity with a single orifice about 2 inches in diameter for the introduction of illumination and instrumentation.

2. The site of operation may be 2 inches from the orifice and not in the direct line of vision but offset in almost any direction.

3. The field of operation is lined with tissue reddish in hue which reflects but a small proportiion of the amount of light entering the oral cavity, in other words, the amount of reflection of light upon which we depend largely for seeing is but a fraction of that which enters the mouth. Compensation for this loss of light is effected to some extent by the use of the dental mirror. However, much of the actual dental operating is performed with the mirror in use merely as a means of facilitating access to the operating field and as a retractor rather than a source of illumination.

4. The eyes of the operator are not continuously engaged in seeing inside the mouth alone as instruments and appliances are changed frequently, necessitating visual attention elsewhere than in the field of operation. Thus may become involved the factor of glare if the source of the light is not shielded.

5. Our experiments indicate that a measurement of foot candles, or the intensity of illumination, at a given distance from the source of light is not a criterion of the amount of light penetrating an open cavity such as the mouth.

6. Reflected light glare from white towels draped about the patient is in the visual field while operating.

7. The inner surfaces of spectacles may offer a reflecting surface at certain angles, though this reflection factor may not be above the threshold of consciousness.

8. The undesirable factor of excessive heat is present when the source of dental illumination is based merely on the production of a high level of illumination. This is one of the disadvantages of the cluster of four 100-watt lamps commonly used for this purpose.

9. Dentistry comes within the classification of serious visual tasks, as the eyes are engaged with minute details for many hours at a time, and the work causes nervous strain in the operator, upon which may be superimposed the nervous tension (2–3) induced by inadequate illumination.

Experiments were conducted with the following dental lighting units in common use and with several other types constructed by ourselves, in order to test various applications of the principles of illumination:

No. 1. Cluster light of four 100-watt lamps with holophane globes.

No. 2. Flood light, 100-watt daylight blue lamp with white porcelainlike glass reflector about 9 inches in diameter.

No. 3. Spotlight with 50-watt inside frosted lamp with daylight color filter and holophane lens.

Accurate measurements of relative illumination of the above units at a distance of 18 inches were:

No. 1 cluster light, 37 microamperes.

No. 2 flood light, 28 microamperes.

No. 3 spotlight, 31 microamperes.

Notwithstanding the greater relative illumination of the cluster light, it offered certain objectionable features which made the spotlight type seem preferable. A qualified physicist (8) was called into consultation. He expressed the opinion that the spotlight type came the nearest to fulfilling our requirements in abstract, but that the particular spotlight used for our experimental work was inadequate and that the engineers who devised it had attempted to incorporate several principles and had succeeded in none. His verdict was that this light did not furnish enough illumination. Correspondence with the manufacturers of this illuminator resulted in the production of a modified type which housed a 75-watt clear tungsten lamp and a holophane lens of the so-called "daylight" type. This modified spotlight, hereafter designated as no. 4, was used in the succeeding tests in lieu of the no. 3 spotlight which housed a 50-watt inside frosted lamp.

The first series of lighting tests merely demonstrated the self-evident fact that four 100-watt lamps gave more light than a lesser number at a given distance, but we doubted that the light was reaching the posterior of the oral cavity. Our problem therefore was to design a device wherein could be simulated conditions found in the mouth and the light entering measured.

A box was constructed (see illustration) which permitted a photronic cell to be placed at the approximate working depth on a molar tooth in the mouth and an orifice about the size of the mouth was cut in the box to permit illumination. The photronic cell was placed in the box and the intensity of illumination reaching the cell through the orifice of the box was measured with the lighting units at distances of 18 and 24 inches, respectively, from the orifice. Almost a reversal of the amounts of illumination given by the different types of illumination was found when compared with the previous tests for relative intensity of illumination. These tests showed:

No. 1 cluster light, 84 foot candles at 18 inches.
No. 2 flood light, 60 foot candles at 18 inches.
No. 4 spotlight, 213 foot candles at 18 inches.
No. 1 cluster light, 63 foot candles at 24 inches.
No. 2 flood light, 37 foot candles at 24 inches.
No. 4 spotlight, 126 foot candles at 24 inches.

It is not uncommon to hear persons, even eye specialists, say there is too much light in a room (2). There is now reason to believe that such fears are groundless, as the result of the investigations extending over many years by Luckiesh and Moss (2–3). When the factors of preventable glare and reflection are taken into consideration and adjustments made in conformity with good "seeing" practice, too little rather than too much light is the complicating problem.

The foregoing results indicate that the spot light, of the type used in our investigations, gave the most illumination in the mouth. With adequate illumination of the oral cavity shadows may be minimized by assuming different working positions. Other experimental data and clinical use indicate that the spotlight gives a minimum of direct and reflected glare.

Almost equally as important as the amount of light projected into the mouth is the amount reaching the eyes of the operator, or in the range of vision while at work or at rest. To determine the amount of direct illumination reaching the eyes of the operator under working conditions with the various types of illuminators, tests were conducted with the photronic cell in the position of the eyes of the operator with the illuminating unit directed at the mouth (see illustration). The amount of light reaching the photronic cell, depending on the position assumed by the operator, was:

101 to 253 foot candles with cluster light.
17 to 43 foot candles with flood light.
2.3 to 3 foot candles with spotlight (with adjustable shield in position).

Clinical use of no. 4 spotlight by three experienced dental officers confirm the experimental data given above. In their opinion, this

light has qualities superior to those of any type previously used by them and is satisfactory for visual acuity.

"Lighting for serious visual tasks is usually best when it consists of localized lighting for the specific visual task, and general lighting for general seeing throughout the surroundings" (2). General illumination of the dental operating room should be sufficient for visual comfort with precautions against preventable glare from light sources and from shiny surfaces. For general illumination, it is believed that 10- to 20-foot candles of light on the bracket table and working surface of the dental cabinet is sufficient for good seeing. A dull-finish paint of a neutral color is preferable to enamel or white paint for the walls of operating rooms (4–5). Barber (6) has suggested the following formula:

White lead	pounds	14
Turpentine	pints	3
Lampblack	ounce	1
Raw oil	pint	1
Dryer	gill	1
Venetian red		Trace

The amount of red used should give a scarcely perceptible tint. He points out that too much red gives a lavender tint which is undesirable. The ceiling is finished in white. A semi-dull finish for walls and ceiling is desirable and may be obtained by stippling.

The reflection factor from a white surface may be as much as 80 percent, whereas that from a dark grey surface may be as little as 8 percent (2).

Tests conducted by us indicate that white towels draped over the patient reflect almost twice as much light in the eyes of the operator than do towels of a neutral grey color. This glare almost constantly within the range of vision of the operator was reduced by the substitution of grey towels reflecting less than 45 percent of the amount of light reflected by white towels. Towels of a neutral grey (7) color similar to those used in these experiments will be issued for dental use in the Navy.

Specifications have been drawn up for the purpose of securing a dental illuminator for use in the Navy, embodying the salient features of spotlight no. 4, which was found to be the most satisfactory for illuminating the dental operating field and affording maximum visual acuity.

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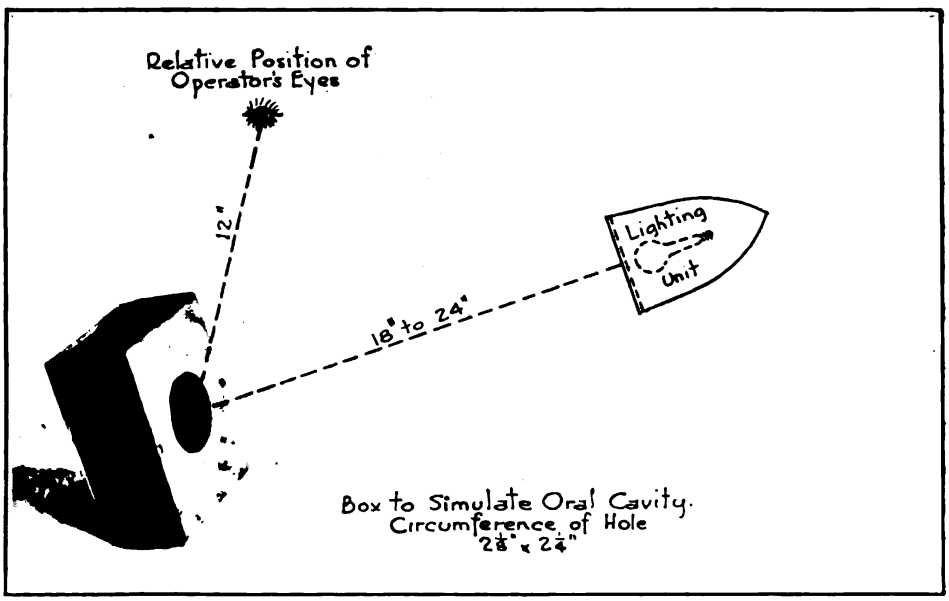

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STUDIES OF ACTIVE PNEUMOCOCCUS IMMUNITY

III

THE DURATION OF TYPE I PNEUMOCOCCUS IMMUNITY[1,2]

By DAVID FERGUSON, Lieutenant Commander, Medical Corps, United States Navy

In previous reports the subject of active pneumococcus immunity was reviewed and satisfactory degrees of immunity were shown to have been produced in experimental animals (1932); the height of the immunity response was measured in three human volunteers and the amount of immunity against type I pneumococcus was calculated in terms of Felton's units (1934).

This report records the degree of immunity measured at intervals over a period of 13 months, which followed the injection into three men of a specially prepared and specially preserved type I pneumococcus vaccine.

HISTORICAL

The work of Wright and Lister in South Africa (1913 and 1917) and Cecil and Austin in this country (1918) has demonstrated that a satisfactory active immunity follows the subcutaneous injection of pneumococcus vaccine in man. The duration of this immunity has never been determined, but it is generally supposed to last only a short time, possibly 4 months.

Recently, Ross (1934) in New York, and Felton (1934) in Boston, have shown that active pneumococcus immunity persists for over a year.

THE PNEUMONIA PROBLEM IN THE NAVY

The pneumococcus problem confronting the Navy is identical with that which obtains during war-time mobilization. Records of the Bureau of Medicine and Surgery show the incidence of lobar pneumonia among recruits is approximately twice that occurring in veteran personnel. The records further show that this increased inci-

[1] Read at staff conference, United States Naval Hospital, Chelsea, Mass., Nov. 1, 1934.
[2] Received for publication Dec. 13, 1934.

dence does not occur at naval training stations where hygienic conditions, including those relative to overcrowding, are very satisfactory.

The greatest incidence of pneumonia occurs when the recruit is sent to sea and subjected to the overcrowding which unavoidably exists on the present day man-of-war. Therefore there is always present this factor tending to produce military ineffectuals where they are least desirable. During military mobilization the same overcrowding of recruits and change to an unaccustomed environment tend to produce pneumonia in epidemic proportions.

Immunization of recruits against type I and II pneumococcus should reduce the incidence of pneumonia 66 percent because of the curious age predilection of the type I organism. While type I pneumococcus is responsible for 25 percent of all lobar pneumonia, it is responsible for 45 percent of the infections occurring in the decade of 10 to 20 years and for 43 percent of the infections occurring between the ages of 20 and 30. The type II pneumococcus is responsible for about 22 percent of the infections occurring between the ages of 10 and 40.

While it is true that the type I and type II antibody solution concentrated by the method of Felton is a most efficacious and satisfactory form of therapy when given early in the disease, yet not only is the preparation very expensive but the patient in the short incubation stage of lobar pneumonia is undoubtedly a highly dangerous source of infection to all contacts.

METHODS

The blood serum of three volunteers, A, B, and C, was measured to determine its protection power against type I pneumococcus before and at intervals after the subcutaneous injection of a type I pneumococcus vaccine. This vaccine was of proven antigenic power and the pneumococci were kept in a satisfactory state of preservation by methods reported in a previous study (1934).

Adult white rats and white mice were used. The volunteer's blood was drawn, the serum separated and kept sterile by aseptic precautions. In determining its protection power, 0.2 centimeters of the serum to be tested was injected into the peritoneal cavity of the experimental animal, followed immediately by the prescribed number of lethal doses of type I pneumococcus.

The lethal doses of pneumococcus were made up in decimal dilutions from a highly virulent culture obtained from the heart blood of a mouse. Broth, not saline, was used as the diluent.

Two animals were used for each dilution.

TABLE I.—*Immunization*

Volunteer	Aug. 23	Number type I pneumococci in millions					Oct. 11	Nov. 22	Feb. 6	Apr. 4	June 5	Aug. 22	Oct. 14
		Aug. 23	Aug. 29	Sept. 3	Sept. 7	Sept. 13							
A	10 d 10 d 100 d 100 d	0	50	200	100	200	10,000 s 10,000 s 100,000 s 100,000 s	10,000 s 10,000 s 100,000 s 100,000 s	10,000 s 10,000 s 100,000 s 100,000 d	100 s 100 s 1,000 s 1,000 d	100 d 100 s 1,000 s 1,000 s	1,000 s 1,000 s 10,000 s 10,000 d	10,000 s 10,000 s 100,000 s 100,000 s
B	10 s 10 d 100 d 100 d	50	200	200	100	200	10,000 s 10,000 s 100,000 s 100,000 s	10,000 s 10,000 s 100,000 s 100,000 s	10,000 s 10,000 s 100,000 s 100,000 s	1,000 s 1,000 s 10,000 s 10,000 s	1,000 s 1,000 s 10,000 s 10,000 s	10,000 s 10,000 s 100,000 s 100,000 s	10,000 s 10,000 s 100,000 s 100,000 s
C	10 d 10 d 100 d 100 d	0	50	200	100	200	100 s 100 s 1,000 s 1,000 s 10,000 s 10,000 s 100,000 d 100,000 d	1,000 d 1,000 d 10,000 d 10,000 d	1,000 s 1,000 s 10,000 d 10,000 d 100,000 s 100,000 s	....	100 s 100 d 1,000 d 1,000 d 10,000 d 10,000 d	100 s 100 d 1,000 d 1,000 d 10,000 d 10,000 d	1,000 s 1,000 s 10,000 s 10,000 s 100,000 d 100,000 d
Decimal dilution:													
Controls no. 6	1 d, 10 ld	.....	.....	.....	.....	.....	1 d, 10 ld	1 d, 10 ld	2 d, 10 ld	2 d, 1 ld	2 d, 1 ld	3 d, 10 ld	2 d, 100 ld
Controls no. 7	1 d, 1 ld	.....	.....	.....	.....	.....	1 d, 1 ld	1 d, 1 ld	2 d, 1 ld	1 d, 1 s, no ld	1 d, 1 s, no ld	3 d, 1 ld	3 d, 10 ld
Controls no. 8	.....	.....	.....	.....	.....	.....	.....	.....	.....	.....	2 d, 2 s	no ld	2 d, 1 ld

NOTE.—D and s preceded by a numeral indicate respectively that the animal died or survived that number of lethal doses of type I pneumococcus. Ld = lethal dose.

THE IMMUNITY TITRE OF THE BLOOD SERUM FOLLOWING ACTIVE PNEUMOCOCCUS IMMUNIZATION (TYPE I)

Table I is a record of the protection power of the blood serum of three volunteers before immunization in August 1933 and the record of their immunization and the protection power of their serum at the following intervals after immunization: October 1933, November 1933, February 1934, April 1934, June 1934, August 1934, October 1934.

This immunization response is shown graphically in chart I. The immunity titre rises abruptly following immunization, and an irregular, but very definite degree of immunity persists.

An examination of the chart I and table I shows that volunteer B received about 750,000,000 type I pneumococci (50 percent more than the other volunteers) and his blood serum has consistently given protection against more than 100,000 lethal doses of type I pneumococcus.

A and C each received about 500,000,000 type I pneumococci, yet the protection power of A's serum has been consistently higher than that of C.

TABLE II.—*Tests of Apr. 4, 1934*

Volunteers	Blood serum 0.2 cc mixed with the number of lethal doses type I pneumococcus as shown
A	100,000 s
	100,000 s
	1,000,000 s
	1,000,000 s
B	1,000,000 s
	1,000,000 s
C	10,000 s
	10,000 s
	100,000 s
	100,000 d
Decimal dilution:	
Controls no. 8	2 d, 1 ld
Controls no. 7	3 d, 10 ld
Controls no. 6	2 d, 100 ld

NOTE.—S indicates animal survived; d that animal died; ld=lethal dose.

Table II records an experiment in which the blood serium and prescribed number of lethal doses were mixed in the same syringe and injected into the peritoneal cavity simultaneously. It is to be compared with tests of the identical samples of sera shown in chart I under date of April 4, 1934.

By this method the protection power of the serum is distinctly higher, but the results cannot be evaluated in terms of the pre-immunization titre obtained by the former method.

This second method was considered but was not employed at the beginning of this study because this test of immunization was

designed to be as severe as possible and *in vitro* effects were not to be confused with *in vivo* results.

The early immunity conferred upon A and B was calculated to be in excess of 1,000 Felton units of pneumococcus antibody, and in the case of C to be in excess of 100 Felton units. As 100,000 Felton units will cure an active case of type I pneumonia, it is conceivable that the immunity induced is capable of preventing the development of type I pneumococcus infections. Whether or not this is true could be determined by one of two methods.

The first method would be by utilizing an experiment recorded in the 1932 report. Rabbits were immunized by the subcutaneous administration of a satisfactory type I vaccine and were then inoculated through a fine catheter passed into a primary bronchus. These

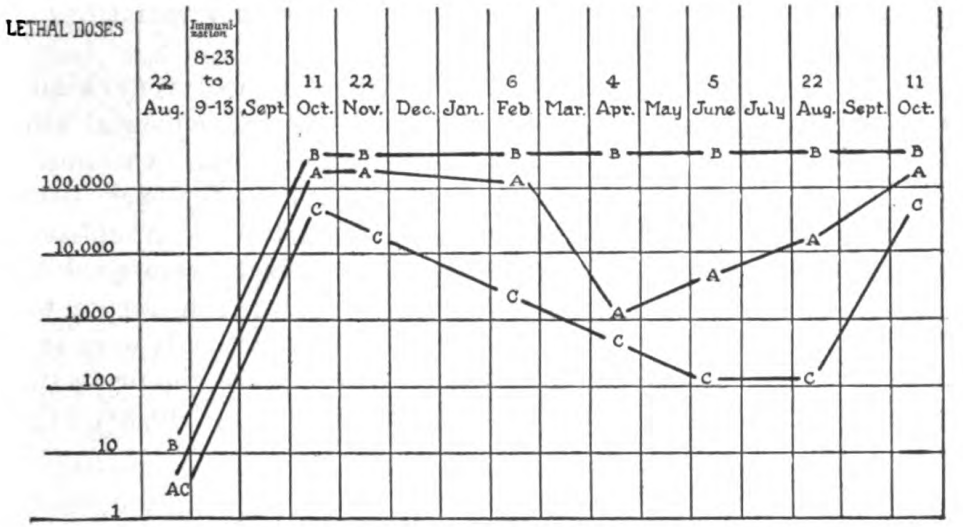

CHART I.

rabbits all survived. X-rays showed some of these rabbits developed a transitory pulmonary consolidation; others did not.

This experiment could be repeated and the protective power of the rabbit's serum could be determined by the technique described under "Methods." This resulting immunity could be calculated in terms of Felton units, and a correlation could then be made concerning the quantity of Felton units necessary in the blood serum to protect against 10,000 or 100,000 lethal doses of homologous pneumococcus.

The second method would be the immunization of all recruits entering the Navy with type I and type II vaccine. After a 2-years' experience the Navy's incidence figures for lobar pneumonia would show whether the calculated minimum benefit of 65 percent improvement had been realized.

COMMENT

Irregularities are present in the curve of A's and C's protection titre and two tests of C's serum were so unsatisfactory that it was impossible to evaluate the results. These irregularities were undoubtedly due to the following defect in the technique employed: The blood serum to be tested was first injected into the peritoneal cavity and the prescribed number of lethal doses of pneumococcus then injected with a separate syringe and needle.

Undoubtedly, if the serum came into direct contact with the pneumococcus in the peritoneal cavity, better protection would result than if both elements were absorbed separately.

The irregular curve of the immunity titre of the serum of two of the volunteers is difficult, or impossible of explanation. It is well known that laboratory animals show individual variations of susceptibility to infection.

Dr. L. D. Felton of the Harvard Medical School very kindly examined the immunity response. From his vast experimental work he had protocols to show that accurate deductions from experiments were impossible when only two animals were used for each lethal dose. He has very kindly permitted me to quote two unpublished protocols. They show that when only two animals were used the results indicated that the protection power of a certain serum was less than 100 lethal doses. When 10 experimental animals were used for each decimal dilution of the pneumococcus culture, the protection power of the same serum was shown to be in excess of 10,000 lethal doses.

SUMMARY

Three men were immunized with a properly preserved type I pneumococcus of known antigenic power. Their blood serum was tested for protection against homologous pneumococcus at 7 intervals over a period of 13 months following immunization. The protection titre of their serum is shown.

CONCLUSIONS

I. Active immunity following type I vaccination has been shown to continue for 13 months.

ACKNOWLEDGMENTS

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To Drs. Felton and Jones and their technical assistants, I am deeply grateful for the use of equipment not available in our laboratory.

It is also a pleasure to express deep appreciation for the interest shown in the work and its possibilities by F. H. Farrell, pharmacist's mate, second class, United States Navy, and E. S. Allen, pharmacist's mate, third class, United States Navy, who served as volunteers for the immunization experiments recorded in this report.

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ENCEPHALOGRAPHY WITH REPORT OF CASES[1]

By P. T. Crosby, Lieutenant Commander, Medical Corps, United States Navy

When this paper was read before a staff conference it brought forth the suggestion that it might be of use to many medical officers in our outlying hospitals where there may be but little access to the literature on so specialized a subject. Should further reason be needed for its appearance in the Bulletin, we may offer those that the matter has not, so far as known to this writer, been previously treated in the Bulletin and that it is a method of wide application where head injuries are frequent, as in naval life.

Following head injuries, there is frequently a train of obscure symptoms which cannot be evaluated or treated successfully without detailed knowledge of their underlying pathology. Signs and symptoms fail to localize the focal points. It is in this class of cases that ventriculography, and, better still, encephalography hold out to us the best avenue of diagnostic approach, especially as concerns the cortex or brain surface lesions.

Post traumatic headache, amnesia, confusion, clouding of consciousness, hypochondriacal behavior, marked change in personality and mood, epileptiform seizures, and obscure hysterical complaints are some of the subjective phenomena which are sequelae of head traumata. These may or may not follow known fractures.

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[1] Read before Staff Conference, United States Naval Hospital, San Diego, Calif., Sept. 20, 1934.

Pneumography is a method of X-ray examination of the contents of the skull after replacing spinal fluid with air.

Ventriculography is the replacing of the fluid within the lateral ventricles of the cerebrum with air, followed by roentgenograms of the result.

Encephalography (subarachnoidography) is the replacement of all the spinal fluid with air by lumbar puncture, followed by the X-ray examinations of the result.

First, let us review the technical steps of the method.

The patient is previously prepared by enema and by hypos of scopolamine and morphine. No breakfast. He is seated on a stool, leaning over a table. The usual lumbar puncture is made except that 2 needles are inserted, 1 above the other; for example, 1 in the fourth and 1 in the fifth interspaces. To the upper needle is fastened a 3-way pet-cock. The stylet is withdrawn and after fluid is shown to be flowing freely, the flow is turned off. To the upper opening of the pet-cock, an Ayer spinomanometer is attached and held in place by an assistant. The pressure reading is taken and recorded. The lower needle needs only to show that spinal fluid flows freely. From the latter and into a graduated container the fluid is collected to the amount of 10 cubic centimeters. The pressure is again recorded at the upper needle. If the pressure remains the same or falls slightly, we are free to proceed. A 50-cubic centimeter syringe is attached to the upper needle and about 2 or 3 cubic centimeters of spinal fluid withdrawn. This amount is sufficient to lubricate the inside of the syringe and helps to seal it air tightly. Now, the syringe is filled with room air, sucked in through a pad of sterile guaze. After each 10 cubic centimeters of fluid have been withdrawn from the lower needle, 10 cubic centimeters of air are injected via the upper but no pressure is used; just enough to slide the plunger along the barrel.

At the end of each 30 cubic centimeters of withdrawn spinal fluid the pressure is taken. The amount of air substituted for the fluid is gradually injected to keep the pressure within normal limits but should never be allowed to exceed the initial pressure.

The fluid from the lower needle is controlled by leaving the stylet in place but partially withdrawn, so that fluid will not escape too quickly, thereby permitting the intracranial pressure to fall too fast or too low. The whole procedure is done slowly and deliberately. When 90 to 150 cubic centimeters of fluid have been withdrawn and have been replaced by not quite as much air, the patient is ready for X-ray. During the latter half of the maneuver the patient's head is slowly rotated forward and backward and laterally to facilitate the emptying of the lateral ventricles.

The patient is now presented to the X-ray in the erect sitting position. With use of the Bucky diaphragm the conventional AP, PA, right and left lateral, and one of lateral steroscopic plates are made. In accomplishing the AP or PA films, the head is so tipped that the straight rays will pass longitudinally through the lateral ventricles but will be above the frontal sinuses.

After the X-ray examination the patient is returned to an ether bed with its foot on shock blocks. The after care is much the same as in the routine surgical case following ether.

During the replacing of fluid by air, patients usually become pale, sweat profusely, sometimes vomit, and complain of marked frontal headache. If blood accompanies the spinal fluid, pointing to intracranial hemorrhage, the procedure is stopped at once.

Now let us consider the cisterns or spaces which appear on the X-ray plates and which are normally seen. Some of these are distorted, displaced, or disappear under varying conditions.

1. The cisterna longitudinalis cerebri (or collosal sulcus) lies just above the corpus collosum and beneath the falx cerebri. It is not seen in the lateral view except when the lateral ventricles are filled by mass lesions or are collapsed. In these events the air follows from the pontine cistern by way of the interpeduncular cistern and along the posterior cerebral artery. In the event of filling of the third or lateral ventricle by a tumor mass, this cistern is arched upward.

2. *Sulcus cinguli.*—This lies above the cisterna longitudinalis and on the inner aspect of the cerebrum. It appears in cortical atrophy.

3. *The fifth ventricle.*—This is an anatomic variant and is found when the columns of the fornix and septum pellucidum are separated, forming a small space between them and appearing over the third ventricle in the plate.

4. *Insula* (*island of Reil*).—In marked cortical atrophy it is seen in AP views.

5. *Cisterna vena magna cerebri.*—This cistern is formed by the tentorium above and the cerebellum below. It extends along the great veins from the cisterna ambiens posteriorly and somewhat laterally.

6. *Cisterna ambiens* (intercommunicans) lies under the posterior end (splenium) of the corpus collosum and about the pineal gland, being bounded below the corpora quadra-gemina.

7. *Cisterna chiasmatis.*—This space surrounds the peduncle of the pituitary body and lies beneath the optic chiasm.

8. *The cisterna interpeduncularis.*—This space lies behind the sella and between the crura cerebri. It is connected with the cisterna ambiens by small communicating sinuses or cisterns around the borders of the crura.

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9. *The cisterna pontis.*—This space is anterior to the pons and connects below with the cisterna magna.

10. *The cisterna magna.*—This space lies below the cerebellum, behind and above the pons and medulla and is connected with the fourth ventricle by the foramen of luschka and magendie. It is freely connected with the subarachnoid space above and below.

11. *The fourth ventricle* is just superior to the cisterna magna and lies beneath the cerebellum and above the pons. It is connected with the third ventricle by the aqueduct of sylvius.

12. *The third ventricle.*—This space lies beneath the corpus collosum in the mid-line somewhat medial to the lateral ventricles. It is best seen in the stereoscopic plates. It is distorted laterally by the middle commissure and the optic thalmus. In AP view it appears as a small circular space. It is connected with the lateral ventricles by the foramena of Munro.

13. *The lateral ventricles.*—These spaces are the largest of the cisterns, and lie within the cerebral hemispheres. In the AP views they form the conventional "butterfly." In the lateral views their fullest extent is better shown. Distortion or displacement of these spaces is of great diagnostic moment. It is often very difficult to fully drain all the fluid from these cisterns. Therefore, their absence is more often due to faulty technic than to pathology.

14. *The cortical pathways or cortical sulci.*—These spaces are often potential and appear usually either as the result of back pressure from below or because of atrophy of the cortex. These cortical markings may be seen over the frontal and temporal areas but never over the parieto-occipital region, except when due to cortical atrophy. Large bodies of air found over the surfaces of the cerebrum are due either to external hydrocephalus or aplasia.

In 1918 Dandy was the first to use ventriculography and in 1921 Bingel and Dandy were the first to use the lumbar route for encephalography. Since then Dandy as well as many others have developed this method of diagnosis. Recently a committee was appointed to standardize encephalography. Dr. Henry Pancoast was named chairman and reported, in substance, the following criteria to be considered normal encephalographic appearances.

A. Subarachnoid or cortical markings are normally 1–3 mm in width and are seen over the cortex except in the posterior temporal region and never over the whole occipital lobe.

B. Depending upon how well the spinal fluid has been drained, the basal cisterns are visible.

C. The ventricles appear in proportion to the drainage also. The third ventricle is always fainter than the laterals. If there is no distortion, nonfilling of these spaces is due to faulty technic.

Indications for encephalography.—1. All cases in which the symptoms are obscure following trauma, inflammation, senility, hemorrhage, tumors, hemiplegias, and birth injuries.

Contraindications.—1. A spinal fluid pressure of over 20 mm Hg in horizontal position.

2. Arteriosclerosis.

3. Meningo-vascular syphilis.

4. Mass lesions or tumors in posterior fossa (subtentorial).

5. However, in some cases of choice, the spinal fluid pressure may be reduced by hypertonic solutions or the pressure carefully reduced on the puncture table prior to encephalography.

Uses approved by the committee.—For the diagnosis and localization of obscure intracranial pathology, it is to be used only after careful neurological measures have failed to point to a definite pathology. This also includes all laboratory procedures.

Let us add here that it is believed by many workers that following trauma there results a localized adhesive arachnoiditis. Fine adhesions are formed and not only block the fluid pathway but irritate the cortex. The air opens these pathways and separates the adherent arachnoid membranes.

Post traumatic headaches and other obscure symptoms related to the intracranial contents are believed due to this type of lesion. It has been commonly found that following encephalography these symptoms disappear. It has been further noted that following encephalography epileptics are free from seizures for long periods of time.

Pneumography exhibits many complications which although not malignant are, to say the least, spectacular and frightening. The graver complications are sudden cerebral hemorrhage, foramenal hernia, and shock. The lesser ones are severe frontal headaches, hyperpyrexia, profuse perspiration, vasomotor depression, and nausea, sometimes with vomiting.

Let us now recapitulate the pathological conditions demonstrated by encephalography.

1. Atrophy of the brain. Subdivided into:

(A) Atrophy of external hydrocephalus.

(B) Atrophy of internal hydrocephalus.

(C) Atrophy of combined external and internal hydrocephalus.

(D) Atrophy following thrombosis.

(E) Idiopathic atrophy. Cause not determined but first noted in advance stage.

2. Aplasia.

3. Arachnoiditis.

4. Porencephaly.

5. Tumors or mass lesions.

External hydrocephalus is that condition which shows atrophy of the cortex or brain substance with resulting large areas of air-filled spaces between the cortex and the cranial walls with no or little distortion of the ventricles.

Internal hydrocephalus is that condition in which the ventricles themselves are generally distorted with little increase in surface pathways.

Combined hydrocephalus is as the word means, a combination of the two above conditions.

Aplasia is a deficient development of the brain beginning at birth or early childhood.

Arachnoiditis is a traumatic reaction in which adhesions form between the layers of the arachnoid matter or between the three meningeal membranes. It may be local or general. When well advanced it tends to pull the outer walls of the ventricle toward the lesion in much the same manner as scar tissue.

Porencephaly is a well-defined funnel-shaped brain defect which extends from the cortex surface toward or into a lateral ventricle. If connected with a ventricle it may empty and fill with air.

Tumors or mass lesions tend to distort or obliterate normal cisterns or surface markings.

Although ventriculography and encephalography are intimately associated, nevertheless we feel these two should be differentiated in order that, as a method of choice, one may be indicated. Let us compare them from this standpoint. We shall be forced to repeat some of the data discussed previously.

Ventriculography.—1. *Technic.*—Here there are made two small trephine holes a few centimeters (normally 3 centimeters) above and lateral to the occipital protuberance. A needle is introduced into the posterior horn of each lateral ventricle, withdrawing the fluid and injecting air. Patient is in the semierect position.

2. *Indications.*—Unlocalized intracranial mass lesions with spinal pressure above 20 millimeters Hg.

3. *Errors in technic.*—Rotation of head at time of operation or during X-ray examination. Mass lesions in the occipital lobes displace or block approach to the posterior ventricular horns.

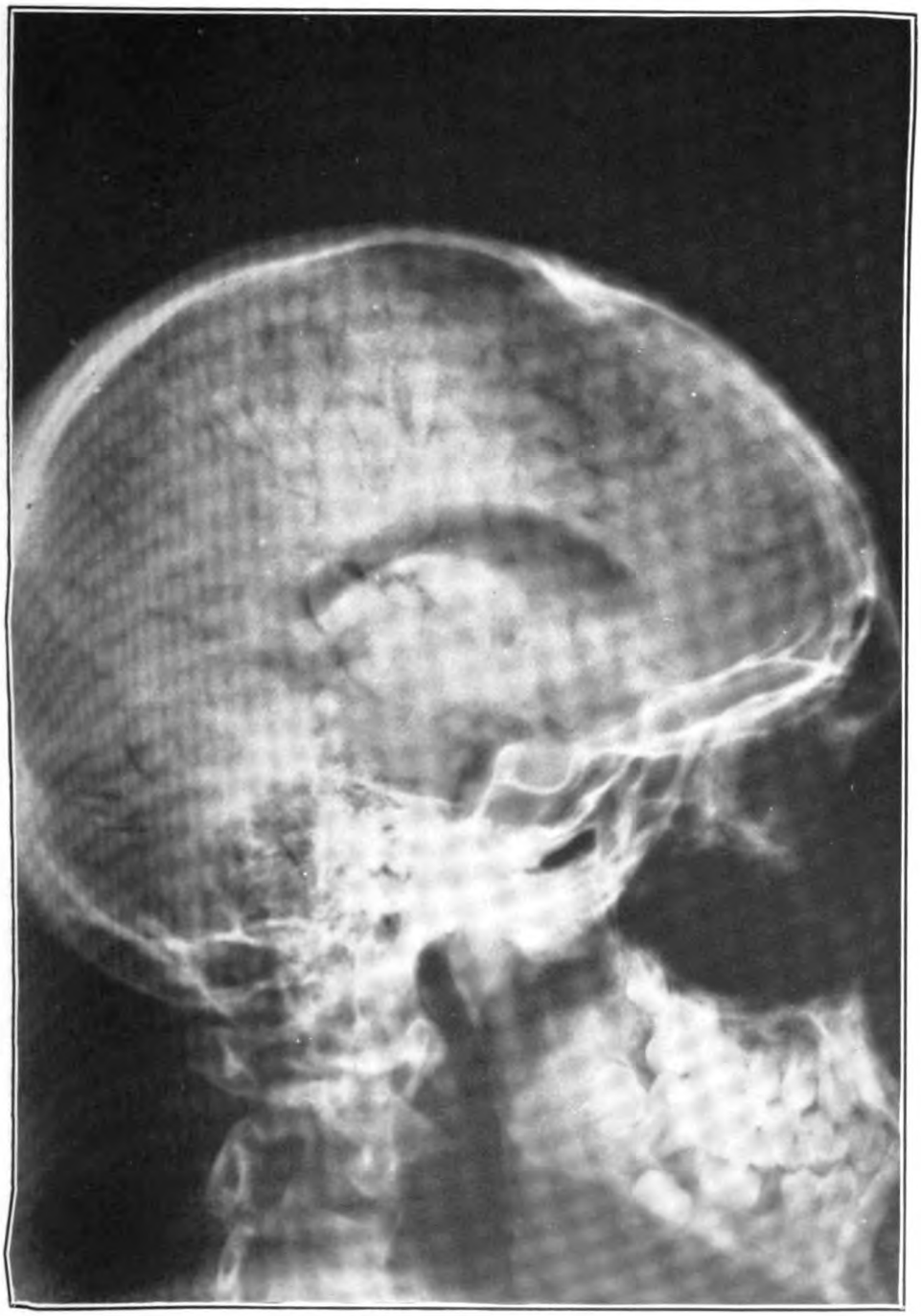
4. *Complications.*—Sudden death. Hemorrhage due to needling the lesions or due to sudden collapse of the ventricle. Mortality 8 percent.

Advantages of ventriculography.—A. Ventricular system is better outlined.

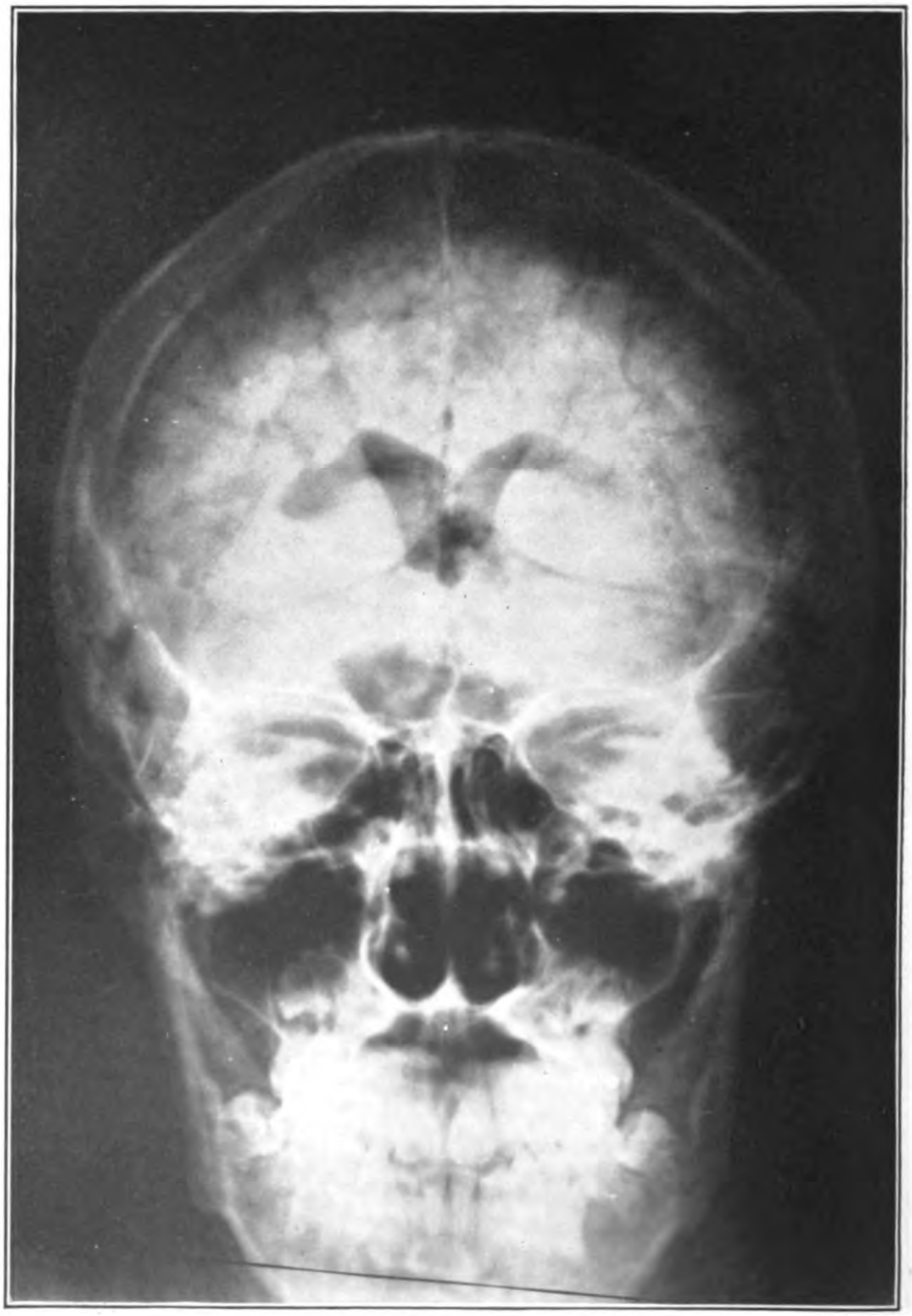
B. Brain or mass lesions are localized.

Advantages of encephalography.—A. The procedure is simple without need of operating room.

B. Little shock results.



LEFT LATERAL VIEW



ANTERIOR POSTERIOR VIEW.

C. Mortality of 0.25 percent.

D. Whole brain and stem visualized.

E. Surface markings and ventricles visualized.

F. Better localization of mass lesions.

G. Post traumatic headache relieved.

Disadvantages of ventriculography.—A. Requires operating room technic.

B. Hemorrhage and shock are probable factors.

C. Mortality of 8 percent.

D. Failure to find ventricles is frequent.

E. No spaces shown except the ventricles.

Disadvantages of encephalography.—A. Cannot be used in posterior fossa lesions (subtentorial).

B. It is dangerous in spinal fluid pressures of over 20 millimeters Hg.

C. Posterior and inferior horns of the lateral ventricles are most difficult to drain.

CASE REPORTS

CASE 1.—D. C., seaman, first-class, admitted April 6, 1934, age 33 years, service 13 years.

Health record abstract. Record of admissions:

Vincents angina, February 29, 1928, 5 sick days.

Contusion, head, February 25, 1930, 5 sick days.

No disease (observation epilepsy) June 14, 1930, 40 sick days.

No disease (observation epilepsy) May 7, 1932, 54 sick days.

Epilepsy, August 17, 1933, 182 sick days.

No disease (observation) February 15, 1934, 117 sick days.

The family history is negative.

The past history as stated by the patient, his brother, and letters from family physician who was present at patient's birth, show no apparent birth injury or evidence of any type of convulsive seizure or fainting spells prior to 1930. We, however, learned that the noticeable asymmetry of the patient's cranium has been present during life; further that at the age of 11 years the patient was kicked in the left side of face by a mule. He does not recall many details of the accident but does admit that he was unconscious for several hours. No sequelae apparently followed this injury.

The patient completed the eighth grade in school and then went to work.

After enlistment for a period of about 8 years all evidence of seizures is denied until shortly after his admission for head contusions in February 1930.

This injury resulted from a fall while walking across the deck of a tug. He fell, striking his head against the wooden gunwale. He was unconscious but we are unable to ascertain definitely whether he became unconscious just before the fall or as a result of striking his head. The chart of his admission shows no apparent injury or wound. He was observed for 5 days and returned to duty. No X-ray or other laboratory procedures are recorded.

His next convulsive seizures were on the nights of June 13 and 14, 1930. He was again hospitalized. He stated that 3 weeks previously he had awakened at night to find his whole body twitching. He denied convulsive seizures prior to this one. The physical, X-ray of skull, urine, B. M. R., E. C. G. examina-

tions were normal. There were no further seizures and patient was returned to duty on July 25.

Since then there have been a few seizures noted by witnesses and followed by hospitalization but as all examinations have been negative and as he exhibited no convulsions during these periods of observation, he was returned to duty. At one hospital it was believed that the seizures followed alcoholic excesses.

This patient came under our observation on April 6, 1934. Again our neurological, physical, spinal fluid, and skull X-ray examination were negative except that a definite asymmetry of the left side of cranium was noted. It was also noted that the patient was left handed. Spinal fluid pressure 180 mm water. Queckenstedt negative.

Believing that there must be some cortex pathology present and suspecting a hematoma or other mass lesion involving the cortex but not causing localizing signs or intracranial pressure increase with papilledema, the patient was transferred to the surgical service for ventriculography, which was done on June 14, 1934. At the time of this operation, no fluid was obtained from the left ventricle, which was considered obliterated. Forty cc of spinal fluid was obtained from the right ventricle. The X-ray plate did not show either ventricle.

Patient made an uneventful recovery and was returned to the neuropsychiatric service for further study.

As subsequent neurological examinations including eye grounds were consistently negative it was decided to accomplish encephalography. This was done on July 19, 1934.

The X-ray report of these encephalograms is as follows: The anterior posterior views show ventricles to be small, compressed, and displaced to the right of the midline. There is marked hydropneumocephalus left side. No cortex can be seen above or behind fluid level. Third ventricle not seen.

The steroscopic left lateral view shows anterior and posterior horns of the lateral ventricle to be compressed. No cortex is distinguished above fluid level.

As a result of these findings, a diagnosis of external hydrocephalus was made. We are unable to determine from the history whether it is due to an unknown birth injury, the kick of the mule, the fall on the tug, or if it is a true aplasia. As no seizures had been witnessed until June 1930, we felt that the hydrocephalus and cortex atrophy were due to injuries and therefore acquired.

This case is a good example of the advantage of encephalography over ventriculography. Had the latter been successful, it would not have revealed the true pathology other than that the lateral ventricles were within normal limits.

Case 2.—W. M., Torpedoman first class, admitted June 27, 1934, age 32 years, service 15 years.

Health record abstract. Record of admissions:

August 1919 to June 1921, none.

Gonorrhea infection urethra, November 1921, 32 sick days.

Gonorrhea infection urethra, June 1925, no sick days.

Undetermined (fracture skull) May 25, 1930.

Fracture skull (right temporal) May 26 1930, 82 sick days.

Otitis media chronic, March 25, 1931, 47 sick days.

Catarrhal fever acute, November 1932, 7 sick days.

Wound lacerated scalp, February 13, 1934, 28 sick days.

Epilepsy, March 13, 1934.

The family history is negative and irrelevant.

The past history shows no seizures previous to 1930. He completed the fifth grade in school. No relevant past diseases or injuries.

On May 26, 1930, he was admitted to the hospital, unconscious. He had been in an automobile accident the night before. Right eye was swollen with paralysis of R. sixth nerve. X-ray revealed a fracture of the squamous portion of the right temporal bone extending up over the vertex to the left side of midline. Blood alcohol at time of admission was 1.5 mg per cc, W. B. C. 12,200, 72 percent polys.

The patient received intravenous hypertonic solutions.

Eye grounds were negative. Still comatose on May 28 but at times asked for water.

On June 12 he was considerably improved.

On July 7 he complained of ringing in left ear. This was found to be due to drum retraction.

On August 15 he was returned to duty well.

On February 13, 1934, while on a submarine in Cavite, P. I., he was struck by a falling tackle in the torpedo room. The blow struck him on the occipital region, producing a long laceration and causing unconsciousness. Upon admission to the hospital he gave a history of 3 or 4 convulsions following his previous automobile injury in 1930. On February 14 he had a mild seizure of epileptiform nature. All physical, neurological, and X-ray examinations were negative and he was considered ready for duty. However, before he was discharged he suffered a typical epileptiform seizure. The diagnosis was changed to epilepsy and he was surveyed to the United States. At a naval hospital he was observed and examined but because of no seizures while under observation and no signs of pathology, he was returned to duty on June 6, 1934.

The patient reported aboard a destroyer and 6 days later had a fainting spell. A convulsive attack is not described. He was then sent to the San Diego Naval Hospital.

Here we failed to find any gross indications of pathology. The neurology is negative except for washed out facies and some indefinite tremors. The mental reactions are slow and intelligence level low but well above a fifth-grade education. Spinal fluid negative. Dynamics normal. Pressure 190 mm water. Queckenstedt negative.

He complained of paraesthesia over site of last injury, together with a feeling of fullness under and behind both ears. At times he appears somewhat confused. Admits to headaches when fatigued, and inability to get to sleep within 1 to 3 hours after retiring.

As there were no outstanding signs or symptoms other than the convulsive seizures and the history of trauma, an encephalogram was accomplished.

The X-ray report of these encephalograms is as follows: The anterior, posterior, and lateral projections of the skull show right lateral ventricle to be somewhat larger than left. Third ventricle enlarged. Fourth ventricle filled. The convolutionary markings are well shown throughout the left frontal and parietal areas. Over the posterior portion of the right parietal area there is an absence of air with localized increased density.

As a result of these findings a diagnosis was made of adhesive arachnoiditis right parietal area, probably due to old hemorrhage followed by organization and retraction.

Again in this case the arachnoidography not only brought to light the pathology but also in view of the subsequent history, furnished a treatment for this pathology. This patient has been completely free from his previous complaints and no seizures or fainting spells have recurred, although he has received no medications and has tried to bring on a seizure by fatigue. How long he will remain symptom free we cannot predict.

Case 3.—M. J. M., seaman, second-class, admitted November 8, 1934, age 18 years, service 7 months (enlisted Apr. 16, 1934).

Fainting attacks. Convulsive movements during attacks were not typical of epilepsy.

Family history negative.

Past history negative up until October 1, 1933, when in a traffic accident he suffered a depressed fracture of the right parietal region. Operated at Berkeley Hospital where a trephine and removal of a portion of the depressed bone was accomplished. About 1 month later he began to have dizzy spells and later fainting spells. Headache is localized about site of injury. On board the *Relief* the neurology was negative except the fanning of the small toes of left foot in the Babinski maneuver. X-ray showed the trephine operation with the outer and inner tables removed. No indication of increased intracranial pressure. This case was extraordinarily well worked up on the U. S. S. *Relief*. In addition, no papilledema was noted and the spinal fluid was negative chemically and dynamically.

He was transferred to the San Diego Naval Hospital on November 8, 1934.

Our findings were identical with those of the medical officers of the *Relief*. We felt that this was a good case for encephalography.

The X-ray report of the encephalograms is as follows: The study of the right lateral projection of the skull, following introduction of 120 cc air, shows an increase in the distribution of air, throughout the cortex, right parietal area, suggesting cortical atrophy. The right frontal area presents a normal distribution of air. The anterior horn of the right ventrical is dilated. The third, fourth ventricles and cisterna are well filled. The interpeduncularis are beautifully shown.

The posterior anterior projection shows the lateral ventricles well filled, but the right is displaced slightly to the left of the midline. The third ventricle is well shown. There is a normal distribution of air throughout the cortical area, left side and throughout the frontal and parietal areas.

Conclusion: Atrophy of the cortex, right hemisphere, parietal area.

Case 4.—C. D. B., private, U. S. M. C., admitted November 26, 1934, age 18 years, service 3 months.

Admission diagnosis.—(Epilepsy) undetermined. While on rifle range he suddenly suffered a cramp in right hand with pain in right arm. Arm became involved in the cramp, and suddenly, in a flash of light, he slipped into unconsciousness. During the unconsciousness he exhibited an epileptiform convulsion.

Family history negative except for broken family and orphans' home.

Past history.—Mumps, pneumonia at 3 years, diphtheria carrier at 8 years.

Auto accident 7 years ago. Head not injured. Another auto accident 6 months prior to enlistment, but again he denies any head injuries. He stated first signs appeared in June 1931, while on fishing trip. Had had several seizures, all of which began in the same manner as described above. A L. M. D. gave him luminal pills which cured him for a period of 2 years prior to enlistment.

Neurology is negative. Spinal fluid negative. Queckenstedt is negative.

X-ray of skull.—The study of the stereoscopic projection of the skull shows evidence of an old stellate, probably depressed fracture of the right parietal bone. There is also evidence of an old linear fracture of the right parietal, which runs parallel to the sagital suture, originating in the right temporal fossa and ending in the neighborhood of the coronal suture. The depression fracture previously mentioned is approximately 2½ by 2½ cm.

Neurology following the encephalography reveals a marked adiadochokinesis and apraxia of the right hand; much decomposition of associated movements of the right hand as in the VN and post-pointing tests. No astereognosis is noted. Subjectively, the patient is conscious all the time that the right hand, arm, and middle fingers of the right hand are numb, and a feeling of impending cramp exists. However, sensory tests are objectively not disturbed.

The encephalograms done on December 7, 1934, are essentially negative, especially the normal distribution of cortical air on the right side. From the clinical findings we suspect strongly a tumor of the left frontal lobe (probably a slowly growing glioma). The eye grounds are still normal.

The patient has suffered but one seizure on the ward, and that on November 26, 1934. He will be kept under observation for additional neurological localizing signs. The apparent Jacksonian aura with each seizure directs attention to the left cerebrum on the opposite side from the skull fractures.

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RECENT PROGRESS IN ELECTRICALLY PRODUCED GAMMA RADIATION[1]

By ALBERT SOILAND, Lieutenant Commander, Medical Corps, United States Naval Reserve

During the past 5 or 6 years scientists have been focusing their attention with constantly increasing interest upon the development and practical application of higher voltage X-ray tubes and apparatus. From a manufacturing point of view, it has been demonstrated that a practical and workable vacuum tube can be produced which will function satisfactorily at potentials of 1,000,000 volts and higher. This accomplishment is, in a large measure, the result of early experimentation conducted by Dr. Lauritsen at the California Institute of Technology of Pasadena, Calif. Dr. Lauritsen and his associates began their work in this direction as early as 1927 and their

[1] Presented before Oklahoma City Clinical Society No. 1, 1934.

first tube to produce X-rays at 750,000 volts was a revelation to the scientific world. It was immediately apparent that here was something of tremendous importance to the radiologist and much untimely headline publicity was broadcast heralding a sovereign panacea for cancer and all neoplastic disease. This was unfortunate, but, no doubt, unavoidable; how many times have we not seen this same thing happen in connection with other scientific developments? The public and the press seem ever willing to jump at premature conclusions. Suffice to say, the actual and practical therapeutic value of supervoltage X-radiation is, to a very considerable extent, yet to be proven. We have high hopes that the future holds results which may perhaps give realization to our expectations.

The clinical tests which were conducted at the California Institute of Technology with a brief history of the immediate results obtained have already been published. The laboratory and developmental work which brought Lauritsen's tube into being resulted quite naturally in other manufacturers investigating along the same lines which has brought forth other good tubes, notably the General Electric and Westinghouse; but so far as we know, the original Lauritsen design is satisfactory. This type tube at the California Institute of Technology is still in operation, as is also one of more recent construction of even higher intensities.

It is not my intention to discuss the engineering or manufacturing possibilities of supervoltage equipment—we already know that tubes have operated and are operating at voltages from 350,000 to 1,000,000. We take it for granted that much higher voltages will be made available in the future.

This is a rapidly advancing electrical age and to think about future possibilities seems irrelevant when compared to the present problem which confronts us. As I see it, that problem is—how can we make available to humanity the maximum therapeutic benefits which can be obtained with the apparatus now in our hands?

The successful operation of X-ray apparatus beyond 500,000 volts cannot be supervised by the medical radiologist alone. In order that the equipment be available for continuous operation with adequate protection to the patient and the administrating personnel, the constant attention of a physicist is imperative. This assures accurate calibration and guarantees to the prescribing clinical radiologist the knowledge that the dosage given will correspond in minutest detail to the dosage prescribed. It is readily apparent, therefore, that any vacuum tube energized by voltages of half a million or more is not a piece of apparatus that can be adequately supervised by the usual clinical technician or doctor of medicine. The possible difficulties and troubles are so numerous and the importance of keeping the tube in constant operation is so paramount that the

resonsibility must, of necessity, rest on the shoulders of a physicist whose training and background equips him for the task. One visit to the control room of a higher voltage therapeutic installation would immediately show the necessity for this type of supervision.

In analyzing the potentialities of higher voltage X-rays, we should not confuse the issue by assuming that this mode of treatment will either supplant or replace radium. It is already apparent, however, that supervoltage roentgen-ray therapy offers a challenge to the radium bomb or telecurie therapy in certain deep-seated conditions. In the case of interstitial radiation, however, we have no substitute for radium made up in highly filtered platinum needles. This is our opinion which also seems to find favor with radiologists in America who have had experience with higher voltage radiation.

To my knowledge, there are now in operation seven of these supervoltage installations in the United States, located at Pasadena, New York City, Chicago, Detroit, Seattle, Lincoln, and in our own plant at Los Angeles, and this rather rapid acquisition of newer and expensive electrical equipment calls forth a brief reference to the economic factors involved. These represent naturally a fairly heavy investment of capital and no one can tell but that the progress of scientific investigation and invention may make them all obsolete before many years roll by, so it would appear that those of us who are convinced that every forward step takes us nearer to the ultimate goal, must be prepared to contribute not only our time and experience to this new science, but our money as well. We can only progress step by step and are convinced from experience that supervoltage X-ray equipment as now designed opens up new fields of clinical investigation for the radiologist. It was only 5 or 6 years ago that Dr. Lauritsen was first experimenting with his large glass tube and only 4 years since we began serious clinical studies; we must admit that time is still too brief to permit of fundamental conclusions. I do feel, however, that those of us who ally ourselves with this supervoltage X-ray movement will be more than repaid for the time and expense necessary to determine its ultimate destiny in cancer therapy.

The experience of our own clinical group, operating a tube at a potential of 550,000 volts, has given us some very definite ideas on the immediate results of supervoltage X-ray treatment, although an estimate of end results would be unwise until a 5-year interval has elapsed. A few of the established facts are, however:

1. With the same filter, the time for a given depth dosage is markedly decreased if compared with standard high-voltage equipment.
2. Nausea and radiation sickness is less than with lower voltages.
3. Resolution of deep-seated malignancies are observed and im-

provements have been noted where the patient had become resistant to lower voltages.

4. A greater proportion of the radiation energy penetrates to the deeper levels of the body, giving a relatively decreased skin reaction.

5. The tube may be provided with sufficient portals to treat from 2 to 4 patients simultaneously.

6. Definite destruction of deeper gland metastases has been observed in post-operative recurrences.

7. Several patients with advanced recurrent malignancies of breast, uterus, prostate, and rectum are symptom free from 1 to 3 years.

TREATMENT OF OBESITY WITH DINITROPHENOL

By Roy J. Leutsker, Lieutenant Commander, Medical Corps, United States Navy

Recent articles by Tainter, Stockton, and Cutting (1) and others (2), (3), (4), on the actions and uses of dinitrophenol have resulted in wide-spread interest in this drug and its possibilities in the treatment of obesity. Undoubtedly large quantities of the drug are being consumed, in some cases under careful supervision and in many cases under no supervision at all.

It has been definitely proven that in moderate dosage it causes a prompt and marked increase in metabolism through direct action on the tissue cells and a definite loss of weight through burning up of the stored fat; if it also can be established that, under careful control its administration is without danger, it will come into its own as a useful and important drug.

In any general group of obese persons there will be a number whose uncontrolled appetites preclude the possibility of success under any weight-reduction regimen. On the other hand there will be some, notably those with depressed metabolic activity, who, in spite of sincere efforts have failed to make a satisfactory reduction in weight. It is in these cases that the aid of dinitrophenol should be enlisted.

Thirty-one cases of obesity have been or are being treated with alpha-dinitrophenol at the United States Naval Hospital, San Diego, Calif. In four of these cases careful records were not kept. In four cases the treatment was terminated for reasons not connected with any action of the drug, before adequate results were obtained. The remaining twenty-three cases have been under closely supervised and controlled treatment for periods long enough to show satisfactory results and provide reliable data. This report is concerned only with these twenty-three cases.

Eleven of these cases have been hospital admissions under hospital control and daily observation while the remaining twelve have

been out-patients. The average weekly loss of weight in the former group was 3.18 pounds as compared with 2.64 pounds in the latter. As will be seen the hospital group were under better dietary control and also under heavier dosage of the drug.

In selecting patients only those who had conscientiously but unsuccessfully tried weight reduction by other means were accepted. Out-patients were required to return weekly for b. m. r., blood pressure and urinalysis. They were given exactly enough of the drug to last 1 week. They were warned as to the possible dangers of the drug and were instructed as to the symptoms of toxicity.

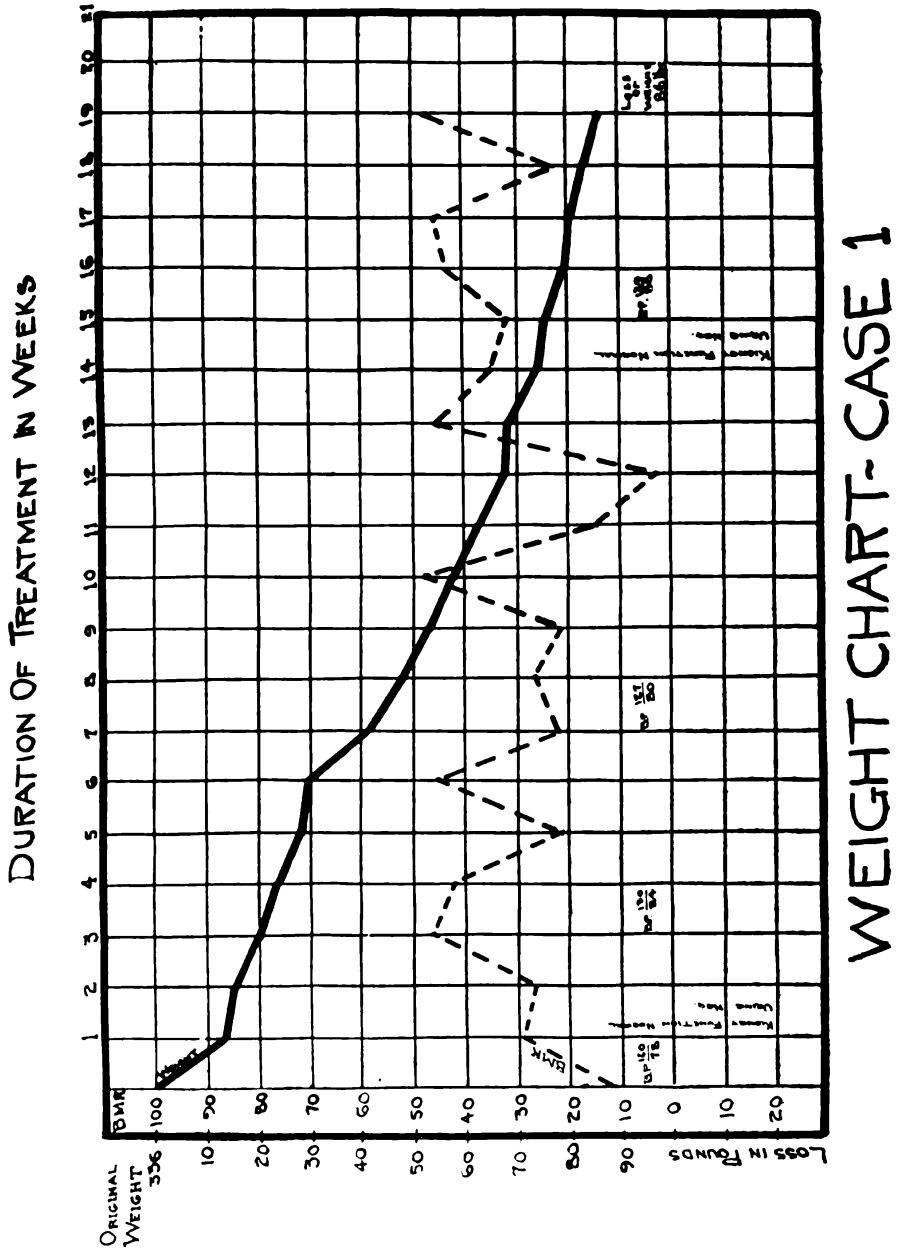
Dosage.—Bearing in mind the possibility of allergic reaction the initial dosage was small and was based more or less roughly on the weight and sex of the patient, 100 milligrams to 300 milligrams daily for the first week. This was then increased until the optimum dose was found, that is one that would cause an appreciable weekly loss of weight without producing unduly unpleasant symptoms of warmth and excessive perspiration. The dosage has never exceeded 400 milligrams daily for out-patients while nine of the hospital cases were kept on 600 milligrams daily for periods up to 4 weeks without apparent harmful effect.

Diet.—The hospital cases were placed on a standard hospital obesity diet averaging 1,700 to 2,000 calories daily. At first no attempt was made to restrict the caloric intake of out-patients. Later, however, a diet regimen was devised, low in fat and starch, which permitted the patients a wide latitude in the selection of food. Such a diet is much less discouraging to the patient and has served the purpose very well.

Reactions.—In none of the thirty-one cases have alarming side reactions been encountered. In four cases the treatment was terminated for reasons not related to any action of the drug. One case complained of loss of the sense of taste which completely returned after 2 weeks' rest. Nearly all the patients have remarked the sensation of warmth and excessive perspiration; when these became too marked for comfort the dosage was reduced. The itching and urticarial eruptions reported by writers and discussed by clinicians have never been encountered.

No effect on blood pressure has been noted except in one case, that of an officer who had been recently retired for arterial hypertension and myocarditis. He was about 40 pounds overweight; his attempts to reduce by diet had been unsuccessful and his physical condition precluded the help of exercise. He had been under this writer's observation for several months prior to the administration of dinitrophenol during which time his systolic blood pressure varied between 180 millimeters and 210 millimeters and his diastolic pressure remained pretty well fixed at 130 millimeters. He was started

at 100 milligrams daily; the second week this was increased to 300 milligrams daily. At the end of the third week he complained of "burning up", excessive perspiration, and general weakness. His blood pressure was found at this time to be 150 systolic and 100 diastolic. Administration of the drug was immediately stopped.



After 3 weeks' rest his blood pressure was found to be back to its former level; he was placed on a daily dosage of 200 milligrams and has not again experienced any difficulty.

Results.—The data obtained in the individual cases is shown in the table. The following figures are averages for the 23 cases:

Duration of treatment, 9.65 weeks.
B. m. r. before treatment, plus 2.87.
B. m. r. during treatment, plus 31.36.
Weight before treatment, 218.2 pounds.
Loss of weight, 28.5 pounds.
Loss per week, 2.44 pounds.

There have been no failures due to any adverse action of the drug. In nearly every case an abrupt rise in the basal metabolic rate was recorded during the first week of treatment. Every case showed an appreciable loss of weight after the second week of treatment and a number of them after the first week.

Comment.—To eliminate the possibility of accumulative action of the drug through diminished kidney function, the kidney function of all cases was investigated. With the hospital cases this consisted of routine and microscopic examination of a series of morning urine specimens, a Mosenthal test and where indicated a concentration-dilution test. In out-patients a combination concentration-dilution test was devised that could easily be done in the home. The patient was instructed to take no fluids from the time of the evening meal until 11 a. m. the following day, then to take 1,500 cubic centimeters of fluid at that time. Urine specimens were taken at 8 a. m., 11 a. m., 1 p. m., and 3 p. m. A specific gravity variation from 1.005 to 1.020 was considered as indicating normal kidney function in the absence of albumin, casts, and red blood cells.

Derrien's test, described in an article by Anderson, Reed, and Emerson (5) is used to detect the presence of an end product of dinitrophenol in the urine, blood, and tissue extracts. According to Perkins (8) a persistence or increase of this substance in the urine is an indication of intolerance to the drug. An attempt is being made to evaluate the Derrien's test as an indicator of intolerance or overdosage. Hospital cases were selected who had been on full therapeutic doses for at least a week. Administration of the drug was stopped suddenly and urine specimens collected at varying intervals and tested by Derrien's method. The results have been, so far, rather inconclusive. Cases I, VII, and X are cited as examples. Under treatment for 19, 17, and 17 weeks respectively with loss of 86, 65, and 30 pounds, respectively, they are considered as responding very satisfactorily to treatment. At no time did any signs of overdosage or intolerance develop and they are, without doubt, in better health today than they were before they were treated. Yet the Derrien's test was definitely and consistently positive in nearly all samples of urine examined and as long as 35 hours after the last dose of the drug had been given.

TABLE

Case no.	Duration of treatment in weeks	Basal Metabolic Rate			Original weight	Present weight	Total loss of weight	Average loss per week
		Before treatment	Average during treatment	Highest				
					Pounds	*Pounds*	*Pounds*	*Pounds*
I. m.	19	+10	+32.5	+48	336	250	86	[1] 4.5
II. m.	11	−5	26	63	289	231	58	[1] 5.27
III. f.	11	−15	22	39	181.5	160	21.5	1.95
IV. m.	17	+25	31	56	206	164	42	[1] 2.47
V. f.	15	−16	19	44	252	207	45	3.0
VI. f.	8	+8	37	58	175	155	20	2.6
VII. m.	17	+45	59	94	253	188	65	[1] 3.8
VIII. m.	5	−8	56	77	188	168	20	4.0
IX. m.	11	+15	19.6	32	206	174	32	[1] 2.9
X. m.	17	+8	35	66	220	190	30	[1] 1.8
XI. m.	9	−10	30	55	194	181	13	1.45
XII. m.	9	−8	20	45	252	222	30	3.3
XIII. m.	6	+4	15	29	204	180	24	[1] 4.0
XIV. f.	5	0	33	48	212	197	15	3.0
XV. f.	10	+2	53	68	194	171	23	2.3
XVI. m.	4	−9	35	62	209	200	9	[1] 2.25
XVII. f.	9	0	24	48	212	189	23	2.55
XVIII. f.	7	+10	21	45	201	191	10	1.4
XIX. m.	8	0	40	70	220.5	193.5	27	3.4
XX. m.	7	−.4	22	40	224	204	20	2.8
XXI. m.	6	−12	21	28	206	191	15	[1] 2.5
XXII. m.	4	+5	41	52	186	174	12	[1] 3.0
XXIII. m.	7	+22	39	67	199.5	184	15	[1] 2.1

[1] Hospital cases.

RESULTS OF DERRIEN'S TESTS

CASE I.—Urine: Negative for albumin, casts and RBC. SpG. variation, 1.001 to 1.019.

Dosage: 600 mgm daily.

Urine series:

1. 8 a. m., positive; 10 a. m., positive; 12 m., positive; 2 p. m., positive; 4 p. m., positive.
2. 8 a. m., negative; 2 p. m., negative; 6 p. m., positive; 10 p. m., positive; 8 a. m., negative.
3. 10 a. m., positive; 12 m., positive; 2 p. m., positive; 6 p. m., positive; 10 p. m., positive; 8 a. m., positive.
4. 1 p. m., positive; 7 p. m., positive; 7 a. m., positive; 12 m., positive; 6 p. m., positive.

Last dose 7 a. m.

CASE VII.—Urine: Negative for albumin, casts and RBC. SpG. variation, 1.005 to 1.027.

Dosage: 600 mgm daily.

Urine series:

1. 8 a. m., positive; 10 a. m. positive; 12 m., positive; 2 p. m., positive; 4 p. m., positive.
2. 10 a. m., positive; 2 p. m., positive; 6 p. m., positive; 10 p. m., positive; 8 a. m., positive.
3. 10 a. m., positive; 2 p. m., positive; 6 p. m., positive; 10 p. m. positive; 8 a. m., positive.
4. 1 p. m., positive; 7 p. m., positive; 7 a. m., positive; 12 m., positive; 6 p. m., positive.

Last dose 7 a. m.

CASE X.—Urine: Negative for albumin and casts, SpG. variation, 1.006 to 1.024.

Dosage: 600 mgm daily.

Urine series:

1. 8 a. m., positive; 10 a. m., positive; 12 m., positive; 2 p. m., positive; 4 p. m., positive.
2. 10 a. m., positive; 2 p. m., negative; 6 p. m., positive; 10 p. m., positive; 8 a. m., positive.
3. 1 p. m., positive; 7 p. m., positive; 7 a. m., positive; 12 m., positive; 6 p. m., positive.

Last dose 7 a. m.

CONCLUSIONS

1. From a limited number of cases treated, it appears that dinitrophenol can be successfully and comparatively safely administered in obesity.

2. Cases should be selected, especially as to kidney function, and should be kept under careful control.

3. Derrien't test does not appear to be a satisfactory indicator of intolerance or overdosage.

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THE TREATMENT OF FURUNCLES AND CARBUNCLES[1]

By WILLIAM H. WHITMORE, Lieutenant Commander, Medical Corps, United States Navy

The pathology of furuncles and carbuncles is too well known to require any description. It may be well, however, to consider certain points that have a bearing on the treatment. The ordinary case of acute furuncle, without complications, seldom comes to the

[1] Read before the Norfolk County Medical Society, Norfolk, Va., May 14, 1934.

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physician for treatment; the cases that are presented for treatment usually have been mistreated by the patient himself, or others, or are of a serious nature on account of their location.

The superficial carbuncle is very vascular, with early multiple skin perforations, and does not leave a deep crater after sloughing. This type is frequent on the face. The deep carbuncle, frequent on the back of the neck, presents a fixed mass with a large area of induration; skin perforation and sloughing occur later, and the pus may burrow in the tissues for several weeks before perforation occurs.

Furuncles and carbuncles may be considered of minor importance unless the mortality reports are considered. A furuncle on the lip or nose may be just as dangerous as a carbuncle, on account of the adjacent anatomical structures, allowing possible extension of the infection through the veins of the cavernous sinus.

Payr (17) reports 103 cases of furuncle of the face with 10.7 percent mortality; Dittrich (7) reports 10 percent mortality; and Hofman (10) reports 182 cases at Bier's Clinic with 8.2 percent mortality. Turner and Richards (21) report 1 death in 63 cases of furuncle of the nasal vestibule, a mortality of 1.5 percent.

The mortality of carbuncles varies with the location, the general condition of the patient, and character of complications. Dittrich (7) reports 40 cases of carbuncle of the upper lip; 22 were incised with 13.6 percent mortality; 18 were not incised with 5.5 percent mortality.

During a 3-year period in the United States Navy there were admitted 958 cases of furunculosis, with 1 death; and 242 cases of carbuncle, with 3 deaths. The mortality rates here are low, as these figures include all cases admitted to the sick list on ships and stations as well as hospital cases, and many of these are cases that would not have sought medical attention outside of the Navy.

Treatment.—The number and variety of treatments offered for carbuncle is good evidence that no one method is entirely satisfactory. Christopher (4), in 1928, published a list of about 30 reported methods of treatment (table 1). Carp (3) advocates the circumjection of autogenous blood; he reports 12 carbuncles with 11 cures in from 11 to 41 days, averaging 23 days. Sheely (18) advocates multiple stellate incisions with undercutting of flaps.

Bullock (2) advocates conservative measures, with ligation of the angular vein in certain carbuncles of the upper lip.

Willmoth (24) coagulates all involved tissue with the bipolar high-frequency current, then removes the coagulum with the curette. Livingston (16) advises the immediate excision of the necrotic mass in carbuncles of the back of the neck, and uses plaster of paris to immobilize the head. Kuhn (13) uses a vacuum cup with strong

suction for one-half to 4 hours, producing hemorrhage around the carbuncle.

Many surgeons believe in the radical excision of carbuncles, either by knife or cautery, but use more conservative measures when the lesion is on the face (3) (4).

White and Cooney (23) report good results in 100 carbuncles treated by allowing the lesion to run its course, aided by nonspecific protein therapy.

X-ray treatment.—Coyle (5) in 1906 reported 5 carbuncles treated by X-ray, 4 of which were aborted. This report apparently attracted little attention. Dunham (8) in 1916 reported 67 successive cases of carbuncle successfully treated by roentgen rays. Hodges (11, 12) reported 26 carbuncles treated by X-ray, with 2 failures.

Light and Sosman (15) treated 50 cases of carbuncle with X-ray, with 2 deaths, and state that diabetes had little or no effect on the result of X-ray therapy.

Leddy and Morton (14) report 100 boils and carbuncles treated by X-ray at the Mayo clinic, with 10 failures, and conclude that "the treatment of these lesions by roentgen rays, although not always efficacious, is superior to the methods generally used." They explain the effect of the X-rays as due to the destruction of lymphocytes in the inflammatory exudate, releasing the protective substances contained in these cells.

The experiments of Thorness (22) tend to confirm that idea. He studied the effects of X-ray on experimental abscesses, and found that the direct destructive action of X-rays was evident 1 day after treatment, while indirect effects were shown by similar changes in untreated control abscesses in the same animals, appearing 4 to 5 days later; this indirect effect is explained as due to circulating hormones liberated at the irradiated area.

We have used X-ray treatment of furuncles and carbuncles at the Norfolk Naval Hospital since January 1927; a series of cases treated between January 1927 and June 1929 and from June 1932 to April 1934 is the basis of this paper. Our method is to give from one-half to one skin erythema dose of the rays, the amount of radiation varying inversely with the size of the lesion, the average treatment being about three-fourths of the skin unit. The filtration is varied according to the thickness of the indurated area. Occasionally a second treatment has been given after 1 week, but usually one treatment has been sufficient. After the X-ray has been given, local applications are avoided, using only a protective dressing and heat.

The most striking result of the treatment has been the relief of pain, and if the treatment accomplished nothing more, it would be justified for this alone. In from 3 to 12 hours after treatment the pain has been completely relieved or considerably decreased. Any

case in which the pain has not been alleviated or relieved has been classed as a failure.

When the treatment was given early, the exudate has been absorbed and the process aborted; when given later, the spread of the infection has been checked, and there has been a rapid central necrosis, often with spontaneous perforation and drainage. When spontaneous perforation did not occur, a very small incision has been made for drainage. When treated after incision or spontaneous perforation, drainage and resolution have been accelerated. The scarring after this method is very slight, and often hardly noticeable.

Only two cases of carbuncle are listed as aborted, but some of the furuncles could have been classed as carbuncles; the cases have been listed according to the diagnosis made by the ward surgeons.

Summary of cases treated

Carbuncles:	
Location	
Neck, posterior	12
Lip and chin	1
Other parts	6
	19
Treated by X-ray	19
Not improved	1
Aborted	2
Improved	16
Spontaneous drainage	7
Furuncles:	
Nose	34
Lip	6
Other parts of face	17
External auditory canal	44
Other parts	50
	151
Treated by X-ray	151
Not improved	4
Aborted	33
Improved	114
Spontaneous drainage	51

Case Reports

Failures

Case 32.—M., Mrs. J. B. Furuncle of nose, with induration of face resembling erysipelas, 2 days duration. Treated September 15, 1927, 1 S. U. D. Result, pain not relieved; developed signs of meningeal irritation that night; treated in another hospital with antistreptococcic serum and recovered.

CASE 58.—R., Miss H. Furuncle of nose, 3 days duration. Treated February 22, 1928, 1 S. U. D. Result, pain not relieved; course of infection not influenced; recovered.

CASE 119.—J., A. M. Lieut. (S. C.), U. S. N. Carbuncle, back of neck; about 1 week duration; multiple pointing with a large area of induration. Treated April 6, 1933, ¾ S. U. D., filtered; April 11, 1934, ½ S. U. D., filtered. Result, perhaps some decrease in pain and increased drainage; improvement too slight to consider treatment successful. On April 21, 1933, the carbuncle was excised. Wound healed completely in 45 days.

CASE 135.—M., W. H., C. G. M., F. N. R. Furuncle and cellulitis of nose, 2 weeks duration. Treated, October 9, 1933, ¾ S. U. D.; October 13, 1933, ½ S. U. D. Result, pain and induration decreased. Two weeks after first treatment there remained a clean ulcer with an indurated base on the inner surface of the ala. Kahn 4 plus. Apparently a chancre with secondary infection.

SUCCESSFUL CASES

CASE 1.—K., J. A. Pvt. Carbuncle, back of neck, duration 1 week. Patient had had little sleep in past 4 days, although morphine had been given several times. Large indurated area on back of neck, extending onto scalp, no pointing and no fluctuation. Treated January 29, 1927, 1 S. U. D. Treatment given at noon. Result, pain completely relieved in 8 hours; patient slept well that night without opiates. Following day, there was a central area of fluctuation; small incision made, with evacuation of large amount of pus; induration subsided rapidly, and there was complete healing in 10 days, with a small linear scar. A temporary alopecia followed the treatment.

CASE 7.—M., R. C. A. M. M. 1c. Carbuncle, neck posterior, duration about 1 week; large area of induration with multiple pointing. Treated, April 26, 1927; 1 S. U. D. Result, pain relieved within 12 hours; spontaneous perforation next day, rapid drainage; complete healing in 16 days.

CASE 56.—G., B. Lieut. Comdr. (M. C.), U. S. N. Furuncle, thigh, duration 3 days. Large indurated mass, anterior surface of thigh, deep seated, with central necrotic area; appearance resembels anthrax carbuncle; smears show only staphylococci. Treated January 24, 1928, 1 S. U. D. Result, pain completely relieved in 3 hours; rapid evacuation of necrotic center; complete healing in 10 days.

CASE 67.—E., J. M. M. 1c. Carbuncle, back of neck, duration 1 week; large area of induration, multiple pointing, litle drainage. Treated, April 30, 1928, 1 S. U. D. Result, pain relieved in 6 hours; following day, necrosis centralized; small incision into necrotic center, with free drainage of pus; complete healing in 25 days.

CASE 71.—K., A. J., F. 3c. Carbuncle, back of neck, duration 1 week; had been treated with ichthyol dressings. Area of induration about three inches in diameter; no pointing or fluctuation. Treated June 25, 1928, 1 S. U. D. Result, pain relieved in a few hours; induration absorbed in 5 days.

CASE 82.—H., B. R., Lieut. (jr. gr.), U. S. N. Carbuncle, neck, posterior, duration about 1 week; had been treated with ichthyol dressing; large carbuncle on back of neck with multiple pointing and partly necrotic center. Very painful. Treated Septemebr 26, 1928, 1 S. U. D. Result, pain considerably decreased in a few hours; following day small incision into necrotic center; free drainage continued 8 days; complete healing in 33 days.

CASE 96.—B., H. G. Lieut. Comdr., U. S. N. Carbuncle, lip and chin; 5 days before admission, had a small boil on chin, from which he squeezed a core on the second day; following this, there was increased swelling with dull pain;

he reported at the sick bay, and the area of swelling was incised, and wet dressing applied; there was relief for 48 hours, then the swelling and pain increased. Patient had a chill night before admission to hospital. On admission, there was a small necrotic area on left side of chin, about which there were multiple small pustular points; induration involved the entire chin and left side of lower lip. Hot wet dressings of magnesium sulphate applied. Two days later there were multiple areas of suppuration around original focus, with increased swelling, and extension upward of the induration; patient had a chill with rise of temperature to 101. Referred for X-ray treatment 7 days after onset. Treated April 5, 1929; ¾ S. U. D., unfiltered, and ¼ S. U. D., filtered. Result, pain relieved in 3 hours; spontaneous perforation and drainage during night; free drainage continued 3 days; complete healing in 10 days, with very little scar.

Case 99.—S., G. E. Sea. 2c. Furuncle, upper lip. Duration 2 days. Day before admission, patient squeezed a small boil on upper lip; three hours later upper lip began to swell, with considerable pain; patient went to dispensary, where furuncle was probed; during night swelling of lip increased; patient suffered throbbing pain and "felt sick all over." On admission, temperature 100; there was a large furuncle on the upper lip, with induration extending into the right nares. Treated April 22, 1929, ¾ S. U. D., unfiltered. Result, pain relieved in a few hours. In 24 hours there was spontaneous drainage; complete healing in 10 days.

Case 101.—N., J. M. Att. 1c.; Furuncle, nose, 3 days' duration; induration of entire nose. Treated April 25, 1929, 1 S. U. D. Result, pain relieved; spontaneous drainage in 12 hours; healed in one week.

CONCLUSIONS

X-ray treatment of furuncles and carbuncles gives better results than other methods; the treatment is painless, is not expensive, and can be given in any hospital or in any office equipped with an X-ray machine of medium power. In the early stages, when all other methods offer the least, the X-rays are most effective. There is no contra-indication to the proper use of this agent, and the use of X-rays does not interfere with later surgical measures, if needed.

With the high mortality of furuncles and carbuncles of the lips and nose, and the well-known harmful effects of trauma in these cases, such cases should be given X-ray therapy early, as soon as possible, and other measures considered only when the X-ray treatment fails.

Any case of furuncle or carbuncle that requires the care of a physician is serious enough to warrant X-ray treatment.

Table 1.—*Treatment of furuncles and carbuncles*

I. Prophylactic.
II. Local.
    A. Local.
        1. Incision or excision by knife.
        2. Incision or excision by cautery.
        3. Ignipuncture (glow needle).
        4. Sounding and dilatation.

II. Local—Continued.
A. Local—Continued.
5. Phenol probe.
6. Rest; avoidance of trauma.
B. Chemical.
1. Cataplasma and poultices.
(*a*) Unguents.
(*b*) Pepsin.
(*c*) Pancreatic ferments.
2. Hypertonic solutions.
(*a*) Saturated boric acid.
(*b*) Aluminum acetate.
3. Antiseptic applications.
(*a*) Phenol.
(*b*) Iodine.
(*c*) Ichthyol.
C. Heat.
1. Hot fomentations.
2. Dry heat.
D. Irradiation, diathermy, etc.
1. X-ray.
2. Ultra-violet.
3. Diathermy, electrocoagulation.
E. Biological.
1. Auto-blood circumjections.
2. Vacuum cupping.
3. Bier's hyperemia.
4. Horse serum.
5. "Histoplast."

III. Systematic treatment.
A. Biological.
1. Vaccines.
2. Insulin.
3. Blood.
4. Nonspecific proteins.
B. Pharmaceutical.
1. Sulphur.
2. Tin.
3. Manganese.
4. Quinine.
5. Mercury.
6. Mercurochrome.
7. Turpentine.
C. Dietetic Measures.
1. Laxatives, rest, fluids, etc.[1]

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INDUSTRIAL MEDICINE

PART II

By H. L. SHINN, Lieutenant Commander, Medical Corps, United States Navy.

The United States Government is by far our largest employer. Thousands of this number represent the United States Navy. Naturally this presents a big problem. Every man or woman employed by the Government who contracts a disease or is injured and this disease or injury can be traced to his employment must, if he is in-

capacitated, be compensated or retired on a pension by the Government. Naturally, from an economic standpoint, then, it behooves the Government to protect its employees. In so doing, it not only protects the Government but benefits humanity and insures better work from the employees. As stated before, a well man is an asset. This is the aim of the Navy Department with regard to the employees—civilian, enlisted, and officers. I will give you here a brief summary or outline of the work being carried out by the Navy Department to protect the employees and personnel. I quote verbatim here a report:

"The Navy Department recognized several years ago that it was economical as well as an humanitarian act to protect its employees from industrial accidents and injuries, therefore a program was inaugurated and placed into effect. A civilian safety engineer was employed and placed in charge of the work. This safety engineer is attached to the Secretary's office, navy yard division. At each of the navy yards and stations there is an officer detailed as safety engineer to carry out the Department's policy locally.

"Each month a report is made to the Department of the number and character of each injury, together with the number of days lost. These data are consolidated, and from this it is possible to ascertain what particular type of injury is most prevalent and to classify the different classes of injuries, then to devise methods of protection and prevention. The Navy's industrial organizations are now leaders in the field of accident prevention and have contributed much, as for instance:

"Welding lenses that have a greater absorption of infrared and ultraviolet than ever before accomplished.

"Protective lenses 50 millimeters that give greater protection against flying particles, range, and clearness of vision.

"Welding helmets affording greater comfort and more protection against the arc-welding operations.

"Sand-blast helmet, respirator protection, better fitting goggles, gloves with a longer life, and many other such items of personal protection.

"Safety engineering is so varied and covers such a wide field that it is almost impossible to know where to start or stop—so many things have a bearing upon industrial accidents.

"A person's mental attitude toward his employer, supervisor, or particular occupation, the employer's or supervisor's attitude toward the employee, mental and physical fatigue brought about by poor lighting, poorly kept shops, misplaced machines, repeated operations, sanitation, not sufficient time and improper place for relaxation at the meal time, improper food, home surroundings, and recreation—all contribute their part.

"The last two mentioned are the hardest to overcome, but much can be accomplished by making the working surroundings such that they will not work a disadvantage upon the nervous system of the employee. By making the working surroundings clean and wholesome and less apt to fatigue, the employee can be placed into a frame of mind that will react upon him outside of his employment and not only make him a safer person but a better workman and citizen. These things are not theory but are based upon fact, proven through years of actual practice.

"The navy yards have made continuous improvement in the conditions above mentioned; however, an educational program must supplement the other activities, in order to prepare the employee for these bettered physical conditions, to make him safety-minded and to create within him a sense of his responsibility to his employer, to himself, and family, as well as society at large, and have him understand that by doing better work in a safe way he enhances his value and thus paves the way to a long life of usefulness. Many injuries have been prevented in the naval establishment—this the records show—but what is not shown, is the human suffering and heartaches that have been prevented by the work accomplished. The physician's part is to create sanitary surroundings, promote cleanliness, and a mindfulness of the necessity of caring for one's health. With healthy bodies, a clear mind, and pleasant surroundings the way is well paved for no industrial injuries."

The above report is a general one and does not give much idea of the medical aspects of the work done. I will therefore endeavor to make you familiar with the subject from the viewpoint of the medical officer.

The writer does not wish to bore his readers with statistics, but will give here the comparative figures on accidents for all navy yards to show what success has attended the Bureau's efforts toward insuring safety. The figures indicate lost-time accidents.

Year	1926	1927	1928	1929	1930	1931	1932
Boston	244	265	139	161	122	105	23
Cavite	319	70	74	105	107	84	54
Charleston	531	11	234	10	16	16	16
Mare Island	68	71	139	189	119	48	39
New York	120	126	133	186	135	123	127
Norfolk	237	214	245	281	98	63	26
Pearl Harbor	41	45	87	99	86	72	56
Philadelphia	226	212	155	125	162	145	83
Portsmouth	66	51	76	71	63	48	32
Puget Sound	154	111	123	161	159	153	51
Washington, D. C	72	113	103	117	84	124	77
Total	1,562	1,289	1,508	1,505	1,151	981	584

It will be noted from this report that over a period of 7 years the accident rate has been cut from a total of 1,562 to 584. No

figures are as yet available for the year 1933, but it is understood that there were considerably less accidents than in 1932.

When the writer of this article reported for duty to the navy yard at Washington, he was informed that his duties would be as adviser to the safety engineering officer and officer in charge of industrial medicine. As stated before, this was all new to me and I hardly knew where to begin. The navy yard in Washington has many hazardous trades. There is much heavy work to be done in the foundries, gun shops, breech-mechanism shops, erecting shops, boilermaker shops, and others. In reality, it is not a navy yard but a gun factory. The commandant here took a distinct personal interest in the health and welfare of the employees and formed an organization to protect them. His organization, as he desired to build it up, is as follows:

1. A naval line officer with the rank of lieutenant commander or lieutenant as safety engineering officer.

2. A medical officer as adviser to the safety engineering officer in matters of health and accident prevention.[1]

3. A civilian assistant to the safety engineering officer selected from among the employees of the yard who is fully qualified by reason of knowledge of all activities in the yard and a man of a cooperative personality.[1]

4. An assistant to the civilian safety engineer of the same qualifications.[1]

With this organization it was hoped that the accidents would be minimized, the health of the command improved, and the work of the men bettered accordingly. In my mind, the above is a good working organization for a plant of this type and should bring results.

Now, as to the duties of each member of this organization.

The safety engineer's paramount duties are to prevent accidents. It is his duty to inspect thoroughly, and to make a general survey of all mechanical conditions of the working places, and to add all acceptable appliances for the protection of the workers. He must make daily inspection of the plants to see that these safeguards against accidents are in working order and being used. He must also investigate every accident in order to devise means to prevent a similar accident. There must be the closest cooperation between the safety engineer and the medical officer, and in some institutions he is assigned to the medical department. The safety engineer must ascertain all hazards and recommend corrective measures. All improvements come under his supervision. A hazard is any condition which will lead directly or indirectly to injury or disease. A minor

[1] Full-time duties.

hazard is a greasy shop floor or an open hatch aboard ship. These hazards may lead to serious injury. A major hazard is represented by unguarded machinery. This, then, in general, is the duty of the safety engineer.

The duties of the medical officer in industrial medicine are varied. He is not interested in machinery to any extent; and this is well, for not many physicians are familiar with machinery or its mechanism. However, he should know enough about the subject to talk intelligently with the safety engineer and be able to recommend and approve safety devices, especially masks, goggles, clothing, etc. This is especially essential in aviation and diving activities in the Navy. The medical officer is primarily the safety engineer of the human body. He acts to a greater degree as a consultant to the safety engineer in all matters pertaining to the general welfare and health of the employees. Hygiene and sanitation are his important duties. He must interest himself in the employees and instruct them in the everyday principles of personal hygiene and self-preservation. He must interest the employees and let them know that he is interested in their welfare. He must instruct them in safety measures. He must keep in constant contact with the employees regarding their health. He must, in other words, enlist the cooperation of all employees and in turn cooperate with them. You will find that this is an important factor. One of the most important factors in the reduction of accidents is to build up a spirit of confidence in the men and, in thus doing, the morale will be brought to a high standard. If a man has confidence in his superiors and knows that they are working for his welfare, he will cooperate in bringing safety to his work place.

The medical officer must next inspect all shops and places where the men are required to work. In making this inspection, if he has the cooperation of the men, many valuable points will be gained. Criticism and comment are urged from the men in a constructive way. When the men know they are free to talk, many helpful points will be learned that could not otherwise have been known. For example, in one shop in the navy yard the men were dissatisfied, and rightly so, because of the poor ventilation in their shop; this caused them much pulmonary distress, due to the constant inhalation of chemicals during the winter months. In the summer when the doors and windows could be kept open they had no complaint, but in the winter it was pretty bad. To one inspecting, this would not be noticed because of the short interval spent inspecting. The men made no complaint to the foreman for fear of losing their jobs. However, when they were taught that this was just what we wanted to know, they did complain. Accordingly, their complaints were investigated, found to be just, and corrective measures were instituted.

In this shop today, although the trade is a very hazardous one from a health standpoint, we have a high morale, little sickness, and very few accidents. This may seem a minor thing to many, and yet it is an important link in the chain leading up to a lowered sick and mortality rate. Without this link the chain would be weak, indeed. This link is called cooperation.

After inspecting, the medical officer must make the necessary recommendations to improve the deficiencies he has noted. It is not enough to make recommendations. At least once a month he must further inspect to determine what action, if any, has been taken on his requests. If none has been taken, then he must get behind whoever is responsible and see that the work is done. To indicate to you the responsibility and authority given the medical authorities in this work I wish to quote an incident which occurred several years ago in a foundry. The medical inspector for the State organization, while inspecting the sand-blast compartment of the foundry, discovered a small leak in the ventilating system which allowed dust to escape into the compartment where the men were working. He ordered it fixed at once. The manager of the foundry protested that it would necessitate shutting down and stopping production for 10 days, and he could not well afford to do the work at this time. However, the health of the workers was at stake, and the medical advisor closed the shop until the work on the ventilator was completed and inspected. This simply indicates again the importance of health over that of production.

The medical officer must further keep in touch with the various heads of departments and encourage in them a spirit of cooperation with other shops and factories. Competition does much toward maintaining health and preventing injuries. All shops in the navy yards of the United States are on a competitive basis, and the shops and yards with the best records are rewarded with trophies, certificates, and efficiency buttons for their excellence. It is surprising to see the keenness of this competition and the results it obtains. This is another link in the chain, secondary, however, in my mind, to cooperation and high morale.

I might best give my readers an idea as to the further duties of a medical advisor connected with industrial medicine by giving here a description of my duties at the navy yard dispensary at Washington. By inspection of the various shops and offices in the navy yard the work done in each has become familiar to me. All prospective employees coming to the yard are given first a complete physical examination. Some of this work I do myself, but the majority is done by other medical officers. However, if there is a doubt as to the ability of an applicant to perform the duties of a certain trade or in a certain shop, I can give the other officers my

opinion of the matter from my knowledge of the character of the work that he is to perform. In this manner we endeavor to get physically fit men. Likewise, many employees have been in the navy yard for many years and have developed various physical disabilities. These men are referred usually to me; they are examined and their disabilities are weighed with the type of work required of them, and, if found unable to do the work, they are either recommended for retirement or a change of work which they can perform without danger to themselves or others.

Much of my time is spent at the dispensary. Every man or woman employee, which now number over 5,000, who is sick or injured must report to the dispensary, where he or she is examined. It makes no difference if the illness is a cold requiring absence from duty or if the injury is only a minor scratch. It has been found that the smallest scratch, especially those caused by emery, brass, etc., will sometimes lead to severe infections. Formerly, first-aid boxes were kept in all shops and offices, but this was discontinued because the men would not properly treat the wounds, and in some cases infections developed which required that the employee be sent home or to a hospital. Every employee therefore, sick or injured, is seen by a doctor at the dispensary and a record kept showing the illness or the injury, its seriousness, and whether or not it was due or not due to employment. These records also indicate whether or not an employee was incapacitated for duty. Insofar as it is possible, it is my duty to see all of these injured employees and to determine the cause of the injury. Each day all injury reports are carefully gone over and are listed according to diagnosis, in which shop the injury occurred, and whether or not the injury necessitated the loss of time from work. It is then my duty to determine the cause of the injuries, i. e., whether or not the accident was avoidable. If avoidable because of some hazard, the safety engineer is notified and measures are taken to prevent a duplication of such injury. It is found that during a certain period of time one or more shops will have an unusual number of accidents. Maybe these injuries are trivial, but it indicates that the supervisors of these shops are getting careless and if the condition is not corrected a serious accident is sure to result. This fact is reported to the masters in the shop and their cooperation solicited. Soon a marked improvement will be noted. Ninety percent of all accidents are due to carelessness. Yet, an employee who is injured through carelessness gets the same compensation as a man who is injured otherwise. It is therefore essential that some check be kept on the careless master and employee. If an employee is hurt several times within a short period of time it is well to have him examined physically to deter-

mine the cause. If it is due to carelessness he should be marked in his efficiency accordingly and discharged if necessary. Physical examinations are of prime importance. Many older employees hate to give up; but sooner or later, due to their physical condition, hurt themselves. Physical examination of the men should be made periodically and assigned to duties less hazardous if necessary, for their own good, as well as for their fellow workmen, whom they are likely to injure because of their physical inability to perform the work they were able to do when younger. Just recently a painter, age 56, had a dizzy spell while up on a scaffold painting. He fell off and luckily only fractured a rib instead of killing himself. Had this man been assigned to a painting job where he did not have to be on a scaffold, this would not have happened.

In the dispensary it is further my duties to investigate all serious accidents or, as we term them, lost-time accidents. It is my duty to see that the employee injured loses as little time from the job as possible. Accidents are rated according to the number of actual hours lost. Some employees demand to stay away from work as long as possible and draw compensation. In all cases the medical officer must determine when he is fit for duty.

I have told you briefly what my duties are with regard to injuries. With reference to diseases they are practically the same. All diseases occurring among the employees which are or might be attributed to their employment must be investigated thoroughly, and if found to be due to their trade corrective measures are taken. So much for the duties of a medical officer assigned to industry.

I have made mention of a civilian assistant to the safety engineer and also his assistant. These men are invaluable if of the right type. They must be men well versed and educated in the trades. They must be good mixers and must inspire confidence. They act as a liason officer between the Navy and civilians. They act under the direction of the safety engineer and the medical advisor, and together they devise ways and means for the protection of the employees.

With such an organization the principles outlined previously for the prevention of accidents have been carried out at the Washington Navy Yard.

It is hoped that the foregoing will give some idea as to the purpose of the duties of a medical officer assigned to industrial medicine. I would like to list here a few figures which indicate to what extent improvement can be obtained by preventive measures, such as are taken at the navy yard.

In the year 1931, prior to the organization and the taking of actual safety measures seriously, the record of accidents was as follows:

Year 1931	Lost-time accidents	All injuries	Year 1931	Lost-time accidents	All injuries
January	10	134	August	11	118
February	11	120	September	8	137
March	9	125	October	10	124
April	14	115	November	8	104
May	19	123	December	3	114
June	15	116			
July	12	156	Total	130	1,486

This organization against accidents was started in December 1931. The figures for the year 1932 are as follows:

Year 1932	Lost-time accidents	All injuries	Year 1932	Lost-time accidents	All injuries
January	5	108	August	10	138
February	6	106	September	4	75
March	7	105	October	4	100
April	5	93	November	12	104
May	6	120	December	2	94
June	12	90			
July	8	141	Total	81	1,274

You will note here a decrease in the number of lost-time accidents of 51 and in the total number of accidents of 212 over the year 1931.

The figures for the year 1933 are as follows:

Year 1933	Lost-time accidents	All injuries	Year 1933	Lost-time accidents	All injuries
January	3	90	August	5	160
February	3	100	September	2	132
March	2	93	October	1	133
April	1	83	November	5	158
May	0	102	December	3	143
June	5	95			
July	4	104	Total	34	1,393

Here it will be noted that there is a decrease in the number of lost-time accidents for the past year over 1931 of 96 and over the year 1932 of 47. It might well be added that in the year 1931 one death occurred, and in 1932 there were two. Further, one will notice in the latter months of 1933 that there was an increase in the total number of all accidents but that the lost-time accidents have remained low. The increase in the total number of accidents is due largely to the increase in personnel. Since July 1933 there has been over 1,000 new employees taken on, due to increased production. These men are not familiar with the safety precautions as yet, but are being instructed as soon as they are accepted for employment. The total number of all injuries may appear high for a complement of approximately 5,500 employees; but this is due to the fact that each and every injury, no matter if it be the smallest of lacerations or abrasions, is listed, and the men are re-

quired to report all such injuries. The reason for this has been previously explained.

This is an example of just what can be done. I may say here, referring again to the organization, that it was impossible to have the civilian assistants referred to, because of economic conditions, assigned. It is firmly believed that with the improvement shown without them that much more headway could or can be obtained with their addition and help. The good work done thus far can be attributed to the splendid spirit of cooperation obtained from the workmen themselves. No doubt, some of my readers have done work along lines that have been described, and others may expect to do this work if they remain in the Navy. However, I feel safe in saying that whether you have or not, whether you do or not, that you will, wherever you are assigned, be required to have a working knowledge of industrial medicine. Therefore, wherever you may be assigned, one of the first things you should do is to learn fully the physical characteristics of the place you are on duty; whether a ship, a barracks, with troops, in a hospital, or elsewhere. Find out the hazards as to accidents and disease to those for whose health you are responsible and endeavor to correct any such condition. Of prime importance to health is the prophylaxis against smallpox and typhoid fever. Personally see that the officers and men under you are fully protected against these two diseases.

In closing, there is just one thing I hope will be accomplished by this paper. It is hoped that others in the Service may see the benefits derived from industrial medicine and will work along these lines. This subject is too large and covers too much territory to be discussed fully in one paper. Therefore, it has been necessary to deal in generalities. However, in general again, it is felt that the accident rate and disease rate in the Navy today can be and should be reduced. In the first part of this paper I quoted the accident rate for the Navy for the year 1932, which to my mind is excessively high. I would like to see the Navy put on a competitive basis regarding health and accident prevention, similar to that carried out in engineering, navigation, gunnery, etc. Why not give a health E to the ship or station showing the best health records for the year? Without health efficiency, records in gunnery or engineering mean little. Should the members of your best-trained gun crew get injured or sick at the time of competition, what chance has your ship to win the trophy?

Therefore, in the interests of a healthier Navy, which will make a more efficient Navy with a high morale, the following plan is suggested:

1. That every ship and station, no matter how large nor how small, organize a department of safety.

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2. That this organization be directly under the commanding officer, who will in turn assign an officer as safety engineer to carry out the duties of such officer as described in this article. If aboard ship or larger station these duties could well be assigned to the first lieutenant.

3. That a medical officer or a hospital corpsman be assigned as adviser to the safety engineering officer and to carry out the duties as described herein.

4. That a safety committee or safety committees be appointed from among the crew and instructed in the principles of safety, that they may assist the safety engineer and medical adviser in building up a spirit of prevention, health, cleanliness, etc., in the other members of the crew. These safety committees are very important and are a great help. Aboard ship, safety committees should be appointed in each division and a spirit of competition developed.

These ideas set forth here are not new nor original. Prevention has always been taught in the Navy, but as stated before has been neglected to some extent. An E for the health of your ship or station would be the finest example of excellence obtainable.

So, if there is no such organization on your ship or station, get one started and cooperate to put industrial medicine on a working basis throughout this big industry. I venture to say that if this is done and done thoroughly that 2 years hence the Surgeon General's annual report will show at least a 50 percent reduction in accidents, less loss of time from work, and generally a healthier personnel.

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CLINICAL NOTES

SPLENOMEGALY, ASSOCIATED WITH SYPHILIS, WITH RECOVERY, FOLLOWING SPLENECTOMY

By R. H. LANING, Commander, and A. W. LOY, Lieutenant, Medical Corps, United States Navy

It is thought advisable to report a case of splenomegaly because of the picture of splenic anemia or Banti's disease, which it presented, and because of its association with syphilis.

Banti described the disease as a primary disease of the spleen with splenomegaly, anemia, hemorrhage, and in some cases, ascites, cirrhosis of the liver, and jaundice. Recovery from symptoms following splenectomy is considered a point in the establishment of the diagnosis.

The etiology is unknown, but with better and easier methods for complete examinations, more cases of Banti's syndrome have been found to be associated with syphilis.

In the differential diagnosis, the following must be considered:

(a) Splenomegaly of the Gaucher's type associated with pigmentation of the skin, liver enlargement without jaundice, and the presence of the typical Gaucher's cell in bone marrow as well as in the spleen.

(b) Splenomegaly in alcoholic or syphilitic cirrhosis of the liver usually following the symptoms of ascites and jaundice.

(c) Splenomegaly associated with pernicious anemia, but with the characteristic blood picture of pernicious anemia.

(d) Splenomegaly of hereditary type, as described by Minkowski, presenting a familial history of the disease, good general health, chronic slight jaundice, urobilin without bile salts in the urine, and characteristic fragility of the red blood cells.

(e) Splenomegaly in Hodgkins disease, accompanied by the general adenopathy which is characteristic.

(f) Splenomegaly in the leukemias, accompanied by the characteristic blood picture.

W. A. D. C. E. M. E. N. R. Admitted July 29, 1933.

Chief complaint.—Nausea, vomiting, hemorrhage.

Social history.—Enlisted man, United States Navy, 1907-22. Married 1925. Wife died 1932 of pulmonary tuberculosis. No children.

Past history.—Usual childhood diseases. Gonorrhea, 1908. Dermatitis, 1906, for which he was treated with salvarsan. No history of a penile lesion.

Present illness.—In January 1933 he had epigastric distress after meals. This was accompanied by dizziness, nausea, and vomiting of blood. At that time, his blood Kahn test was 4 plus. The spinal fluid Kahn test was negative. His stools were of dark color and they were positive for occult blood.

X-ray of the chest showed normal lung fields.

After a series of blood transfusions an exploratory laparotomy was done on April 28, 1933. An enlarged spleen and a large amount of ascitic fluid were found with no evidence of pathology of the gastrointestinal tract to account for the hemorrhage. The liver was apparently normal. The abdomen was closed without further operative procedures, on the assumption that the splenomegaly was due to syphilis, and that a course of antisyphilitic treatment should precede splenectomy.

On May 15, 1933, paracentesis was done and 4,200 cc of fluid were removed. On May 25, 1933, 3,500 cc of fluid were removed. On May 31, 1933, 3,200 cc of fluid were removed. He was discharged to home on June 23, 1933, and he was placed under intensive antisyphilitic treatment.

The red-blood cell count at this time was 4,500,000. The hemoglobin was 65 percent (dare). The white-blood cell count was 6,450. P. M. N., 68; Lymph, 32.

After 5 weeks, he had a sudden attack of vomiting of a large amount of bright red blood. About 24 hours later he was readmitted to this hospital.

Physical examination.—Age 43. Semiconscious. General appearance is that of exsanguination. Skin, normal. Mucus membranes, pale; E. E. N. T., no disease; chest, heart, and lungs normal; abdomen, slightly distended. A large spleen is palpable, firm, and nontender.

Genetalia, negative; extremities, normal.

A blood transfusion of 500 cc of blood by the citrate method was done on the day of admission.

On July 31, 1933, the red-blood count was 1,330,000, and hemoglobin was 10 percent (dare). A second blood transfusion of 500 cc of blood was done on this date.

On August 1, 1933, the blood count was as follows: Red-blood count, 2,050,000; hemoglobin was 38 percent (dare). A third blood transfusion of 500 cc of blood was done this date.

On August 9, 1933, the white-blood count was 6,000; mature, 74; bands, 3; young, 1; mono, 3; Lymph, 19.

On August 11, 1933, after a fourth blood transfusion of 500 cc blood, the red-blood count was 2,070,000 and the hemoglobin was 29 percent (dare). On this date splenectomy was done.

Histopathological report.—Specimen consists of an enlarged spleen measuring 16x9x8 cm. The organ is slate colored, the capsule is thickened and wrinkled, suggesting a recent loss of substance with a corresponding lessening of tension upon the enveloping fibrous capsule. On cut surface the color is beefy red and the consistency is firm.

Stained microscopic sections show the capsule to be thickened. The reticulum is increased and thickened to such an extent that the normal architecture is obscured. There is a slight increase in the number of sinuses and the individual sinuses are dilated and for the most part are empty. This fact suggests an organ that was congested and partially explains the wrinkling of the capsule. A moderate amount of infiltration of the reticulum by red blood cells and a few leukocytes is present. A few isolated collections of

lymphoid tissue are scattered throughout the section. Uo evidence of perivascular infiltration is noted.

Following operation, the gastrointestinal symptoms subsided, and dietary treatment for anemia was instituted. There was a gradual improvement in the blood picture over a 2-month period; and on October 13, 1933, the red blood count was 4,180,000, and hemoglobin was 85 percent. On October 16, 1933, he was discharged to his home. Antisyphilitic treatment had been given during the last three weeks of his stay in the hospital, and this treatment continued at home.

A recent report from the patient, ten months later, shows that he has been free of symptoms since his discharge from the hospital, and that he is now in good health.

Active antisyphilitic treatment has been given, but his blood Kahn test remains 4 plus.

SUMMARY AND DISCUSSION

Greene summarizes the symptoms of splenic anemia and Banti's disease by the words, "not a clinical entity." He believes that the symptom complex is a manifestation of syphilis or of malignant disease.

This case presents an exaggeration of the early symptoms of the symptom complex described by Banti, with profuse frank hemorrhages from the gastrointestinal tract, and anemia of the secondary type.

At the time of operation, involvement of the liver was not apparent. The case presents further evidence of syphilis as an eliological factor in the symptom complex known as Banti's disease.

This case would seem to indicate that antisyphilitic treatment alone will not cure cases of this nature and that a splenectomy is indicated as a primary procedure.

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DISLOCATION OF THE HEAD OF THE FIBULA WITHOUT FRACTURE

By W. D. SMALL, Lieutenant Commander, Medical Corps, United States Navy

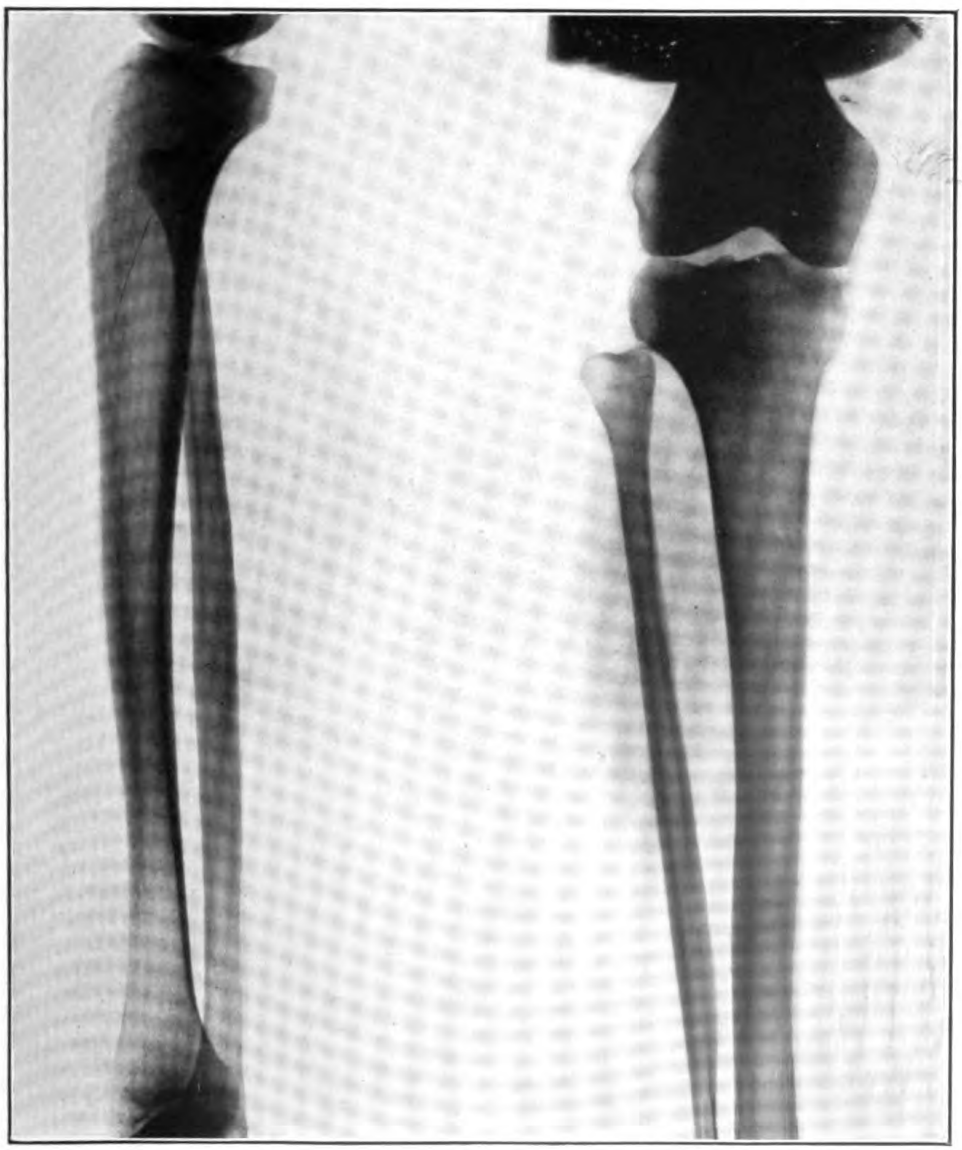
This instance of a rather rare injury is presented, first, as somewhat of a curiosity; and second, because current available authorities were not only in error as to certain features of the case, but were also of no assistance in the treatment.

C. B. C. Private, age 21, was admitted to the hospital late in the evening of September 1, 1934, for treatment of an injury to the right leg received about 1 hour previously. While going down an inclined gravel walk at a local amusement park, he slipped and fell forward with the right knee extended. He endeavored to regain his footing but on account of the loose, rolling gravel was unable to do so. He felt an immediate sharp pain in the region of the outer aspect of the right knee and upper leg.

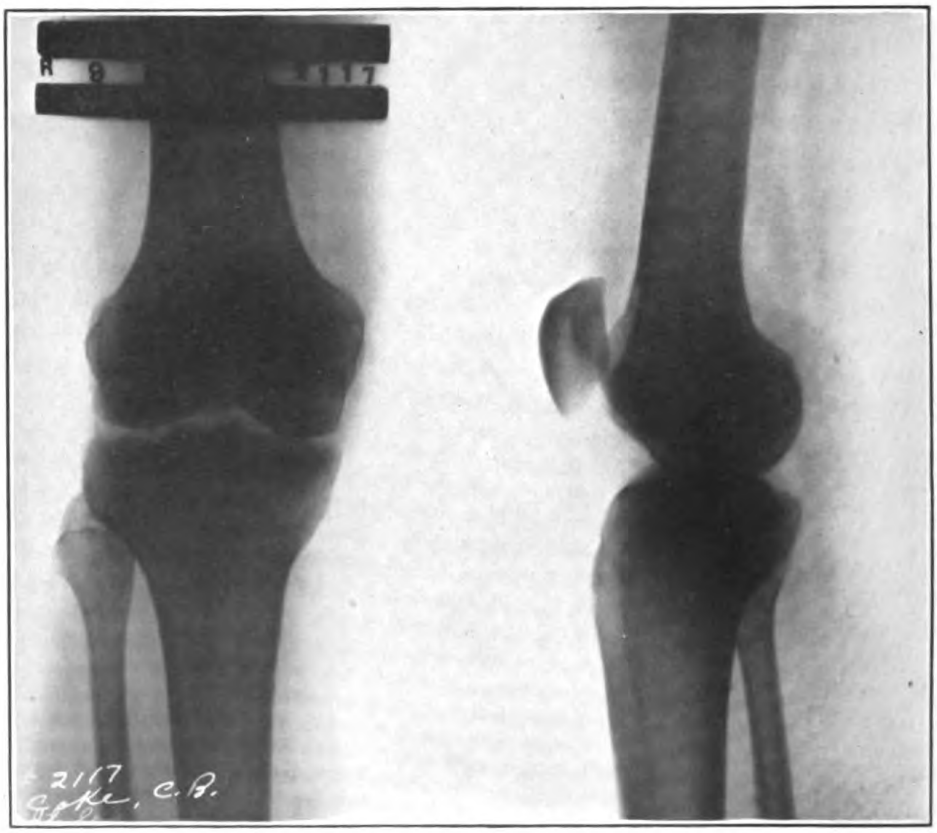
Clinical examination showed an undue prominence of the head of the right fibula anteriorly, with markedly increased tension of the outer hamstrings. Motions of the knee were smooth, and produced no particular pain except through the arc of approximately 135 to 90°, the hamstring tension being greatest at this point. Palpitation over the fibula produced moderate tenderness. There was no evidence of damage to anterior tibial or peroneal nerves, and no evidence of fracture of the bone. X ray showed the displacement very well, and it was apparent that the proximal end of the fibula was firmly wedged under the external tuberosity of the tibia. All immediate manipulative attempts at reduction were unsuccessful, so the limb was supported on pillows and left alone.

The following morning, a number of the better known contemporary authorities were consulted, and were singularly devoid of useful information on the subject. Such standard works as Wilson, and Cochrane, Scudder, and Nelsons Loose Leaf Surgery simply mentioned the condition and stated that it was rarely seen. Kellogg Speed, however, went into considerable detail as regards etiology and mechanics, describing a dislocation by muscular action similar to that which was the probable etiologic factor in this case. A manipulative measure for reduction was also described. Speed further stated that reduction was easily obtained by pushing the head into place with the fingers, but was difficult to maintain owing to poor retention by the torn ligaments.

Accordingly, further attempts at reduction were made. All the pressure that several of us could apply with fingers and hands was not successful, and the manipulative measures described by Speed were likewise failures. It was then felt that reduction must be obtained even at the risk of fracture of the bone by so doing. The area was thoroughly infiltrated with one percent procaine solution. Then, with the knee flexed to nearly 90° and the foot everted, the head of the fibula was driven back into place by use of a mallet and a block of wood, a small piece of felt being utilized to protect the skin under the wooden block. It was surprising how heavy a blow was required to accomplish reduction, and, when the head finally



CONDITION ON ADMISSION.



POST REDUCTION APPEARANCE.

returned to its normal position, it was with a snap distinctly audible to those nearby. After reduction the head seemed to be securely in place, and all ordinary movements of the knee failed to produce a recurrence. Nevertheless a light plaster of paris retention was applied for 2 weeks. It was also somewhat of a surprise to note in the check-up films that no fracture had been produced by the rather violent technique of reduction. It is believed that there is sufficient flexibility to the bone in an individual of this age to allow the necessary amount of bending to take place in the shaft.

The patient was discharged to duty with full, painless function 3 weeks after injury.

ACUTE MYELOBLASTIC LEUKEMIA

REPORT OF A CASE

By T. E. Cox, Lieutenant Commander, and E. Ricen, Lieutenant (Junior Grade), Medical Corps, United States Navy

Certain hyperplasias of the blood-forming tissues present fairly characteristic qualitative and quantitative changes in the cellular elements of the circulating blood. In these types of cases a definite diagnosis can sometimes be established on the basis of lymphoid or myeloid hyperplasia. In some forms of blood dyscrasias, however, no striking variations from the normal occur in the cellular constituents obviously making a definite diagnosis a more difficult problem. There are also those abnormalities of the blood-forming organs in which the initial picture is that of a panmyelophthisis or aplasia of all the blood elements, the true nature of the disorder being revealed only in the terminal course of the disease. It is this latter disorder which is usually erroneously called aleukemic leukemia, although a careful search will often reveal the true leukemic nature of the disorder by the presence of immature white blood cells despite the absence of the leukocytosis commonly associated with the leukemic state. Acute leukemic myelosis may and often does present a normal quantitative blood picture or even a leukopenia. These quantitative changes suggestive of an aleukemic state may however be coexistant with the presence of premature cells, which finally determines the true leukemic nature of the blood picture. It is especially this type of case which necessitates niceties of clinical skill combined with the assistance of the trained hematologist, in order to positively identify the particular hyperplastic element. The case which we report below belongs to this latter group of blood dyscrasias.

The patient on admission complained of a sore mouth and gums, weakness, and a discharging lesion on the left ankle. The blood picture at the time of admission was as follows: Red blood count, 2,290,000;

white blood count, 1,650; hemoglobin, 38 percent; granulocytes (polyneutrophils), 22 percent; and nonsegmented forms (large lymphocytes), 78 percent. On the basis of the ulcerative lesions in the mouth, the leukopenia, and the low percentage of granulocytes, a tentative diagnosis of granulocytopenia was made. Subsequent blood studies seemed to bear out this diagnosis. The white blood count varied between 750 and 1,450, red blood count between 2 and 3 million, granulocytes (polyneutrophils) between 14 and 44 percent, nonsegmented forms (large lymphocytes) between 56 and 86 percent, platlets between 15,000 and 34,000. On the fifth and eighth hospital days a transfusion of 500 cubic centimeters of whole blood was given with little change resulting in the blood picture. On the ninth day, 20 cubic centimeters of pentnucleotide were given intramuscularly, twice daily and continued for 5 days; 10 cubic centimeters of pentnucleotide were given twice daily until the patient expired. On the 18th day the white blood count jumped to 8,500, granulocytes (polyneutrophils) remained at 32 percent; large lymphocytes, 64 percent; myelocytes, 4 percent; red blood count, 2,320,000; hemoglobin, 36 percent. On the 19th day, the white blood count was 8,450; hemoglobin, 28 percent; red blood count, 2,040,000; granulocytes (polyneutrophils), 18 percent; Myelocytes, 7 percent; large lymphocytes, 71 percent; and band forms, 4 percent. At this time the patient was hemorrhaging profusely from the nose and mouth. Due to his poor general condition and in the hope of abating the hemorrhage, another transfusion was given. The patient expired a few minutes after completion of this transfusion.

K. M., age 45, Veterans' Administration patient, was admitted February 6, 1933, for observation, complaining of weakness, sore mouth, and a discharging sinus in the region of the left ankle.

Past and family history.—Negative.

Present history.—About 7 weeks previous to entrance, patient scratched his left ankle while putting on a shoe. This became infected and began discharging pus. A few days later, entire left leg became red and swollen. There was also an enlargement of the glands in the left groin. The swelling subsided in about 2 weeks, but the ankle continued to discharge pus. About 3 weeks previous to admission, patient's mouth and gums became very sore, making it extremely difficult to take nourishment. About this time, patient began to notice general feeling of weakness which persisted up until time of admission to hospital.

Physical examination.—Pale appearing, slender, developed man of 45, mentally clear and rational.

Head and neck.—Negative except for scattered ulcerative lesions about the gums and an ulcer about 1 cm in diameter, situated at the base of the frenum of the tongue.

Chest.—Symmetrical, expansion free and equal.

Heart.—No enlargement. Apex beat forceful. Rhythm rapid but regular. No murmurs. Blood pressure 115/75.

Lungs.—Negative.

Abdomen.—Negative.

Glandular system.—Negative.

Extremities.—There is a brownish discoloration of the median aspect of the left ankle. In the center of this discolored area is a draining sinus, 2–5 cm in diameter.

Reflexes.—All elicited and equal.

COURSE

February 7, 1933.—White blood count 1,650, granulocytes (polyneutrophils) 22 percent, nonsegmented forms (large lymphocytes) 78 percent. Red blood count 2,290,000, hemoglobin 38 percent. Numerous macrocytes present. Also poikilocytosis and central achromia.

February 8, 1933.—Smear from gum reveals numerous spirilli vincenti; numerous B. Fusiformis.

February 9, 1933.—Kahn, negative.

February 10, 1933.—White blood count 750, granulocytes (polyneutrophils) 27 percent, nonsegmented forms (large lymphocytes), 73 percent. Red blood count 2,680,000, hemoglobin 45 percent. Numerous macrocytes present. Also poikilocytosis and central achromia. Blood culture reveals strep nonhemolyticus, 500 cc blood by transfusion.

February 11, 1933.—White blood count 750, granulocytes (polyneutrophilis) 37 percent, nonsegmented forms (large lymphocytes) 63 percent. Red blood count 2,290,000, hemoglobin 37 percent. Numerous macrocytes present. Also poikilocytosis and central achromia. Temperature 103, pulse 104, respiration 32.

February 12, 1933.—White blood count 800, granulocytes (polyneutrophils) 44 percent, nonsegmented forms (large lymphocytes) 56 percent. Red blood count 2,150,000, hemoglobin 40 percent. Numerous macrocytes present. Also poikilocytosis and central achromia. Blood culture negative.

February 13, 1933.—White blood count 1,100, granulocytes (polyneutrophils) 40 percent, nonsegmented forms (large lymphocytes) 60 percent. Red blood count 2,450,000, hemoglobin 45 percent. Numerous macrocytes present. Also poikilocytosis and central achromia. 500 cc blood by transfusion.

February 14, 1933.—White blood count, 1,150; granulocytes (polyneutrophils), 22 percent; nonsegmented forms (large lymphocytes), 78 percent. Red blood count, 2,440,000; hemoglobin, 58 percent. Numerous macrocytes present. Also poikilocytosis and central achromia. Pentnucleotide, 20 cc, twice daily.

February 15, 1933.—White blood count, 1,250; granulocytes (polyneutrophils), 17 percent; nonsegmented forms (large lymphocytes), 83 percent. Red blood count, 2,290,000; hemoglobin, 60 percent. Numerous macrocytes present. Also poikilocytosis and central achromia. Blood culture negative. Pentnucleotide, 20 cc, twice daily.

February 16, 1933.—Pentnucleotide, 20 cc, twice daily.

February 17, 1933.—White blood count, 1,150; granulocytes (polyneutrophils), 36 percent; nonsegmented forms (large lymphocytes), 64 percent. Red blood count, 2,720,000; hemoglobin, 42 percent. Numerous macrocytes present, Also poikilocytosis and central achromia. Temperature, 101; pulse, 104; respiration, 32. Pentnucleotide, 20 cc, twice daily.

February 18, 1933.—White blood count, 1,000; granulocytes (polyneutrophils), 29 percent; nonsegmented form (large lymphocytes), 71 percent. Red blood count, 2,540,000; hemoglobin, 38 percent. Numerous macrocytes present. Also poikilocytosis and central achromia. Platlets, 15,240. Pentnucleotide 20 cc, b. i. d.

February 19, 1933.—White blood count, 1,250; granulocytes (polyneutrophils), 19 percent; nonsegmented forms (large lymphocytes), 81 percent. Red blood count, 2,320,000; hemoglobin, 38 percent. Numerous macrocytes present. Also poikilocytosis and central achromia. Platlets, 18,480. 10 cc calcium gluconate intravenously. Pentnucleotide, 20 cc, b. i. d.

February 20, 1933.—White blood count, 1,450; granulocytes (polyneutrophils), 22 percent; nonsegmented forms (large lymphocytes), 78 percent. Red blood count, 2,320,000; hemoglobin, 38 percent. Numerous macrocytes present. Also poikilocytosis and central achromia. Platlets, 13,920. Temperature, 103; pulse, 112; respiration, 30. 10 cc calcium gluconate intravenously. Pentnucleotide, 10 cc intramuscularly.

February 21, 1933.—Pentnucleotide, 10 cc intramuscularly.

February 22, 1933.—White blood count, 1,400; granulocytes (polyneutrophils), 14 percent; nonsegmented forms (large lymphocytes), 86 percent. Red blood count, 2,320,000; hemoglobin, 38 percent. Numerous macrocytes present. Also poikilocytosis and central achromia. Platlets, 16,240. Pentnucleotide, 10 cc, b. i. d.

February 23, 1933.—White blood count, 1,650; granulocytes (polyneutrophils), 15 percent; nonsegmented forms (large lymphocytes), 85 percent. Red blood count, 2,190,000; hemoglobin, 39 percent. Numerous macrocytes present. Also poikilocytosis and central achromia. Platlets, 19,710. Blood culture negative. Temperature, 103; pulse, 112; respiration, 30. Pentnucleotide, 10 cc, b. i. d. Patient bleeding from nose.

February 24, 1933.—White blood count, 8,500; granulocytes (polyneutrophils), 32 percent; nonsegmented forms (large lymphocytes), 64 percent; myelocytes, 4 percent. Red blood count, 2,320,000; hemoglobin, 36 percent. Numerous macrocytes present. Also poikilocytosis and central achromia. Patient hemorrhaging profusely from nose and gums. Pentnucleotide, 10 cc, b. i. d.

February 25, 1933.—White blood count, 8,450; granulocytes (polyneutrophils), 18 percent; nonsegmented forms (large lymphocytes), 71 percent; band forms, 4 percent; myelocytes, 7 percent. Red blood count, 2,040,000; hemoglobin, 28 percent. Numerous macrocytes present. Also poikilocytosis and central achromia. Platlets, 24,480. Temperature, 104; pulse, 120; respiration, 36. Blood transfusion, 500 cc. Patient expired at completion of blood transfusion.

A review of this case instantly reveals the diagnostic difficulties encountered by the clinician in disorders of the hemopoietic system. It was only during the last two days of the disease when the white blood count jumped to above 8,000 and myelocytes began to appear that any inkling of the true nature of the disorder became apparent. Even at this stage an absolutely accurate diagnosis was impossible, until the pathologist's report on the microscopic section of the bone marrow revealed the following:

"The marrow is packed with nucleated cells believed to be mostly immature granulocytes, myelocytes, and myeloblasts. Many of the cells are "smudges." Eosinophilic myelocytes are very numerous and occasional basophilic myelocytes are present; well differentiated polyneutrophils are very rare. Most of the nuclei are very pale and show nucleoli. Red cells are relatively rare and only an erythroblast is seen."

On the basis of the pathologist's report and the presence of immature white cells in the circulating blood a diagnosis of acute myelogenous leukemia was first established.

AUTOPSY

External description.—Body of a poorly developed and poorly nourished white male; apparently about 45 years; height 66 inches; weight about 120 pounds. All the skin surface is extremely pale. No lymph gland enlargement demonstrable; no edema. Just posterior to the left internal malleolus there is an irreguar oval ulcer 2 cm in diameter extending into the subcutaneous tissues. Over the insertion of right deltoid there is an oval dark red purpuric spot 3.0 x 5.0 cm surrounded by a ring of pin point to pin head purpuric spots about 8 cm in diameter. Pupils are regular, equal, moderately dilated. Conjunctivae and mucous membrane of mouth are very pale. Teeth in poor condition. All of the floor of the mouth in front of the tongue is the site of a gangrenous stomatitis; dark brown-red, foul smelling ragged tissue extending downward several centimeters into an indurated area under the skin of the submental area. The necrosis involves the inferior maxilla of this area and the incisor teeth are dislodged by slight pressure. There is a muco-sanguinolent discharge from the nose and mouth. The throat other than marked pallor shows nothing remarkable.

Abdomen is two fingerbreadths below the level of the chest.

M. L. F. 15. Fat has a yellowish tint.

Calcellous bone of gladiolus is a pale-brown color with a somewhat muddy appearance. The cancellous bone of rib is a pale-brown color.

Abdominal cavity.—There is no gross abnormality of the abdominal viscerae in situ.

Thoracic cavity.—The upper and middle lobe of right lung are united to the chest wall by dense adhesions. The left upper lobe is adherent to the chest wall posteriorly.

The pericardial sac is free and contains about 50 cc of straw colored fluid.

Heart.—Weight 380 grams. L. V. 17; R. V. 5. Myocardium is pale brownish red, fairly firm. All valves are smooth and pliable.

Aortic 1st measurement 72 mm. The intima is smooth and glistening. Both coronary arteries are smooth and pliable.

Lungs.—Voluminous, moist, crepitant throughout and very dark in color—bluish-black mottled with greyish-pink. On section there is nothing remarkable.

Spleen.—Weight 395 grams. Color pale grey-pink; consistency somewhat soft. Capsule shows numerous yellowish-white elevated discrete spots up to 3 mm. They are confined entirely to the capsule and on section are about 1 mm thick. Cut surface, pale reddish-grey. The markings are obscured.

Liver.—Weight 2,400 grams. Surface is smooth; consistency average. Color, pale brownish red. Cut surface, markings are obscured; color, pale brown; homogeneous throughout.

Gall bladder.—9 x 3 cm. Wall is smooth and pliable; filled with dark brownish-yellow ropy bile. Otherwise nothing remarkable.

Pancreas.—Weight 110 grams. Consistency fairly firm; color, pale yellow. Multiple sections show nothing remarkable.

Kidneys.—Weight 415 grams. Capsule strips with slight difficulty, leaving a brownish-purple slight granular congested surface. Cut surface pale pinkish-grey, slightly granular; markings somewhat blurred.

Adrenals.—Weight 20 grams. Cortex yellowish-brown. Medulla dark brown.

Bladder.—Filled with pale clear amber urine. Mucous membrane is quite pale.

Prostate and seminal vesicles show nothing remarkable.

Stomach.—Rugae are distincted. Mucous membrane is a pale brown red color and unbroken.

Small intestines.—Except for marked pallor show nothing remarkable.

Large intestines.—Answer to the same description.

There are no hemorrhages found in the gastro-intestinal tract.

Aortic 2nd measurement 42.

Aortic 3rd measurement 34.

The intima is smooth and glistening throughout.

Bone marrow of upper end of right femur is pale brown-red with a distinct muddy quality.

ANATOMICAL DIAGNOSIS

1. Blood dyscrasia of undetermined type (See microscopical report).
2. Gangrenous stomatitis; ulcer, left heel.
3. Hemorrhage, subcutaneous.
4. Splenomegaly.
5. Parenchymatous degeneration liver and kidneys.
6. Pleural adhesions.

Consideration of this case would not be complete without contemplating the possibility of the terminal blood picture being activated by the intensive course of pentnucleotide therapy administered. It is a be regretted that a biopsy with examination of the bone marrow in this case was not obtained prior to the administration of the pentnucleotide. In his experimental work on rabbits Charles A. Doan has definitely demonstrated that nucleic acid and its degradation products are capable of stimulating the hyperplasia of myeloid foci in the bone marrow. Microscopic section of the bone marrow in his cases showed definite myeloid hyperplasia. Immature forms were also found present in the circulating blood. Spleenomegaly was also a constant feature in the rabbits. These experimental findings coincide strikingly in many respects with our own findings of:

(1) An increase in the total white blood count following pentnucleotide therapy.

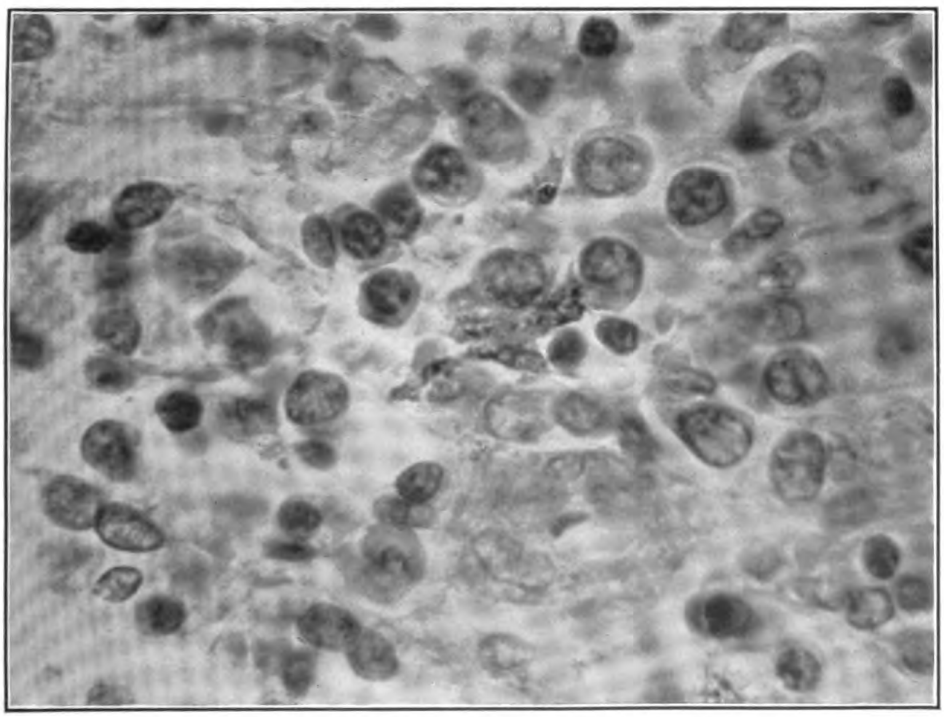
(2) Presence of immature forms (myelocytes) in the circulating blood.

(3) Stimulation of the myeloid foci in the bone marrow.

(4) Spleenic enlargement following pentnucleotide therapy.

The spleen in this case was not palpably enlarged at the time of admission prior to the administration of pentnucleotide. Whether or not the pentnucleotide could act as a hemopoetic stimulant, capable of producing a leukemic arrhythmia in the circulating blood, was never fully determined. Unfortunately the patient expired at the time the most interesting blood changes were occurring.

There is also the possibility that the true disorders was that of a marked depression of the entire hemopoetic system, due to some specific toxic effect of the original infection. To support this assumption we have the following evidence:



SECTION OF BONE MARROW (FROM UPPER SHAFT OF RIGHT FEMUR) SHOWING THE MARROW PACKED WITH IMMATURE GRANULOCYTES: MYELOCYTES AND MYELOBLASTS.

(1) The family history of this patient is entirely negative in regard to any blood dyscrasias. In addition, there is the history of previous good health until the onset of a severe infection involving the left ankle and leg, 7 weeks prior to admission.

(2) One blood culture position for nonhemolytic streptococcus at time of admission to the hospital.

(3) Blood studies before the administration of pentnucleotide revealed a leukopenia, a low percentage of granulocytes (polyneutrophils), a dimunition of red blood cells and platlets and also the total absence of any immature white blood cells in the circulating blood stream. The blood counts in this case prior to the administration of pentnucleotide were very similar to that of a fatal case of aplastic anemia or panmyelophthisis seen at the Great Lakes Naval Hospital some months previous. Microscopic examination of a portion of the sternum obtained at biopsy in this latter case was reported by the naval medical school as follows: "Section of the bone marrow shows marked hypoplasia of all the blood forming elements. Sinosoids contain fairly numerous erythrocytes with scattered normoblasts. No megakaryocytes are seen. Leucocytes are greatly diminished in number and are often degenerated with pyknotic nuclei. Scattered myeloblasts and myelocytes are present with maturing types. Some of the myelocytes are in mitosi."

Here again it is to be regretted that a biopsy of the bone marrow was not obtained prior to the administration of the pentnucleotide. If this had been accomplished it is entirely with the realm of possibility that such a biopsy might have shown a blood picture very similar to the report above rather than one indicating a leukemic state.

The diagnosis of acute myelogenous leukemia established in this case is more or less of academic significance, none of the therapeutic measures instituted materially affecting the clinical course of the disease.

We wish to emphasize, however, that in all disorders of the hemopoetic system it is wiser to withhold a clinical diagnosis until the patient recovers or microscopic sections reveal the true nature of the disorder. This statement is intended neither to reflect discreditably on either the clinician or hematologist, but rather to show the importance of withholding an opinion until we are reasonably sure of its accuracy.

CONCLUSIONS

1. In acute and chronic myelosis there is not always a constant increase in the total white blood count. The white blood count may be normal or a leukopenia may be present. The diagnosis of leukemia is established on the presence of immature white blood cells in the circulating blood.

2. Ulcerative lesions of the mouth occur as commonly in the various leukemic states, as in agranulocytic angina or granulocytopenia. Consequently, in all cases of gangrenous stomatitis, the leukemic disorders should be kept in mind.

3. The assistance of the hematologist and the pathologist is essential in making a diagnosis involving any affections of the blood-forming organs.

4. Opinion as to the nature of the disorder should be withheld until confirmation is obtained from the pathologist.

5. Nucleic acid and its degradation products are powerful stimulants of myeloid foci in the bone marrow. The administration of these substances may be responsible for the terminal blood picture rather than any inherent defect in the hemopoetic system. This fact should be seriously considered in making a diagnosis of any blood dyscrasia, in which nucleic acid derivatives have been administered.

REFERENCES

(1) Doan, Charles A., The Neutropenic State: Its Significance and Therapeutic Rationale. J. A. M. A. 99:194, July 16, 1932.

(2) MacCallum, W. G., Textbook of Pathology. Ed. 5. Philadelphia, Saunders, 1932. pp 764:67.

NAVAL RESERVE

MEDICAL CORPS

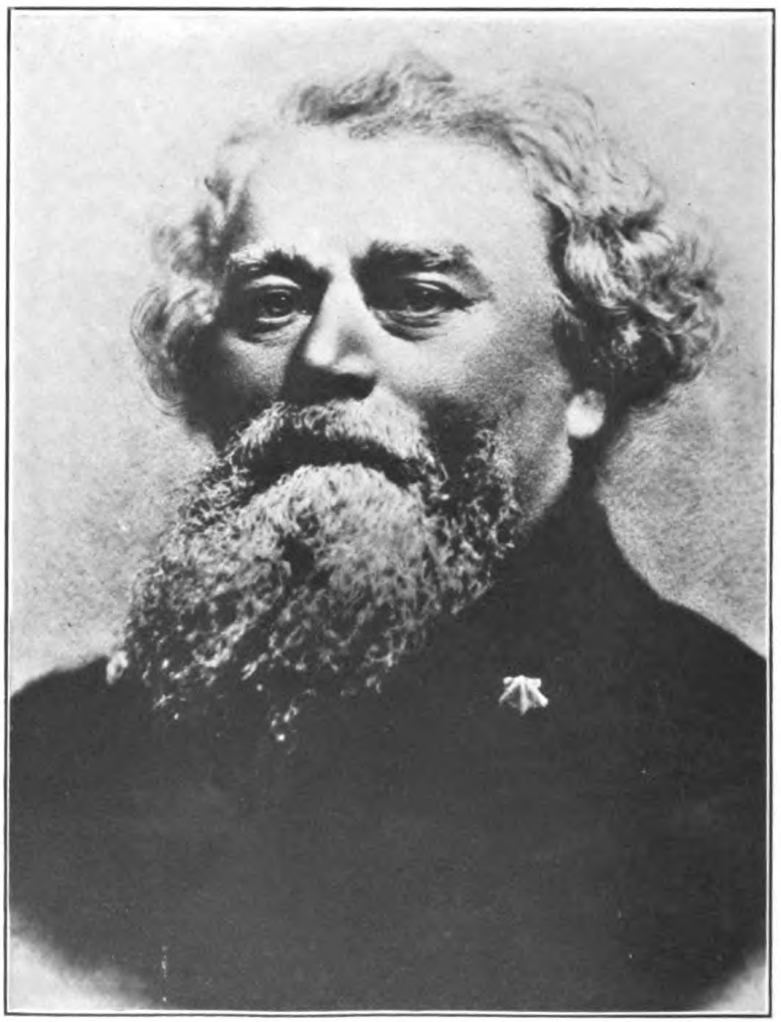
PROMOTIONS, FOURTH QUARTER, 1934

Name	From—	To—	Appointed
Meehan, George E	Lieutenant (junior grade), M. C.-V.(S.), U. S. N. R.	Lieutenant, M. C.-V.(S.), U. S. N. R.	Sept. 26
Monat, Henry A	Lieutenant (junior grade) M. C.-V.(G), U. S. N. R.	Lieutenant M. C.-V. (G), U. S. N. R.	Oct. 6
Hatchette, Charles V	do	do	Oct. 31
Livingston, Stanton K	do	do	Nov. 16

DENTAL CORPS

PROMOTIONS

Name	From—	To—
Clark, Edmond N	Lieutenant (junior grade) D. C.-V.(G), U. S. N. R.	Lieutenant D. C.-V. (G), U. S. N. R.
Nusbaum, Samuel L	do	Do.
Swanson, Arthur M	do	Do.



WILLIAM MAXWELL WOOD.

NOTES AND COMMENTS

WILLIAM MAXWELL WOOD

THE FIRST SURGEON GENERAL, U. S. NAVY, AND THE FIFTH CHIEF OF THE BUREAU OF MEDICINE AND SURGERY

By an act of Congress, March 3, 1871, it was provided that "the chiefs of the bureaus of medicine and surgery, provisions and clothing, steam engineering, and construction and repair, shall have the relative rank of commodore, while holding said position, and shall have respectively the title of surgeon general, paymaster general, engineer in chief, and chief constructor." The first to bear this new title of Surgeon General, though the fifth to hold office as Chief of the Bureau of Medicine and Survery, was William Maxwell Wood, of Maryland. He was born in Baltimore on May 27, 1809, and appointed an assistant surgeon in the Navy May 16, 1829. He was commissioned a passed assistant surgeon January 1, 1835, and surgeon February 20, 1838. His service included duty during the period connected with the suppression of piracy and the slave trade, the Seminole War, and the Mexican and Civil Wars. At the outbreak of the Mexican War he rendered a great service to his country by furnishing the earliest possible information to Commodore Sloat regarding the beginning of war. Dr. Wood had been ordered home from duty with the Pacific Squadron and was crossing Mexico from San Blas to Vera Cruz, bearing important dispatches. Learning of the opening of hostilities he sent a message to Commodore Sloat at Mazatlan which enabled that officer to begin the operations which resulted in taking possession of California. Dr. Wood's part in the acquisition of California was warmly acknowledged by Commodore Sloat, and the Chairman of the Naval Committee of the United States Senate in commenting on it, said, "Every intelligent mind must at once appreciate the importance of the service which you have rendered the country, and your personal hazard in traveling through the heart of the enemy's country, communicating with your military superior, and furnishing him with the sole and otherwise unattainable information upon which he based the acquisition of California. The importance of this acquisition can best be estimated by asking ourselves, what would have been our national position in the Pacific

and upon our Oregon frontier had Great Britain, instead of ourselves, acquired permanent possession of it? I have always contended that its acquisition constitutes one of the Navy's strongest claims upon the gratitude of the Nation, and this chapter in its history, furnished by your own service, but strengthens this conviction." Dr. Wood was fleet surgeon of the Asiatic, or as it was then called, the East Indian Squadron, from 1856 to 1858, and participated in the capture of the Barrier Forts below Canton. During the Civil War he was fleet surgeon of the North Atlantic Blockading Squadron from 1861 to 1864, and rendered conspicuous service in that capacity. He was appointed Chief of Bureau July 1, 1869, and was retired May 27, 1871. His death occurred at his home at Owings Mills in Baltimore County, Md., on March 1, 1880.

This first Surgeon General of the Navy was one of the most outstanding incumbents of that office. A man of vigorous intellect and much force of character, he rendered service of great value to the Navy during a long and useful career. He did much to enhance the prestige of the Medical Corps, and his strong common sense, and his persevering attention to any measure likely to improve the health and well-being of the officers and men of the Navy drew the attention and praise of all those concerned with administration of naval affairs during his time.

ARTICLES OF SPECIAL MERIT PUBLISHED IN THE NAVAL MEDICAL BULLETIN IN 1934

It has been customary for a number of years for a board to select from the articles published in the NAVAL MEDICAL BULLETIN during each calendar year, those having special merit. The writers of articles thus singled out then receive from the Surgeon General a letter of appreciation which, of course, is highly valued by the recipient and may be made a part of his official record. In recent years the Postgraduate Board has exercised the function of selecting these articles. In 1934 there were three selected to receive letters of appreciation. In addition, four others were selected for honorable mention, their names to be published in the BULLETIN at this time though without receiving special letters. The names of those to receive such letters, and those considered by the Board as deserving of honorable mention, follow, with the titles of the articles written by them set after their names:

Lt. (jg.) W. T. Buddington (M. C.), United States Navy. A Naval Medical Officer with the Civilian Conservation Corps. (July number.)

Lt. Comdr. R. P. Parsons (M. C.), United States Navy. Syphilis in the Navy. (October number.)

Commander Paul W. Wilson (M. C.), United States Navy. Incidence of Yaws and Syphilis in Five Rural Villages, Republic of Panama. (October number.)

HONORABLE MENTION

Lt. Comdr. S. S. Cook (M. C.), United States Navy. Insect Control. (April number.)

Lt. Comdr. David Ferguson (M. C.), United States Navy. The Human Response to Type I Pneumococcus Vaccine. (April number.)

Commander H. E. Harvey (D. C.), United States Navy. Nutrition in Relation to Dental Disease. (July number.)

Lt. Comdr. M. D. Willcutts (M. C.), United States Navy. Treatment of the Chinese Wounded at a Base Hospital in Peiping, China. (January number.)

The general character of many other articles was very high and it was only after the most careful consideration that the above selections were made by the Board.

SPECIALIST REQUIREMENTS OF THE NAVY IN 1935

Last year in the January BULLETIN there was published a brief note regarding the Postgraduate Board and one of its functions, that of appraising the specialist requirements of the Navy and selecting officers for postgraduate courses who desired to specialize. The requirements of the Navy a year ago and now are given below, the lists being arranged in order of the greatest need:

1934	1935
Clinical laboratory and pathology	Aviation medicine
X-ray	Dermatology
Neuropsychiatry	Clinical laboratory and pathology
Submarine duties	X-ray
Surgery	Submarine duties
Obstetrics and gynecology	Field service
Internal medicine	Obstetrics and gynecology
Eye, ear, nose, and throat	Neuropsychiatry
	Surgery
	Preventive medicine
	Tropical medicine

THE GEOGRAPHICAL DISTRIBUTION OF BACTERIA

The geographical distribution of plants and animals has been the subject of extensive study and indeed a separate branch of biological science, ecology, has grown up from this study. The geographical distribution of bacteria has received less attention than most other groups. Agricultural botanists have investigated the distribution

of nitrifying bacteria in some small areas but knowledge of distribution has lagged behind other fields of ecology. The importance of such knowledge is seen, however, if some of the experiences of the World War are recalled. No one, before the war, realized the toll that tetanus was to take and the necessity for large quantities of tetanus antitoxin and wholesale prophylactic immunization. Yet the Russian front was relatively free from tetanus and in sectors on the western front the number of cases were small. If exact knowledge of the geographical distribution of tetanus bacillus had existed, possibilities on the different fronts might have been foreseen and adequate supplies of the antitoxin provided where needed and appropriate administration orders for prophylaxis instituted in advance. This is only one example of the practical advantage of exact information regarding the distribution of bacteria. There is a field of research here for enterprising young bacteriologists to add to the sum of knowledge which may be of a most useful character.

ABUSE OF THE FLUOROSCOPE

Under this title an editorial appears in the October 1934 number of Radiology, drawing attention to certain features in the use of the fluoroscope of such importance that some of the criticisms and recommendations made are reproduced here for the benefit of medical officers of the Navy. Radiology is the official journal of the Radiological Society of North America. The editorial points out that the use of the fluoroscope in the diagnosis of tuberculosis, particularly to replace the regular plate, is most undesirable as it does not furnish the requisite diagnositic information supplied by the film. While the fluoroscope may be of value as an auxiliary aid in visualizing adhesions in general, the dangers to both patient and operator attendant upon its use, and its limitations in visualization of the lung compared to the film, make it only a secondary diagnostic method in tuberculosis. Its increased use in Europe during the last few years is due to its relative cheapness and it was resorted to because of the economic depression. The fluoroscope is only an auxiliary method, however, and cannot replace careful history taking, physical examination, and a good plate in the diagnosis of early tuberculosis.

THE DEATH RATE OF VARIOUS OCCUPATIONS

This is a subject of interest to all, and any new statistics are always welcome. The latest are those offered by the National Tuberculosis Association as the result of a 5-year study. The death rate of all

"gainfully employed males", from 15 to 64 years of age, was 8.70 per thousand. Compared with this, sailors and deckhands (merchant marine) had a rate of 17.28 per thousand. The relative dangers of handling horses and automobiles is expressed by a death rate of hostlers and stablemen being 36.22 and that of garage employees 6.55 per thousand. The lowest rate was for school teachers and social welfare workers, 4.42 and 2.75 per thousand, respectively. Among the professions, physicians are 10.69, clergymen 10.33, and lawyers 7.89 per thousand. The highest rate from heart disease was in unskilled labor, and the tuberculosis death rate for unskilled laborers was double that of other groups. The effect of social and economic status here is marked.

The death rate for the entire Navy in 1933 was 4.09. The death rate for officers was 6.77 per thousand.

IMPORTANT ANNUAL MEETINGS IN 1935

The following are some important meetings of medical and scientific societies during the year 1935.

The American College of Physicians, Philadelphia, Pa., April 29–May 3, 1935. This is the nineteenth annual meeting.

The American Medical Association, Atlantic City, N. J., June 10–14, 1935.

The American College of Surgeons, San Francisco, Calif., October 28–November 1, 1935.

The Association of Military Surgeons. New York City. Exact date not set but probably in October.

The American Public Health Association, Milwaukee, Wis., October 1935. This is the sixty-fourth annual meeting.

The American Pharmaceutical Association, Portland, Oreg., the first week in August 1935. This is the eighty-third meeting.

The American Dental Association, New Orleans, La., in November 1935.

The American Association for the Advancement of Science, Minneapolis, Minn., June 24 to 29.

The American Association of the History of Medicine, Atlantic City, N. J., May 16, 1935.

BOOK NOTICES

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

The Editor,
UNITED STATES NAVAL MEDICAL BULLETIN,
Bureau of Medicine and Surgery, Navy Department,
Washington, D. C.
(For review.)

AMEBIASIS AND AMEBIC DYSENTERY, by *Charles F. Craig, M. D., M. A. (hon., Yale), F. A. C. S., F. A. C. P., colonel United States Army (retired), D. S. M.* 315 pages, 53 illustrations. Charles E. Thomas, Baltimore.

In this timely and excellent monograph is presented a comprehensive summation of existing knowledge of human amebic infections. The epidemic of amebic dysentery in Chicago in 1933 focused attention on this condition and caused many physicians who had previously regarded amebiasis as a tropical disease to realize that *E. histolytica* is not restricted by geographic boundaries.

The author, an outstanding authority, has performed a real service by bringing together in comparatively small space so much valuable information.

There are chapters on etiology, epidemiology, pathology, symptomatology, complications, diagnosis, complement fixation tests, prophylaxis, and treatment.

Historically the book begins with the discovery of *E. histolytica* by Losch in 1875, includes the significant findings of the consultant board in Chicago in 1933, and concludes with a review of several articles which were published in 1934.

Craig emphasizes his belief in the nonexistence of avirulent strains of *E. histolytica* and of healthy carriers. He is of the opinion that any person who harbors this parasite is a potential if not actual clinical case of amebiasis.

In discussing prophylaxis the author states, "The prophylaxis of amebiasis, including amebic dysentery, depends upon a clear conception of the life cycle of *Endamoeba histolytica*, the methods of transmission of this parasite, its resistance to chemical and physical agents, and the most practical methods of applying this knowledge in the prophylaxis of the infection."

With regard to the drug to be used in treatment he states, "It is the writer's belief that we possess several drugs that are specific in the treatment of amebiasis and that there is little choice between them, if one carefully evaluates the results that have been published by those who have introduced them or have used them extensively."

OSTEOMYELITIS. ITS PATHOGENESIS, SYMPTOMATOLOGY AND TREATMENT, by *Abraham O. Wilensky, A. B., M. D., F. A. C. S., New York City, Fellow of the American Medical Association, Fellow of the American Association for Thoracic Surgery, Attending Surgeon to the Bronx Hospital and Dispensary, Associate Attending Surgeon to the Mount Sinai Hospital.* New York, The Macmillan Co., 1934. Pp. 433, ill. 104. $9.

For several years past important articles on osteomyelitis by this author have appeared from time to time, each one being an elaborate study of some phase of the disease. Now they have all been brought up to date and combined in one volume, the most complete monograph on the subject that the reviewer has seen.

The author has gone deeply into anatomy, bacteriology, pathology, and other aspects of osteomyelitis. In the chapter on roentgenology he stresses the fact that "Roentgenology has nothing to offer in the elucidation of an early diagnosis, and repeated roentgenological observations have amply demonstrated the fact that at the beginning and in the early stages of the development of a focus of osteomyelitis there is no evidence observable on a roentgenogram." Concerning the treatment of the acute condition he states that there is no need in the average case of rushing headlong into any operation, and one can afford to wait and observe the case for 24 or 48 hours. In the chronic stage, curetting or scraping the sinuses "is an operation of absolutely no worth, is a waste of time, and is one of the procedures which has brought the treatment of osteomyelitis to its hitherto low repute."

The chapter on osteomyelitis of the jaws is of the greatest importance and should be read by every dentist and every surgeon. This section is worth the price of the book. The relation of this condition to dental operations is so frequently observed in our hospitals and noted in medico-legal reports that dentists and surgeons should be conversant with the latest views on it.

Methods of treatment advocated by others are described in detail and fully discussed. It is unfortunate that M. A. Stewart's article on the picric acid-calcium carbonate treatment did not appear earlier so that Wilensky could include in his discussion a comparison of it with the maggot method and Orr's procedure.

It is an excellent book, worthy of close study by all surgeons.

A TEXT BOOK OF PATHOLOGY, by *E. T. Bell, M. D., Professor of Pathology in the University of Minnesota, Minneapolis, Minn.* Second edition, enlarged and thoroughly revised. Lea & Febiger, Philadelphia. Price $8.50.

This volume is a most excellent student's textbook and a valuable aid to the general practitioner. It has taken the subject out of the dead pathology class and deals with it from a pathological-physiology point of view.

The first seven chapters dealing with the predisposition to disease, mechanical, physical, and chemical agents, circulatory disturbances, retrogressive tissue changes, and inflammation are very concise and clear and form an excellent explanatory basis for many later subjects.

Throughout the pathology of disease is associated with etiology, signs and symptoms, and termination as actually observed.

It is a practical pathology for the majority.

Each subject is supplemented by a wealth of references for those interested in more detail.

A TEXT-BOOK OF PATHOLOGY, by *William Boyd, M. D., Professor of Pathology in the University of Manitoba.* Second edition, 1,047 pages, 416 engravings, and 8 colored plates. Lea & Febiger, Philadelphia. $10.

A work worthy to stand beside the great textbooks of Adami and Delafield and Prudden, was this book of Boyd's published 2 years ago. Its excellence is now further recognized by the demand for a new edition which includes many new facts. Among important new subjects dealt with are lead poisoning in children, tobacco hypersensitiveness in thrombo-angitis obliterans, the St. Louis type of encephalitis, and Cushing's work on the basophilic invasion of the pituitary gland in hypertension. There is a new chapter on the influence of heredity, constitutional diathesis and sex on the etiology and course of disease. A new chapter has also been added on diseases of the teeth. This book, with its excellent subtitle, An Introduction to Medicine, is one of the most important texts of the subject since the publication of Adami's great Principles of Pathology. Furthermore, in form, size, color, and cover design, as well as in printing and illustration, it represents one of the handsomest of medical volumes.

CATARACT: ITS ETIOLOGY AND TREATMENT, by *Clyde A. Clapp, M. D., F. A. C. S., associate professor of ophthalmology, Johns Hopkins University; professor of ophthalmology, University of Maryland; visiting ophthalmologist, Johns Hopkins Hospital and Wilmer Institute; ophthalmologist, University of Maryland Hospital.* Lea & Febiger, Philadelphia, 1934. $4.

We find in this volume of only 244 pages a treatise on cataract, a monograph valuable in its able and comprehensive manner of treatment.

Not alone have we been given a clear and concise story of the human lens from its development, but interwoven is an historical background which lends greatly to the delight in reading.

The assembly of the material from chapter I, The Development of the Human Lens, to chapter XXV, Aphakia and Its Treatment,

is so well mapped out and then so graphically written that the subject is placed before the reader in a most welcome manner.

Treatment nonoperative and operative, including pre- and post-operative care, is carefully handled and demonstrates that Dr. Clapp has a background, practical as well as theoretical, from which he has given this wealth of material in so small a space.

INTERNAL MEDICINE, by *John H. Musser, B. S., M. D., F. A. C. P.* Lea and Febiger. 1;296 pages. Price $10.

The second edition of Dr. Musser's book is keeping to the high standard set by the first edition. Considerable revision in text has been made, which brings the book up to date and incerases its value as a reference, both to the practitioner and the student.

Since each writer is an outstanding authority on his special subject, there is provided under one cover an unusual collection of information. Internal medicine being as developed as it is, has become too broad a subject for one writer to cover everything without assistance. It is much to the credit of Dr. Musser that he has so well chosen his contributors.

DEFINITE DIAGNOSIS IN GENERAL PRACTICE, by *W. L. Kitchens, M. D.* 958 pages. W. B. Saunders Co., Philadelphia and London. Price, $10.

This book is another work written by the specialist primarily for the general practitioner and consists of two parts, one a check list of symptoms and signs and the other a check list of diseases with a cross-index. Under any particular symptom are the diseases in which that symptom is to be found; under the disease is to be found a list of symptoms. It is an attempt to reduce diagnosis to the ABC plan. While of course that is impossible with a subject so extensive and intricate, such a book is a help and is one of those works that assist in giving one a birdseye view of a clinical situation that often is needed in making a diagnosis.

AUTONOMIC NERVOUS SYSTEM, by *Albert Kuntz, Ph. D., professor of microanatomy in St. Louis University School of Medicine.* Second edition, enlarged and thoroughly revised. 697 pages, 73 engravings. Lea & Febiger, Philadelphia, 1934. Price, $7.50.

This is a revision and enlargement of a splendid monograph on the anatomy and physiology of the sympathetic nervous system, with the latest experimental and clinical data.

DIABETIC MANUAL FOR PATIENTS, by *Henry J. John, M. D., director of the diabetic department and laboratories of the Cleveland Clinic.* Second edition. 217 pages. C. V. Mosby Co., St. Louis, 1934. Price, $2.

This is another useful little manual like that of Joslin, and a very excellent book. These manuals are indispensable for the diabetic patient for whom they are written, and a companion blessing to insulin. The diabetic may well rejoice in a statement of Dr. John's,

"How much, for example, would those afflicted with cancer or leprosy or sarcoma give if there were only some treatment which would assure them, as long as they adhered to it, a normal and useful life. Compare their lot to that of a diabetic who can look forward cheerfully to a long and useful life."

A MANUAL OF THE PRACTICE OF MEDICINE, by *A. A. Stevens, M. D., formerly professor of applied therapeutics in the University of Pennsylvania.* W. B. Saunders Co., Philadelphia, 659 pages, thirteenth edition, 1934. Price $3.50.

Everyone will welcome this excellent little book. Nearly every medical man has a copy of the 12th edition published in 1928, and will no doubt want a copy of the new edition brought up to date. There is an index of 24 pages that add to the usefulness of this extremely useful manual of the practice of medicine.

CONCEPTION PERIOD OF WOMEN by *Dr. Kyusaku Ogino, Head of Gynecological Section of Takayama Hospital, Niigata, Japan.* Medical Arts Publishing Co., Harrisburg, Pa. Price $1.

This is a pamphlet of 94 pages, giving what amounts to a popular exposition of the Ogino-Knaus Law, now well known to the medical profession. It contains excellent abstracts of the scientific work and observations done on the subject by both physiologists and clinicians and is an excellent little book for disseminating to both the laity and the medical profession what is probably the most important contribution to gynecology since the days of James Sims and Ephraim McDowell.

TUBERCULOSIS OF THE LYMPHATIC SYSTEM by *Richard H. Miller, M. D., Assistant Professor of Surgery, Harvard Medical School.* 239 pages, illustrated. The Macmillan Company, New York, 1934. Price $4.

This is one of the Macmillan medical monographs and covers the special subject in an excellent and authoritative manner. An interesting feature in the treatment is that while more than half the cases of cervical lymph node tuberculosis require some sort of surgical intervention, tuberculous retroperitoneal nodes rarely and then only such surgical procedure as is necessary for diagnosis.

ALLERGY AND APPLIED IMMUNOLOGY, by *Warren T. Vaughan, M. D., Richmond, Virginia.* Second edition. 413 pages, illustrated. The C. V. Mosby Company, St. Louis, Mo., 1934. Price $5.

Dr. Vaughan is well known for his work on allergy and has attempted here with marked success to write a book that would present authoritative information to the general practitioner in the simplest and yet complete manner. Features of the book are the fine maps and tables showing the distribution of allergic agents and the commonsense handling of the therapy.

SYNOPSIS OF GENITOURINARY DISEASES, by *Austin I. Dodson, M. D., Professor of Genitourinary Surgery, Medical College of Virginia, Richmond.* 265 pages, 111 illustrations. The C. V. Mosby Co., St. Louis, 1934. $3.

A small well written and useful compend of urology. This book is intended for the general practitioner and minor urologic procedures are described in some detail the major ones coming into the province of the urologist are given less fully.

DISEASES OF THE SKIN, by *Ernest Dore, M. D., Consulting Physician for Diseases of the Skin, St. Thomas' Hospital, London;* and *John L. Franklin, M. D.* 387 pages, 46 plates. D. Appleton-Century Co., New York, 1934. $5.

This is a small handbook compend of dermatology designed primarily for both medical students and general practitioners. It is a very good one, and in small space is given an excellent description of the diseases of the skin. A feature is the illustrations, remarkably clear and beautiful photographs of skin lesions.

RULES FOR RECOVERY FROM TUBERCULOSIS, by *Lawrason Brown, M. D.* 257 pages, no illustrations. Sixth edition. Lea & Febiger, Philadelphia, 1934. $1.75.

This is a new edition of a guide for the layman which long ago proved its value.

A TEXTBOOK OF HISTOLOGY by *Harvey Ernest Jordan, A. M., Ph. D., Professor of Histology and Embryology, University of Virginia.* Sixth edition, 738 pages, and 610 illustrations. D. Appleton-Century Co., Inc., 1934 Price, $8.

Although this book has been prepared primarily to supply the needs of undergarduate medical students in this essential basic science, it richly deserves a prominent place in all medical libraries. It covers the field of microscopic anatomy and cystology with admirable simplicity, and stimulates the reader's interest by constant references to the functions of the tissues under discussion.

In spite of the fact that the great bulk of the subject matter of histology is firmly established, rather extensive revisions have been made in this edition. The most noteworthy of the newer material lies in the subjects of the reticulo-endothelial system, blood, endocrine tissues, and the lymphoid organs. The text is richly supplemented by remarkably excellent illustrations which have been reproduced faultlessly by the printer, quite a number in colors.

This book is recommended unhesitatingly both to medical students and to those who wish a readable, practical, and authoritative reference volume on histology and histologic technic.

ESSENTIALS OF HISTOLOGY, by *Sir E. Sharpey-Schafer, formerly Professor of Physiology, University of Edinburgh. Edited by H. M. Carleton, Lecturer on Histology, University of Oxford.* Thirteenth edition, 618 pages, 721 engavings. Lea & Febiger, Philadelphia, 1934. Price $5.

Here is a textbook that has been in use for 50 years, it having been published when the author, now a man of 84 years of age, was in his early thirties. The book has certainly stood the test of time. One of the reasons for its continued popularity is that it was written

with the teaching aspect of the subject continually in view, being arranged in a series of 50 lesions requiring from 1 to 3 hours of lectures and laboratory for each. The descriptions of tissues are simple and accurate. A valuable feature is an appendix containing a summary of histologic methods.

SCIENCE AND PRACTICE OF SURGERY, by *W. H. C. Romanis, Fellow of the Royal College of Surgeons (England)*, and *P. S. Mitchiner, Hunterian Professor, Fellow of the Royal College of Surgeons (England)*. Fifth edition, 2 volumes, 1901 pages, 758 engravings. Lea & Febiger, Philadelphia. 1934. Price $13.

The first volume of this well known textbook covers general surgery, the second volume covers regional surgery. Adequacy of information and yet conciseness are the outstanding features, very desirable ones it must be allowed, in either a textbook or a manual of surgery. Each chapter constitutes a complete monograph at once authoritative and revised to include the most recent advances.

TEXT BOOK OF HISTOLOGY, by *E. V. Cowdry, M. D., Professor of Cytology in the School of Medicine, Washington University, St. Louis, Mo.* Lea & Febiger, Philadelphia, 1934. Price $5.50.

This book compares favorably with the author's previous works. Although not as complete as his editions on special cytology, it is well adapted for the medical student and physician.

The book is written in a most interesting style. Each chapter closes with a summary which is a step forward.

There are several illustrations. Some are semidiagrammatic, while others are well-made photomicrographs. The use of colored plates is an excellent addition to any book.

This book not only includes histology, but a brief reference is also made to physiology and embryology. The chapters on the reproductive organs include the present conception of hormonal activity.

The references are well chosen and mainly those of recent articles.

ADVANCES IN MEDICINE AND THE MEDICAL SCIENCES DURING THE YEAR 1934 [1]

The following is a brief résumé or calendar of the more recent advances in the clinical branches of medicine, as well as the medical sciences. An attempt has been made to confine it to discoveries or important advances that were made during the past year, though this has not always been possible as some of the work extends several years back and has only become recognized during 1933. Furthermore, it is naturally not possible in so brief a compass as a few pages to mention but the most outstanding achievements. With work so recent, too, it is difficult to appraise with absolute accuracy that which will stand the test of future experience.

The calendar of recent advances in the medical sciences published in the April 1933 number of the BULLETIN has proved so popular that it has been decided to repeat it for 1934. As in the résumé of last year, only the most outstanding events are mentioned and as far as possible limited to advances made in 1934, though this latter purpose cannot always be realized as the original work may have been done on a subject in previous years and brought to a final and successful conclusion in 1934. Of course, it is also obvious that when discoveries so recent have to be appraised without the trial of use which time alone can give, errors of commission or omission may naturally result.

MEDICINE

The treatment of chronic muscular dystrophy with glycin. This work by Authbertson and Maclachlan, published in the Quarterly Journal of Medicine July 1934, represents at last an effective treatment of these rare but hitherto hopeless cases.

An important advance in the attack on influenza was made during this year, and is the work of Andrews, Laidlaw, and Smith, published in the Lancet October 20, 1934. One difficulty in the study of influenza has been the fact that an animal susceptible to the disease was not known. These inestigators have found that the ferret is susceptible. It is believed by many that this discovery will, by offering an opportunity for laboratory experimental study of the disease, provide the key to the unsolved problem of this important disease. Of

[1] This material was prepared with the assistance of the staff of the Naval Medical School and Naval Hospital, Washington, D. C.

practical importance is the discovery that influenza virus, isolated in ferrets, may be propagated indefinitely in the brains of mice.

A notable contribution to dermatology is the work of Grutz and Burger of Bonn, on psoriasis. It has long been known that there was an increase of lipords in the scales. The two investigators named above discovered that the cholesterin in the blood was increased to a marked degree and that by drastically reducing the fat intake, allowing but 20 grams daily to adults and 10 grams daily to children, marked clinical results have been obtained.

Further confirmation of the work of Kracke and Parker of Emory University, as well as a number of other investigators, indicates that amidopyrine and other chemically similar remedies, are *the cause of agranulopenia.*

Successful use of *theelin in the treatment of involulutional melancholia* has been reported by Werner and others of the St. Louis University School of Medicine.

Treatment of the bite of the black widow spider by an immune serum has been successfully used by Dr. Fred D'Amour of the University of Denver.

The Nobel Prize for Medicine was awarded this year to three medical men from the United States, Dr. George Minot and Dr. William Murphy, both of Harvard, and Dr. George R. Whipple, of the University of Rochester, for their work in the treatment of pernicious anemia. Whipple in an elaborate study of hemoglobin regeneration, following secondary anemia, found liver the most effective agent to accelerate hemoglobin formation. Minot and Murphy then showed that feeding liver to patients with pernicious anemia was followed by complete remission and the disease remained in abeyance as long as liver in sufficient quantity was given. Their results were announced in 1926. The amount of the Nobel Prize this year is stated by the press to be $41,806, so that each of these medical men receives nearly $14,000.

The isolation of the purified protein derivative of the tubercle bacillus in crystalline form is reported in the American Review of Tuberculosis for December 1934, by Dr. Florence Seibert.

In 1934 *the hormonal mechanism of nerve action* on cells was demonstrated: Animal hearts were subjected, some to vagus excitation, some to sympathetic stimulation, for considerable periods. The typical physiological reaction was found to be due: In the case of vagus stimulation to the formation within the cell of the depressor substance, cholin (?); in the case of sympathetic stimulation, to the elaboration of a substance indistinguishable from epinephrin. The extract of a heart so excited transferred to a normal heart produced effects appropriate to the kind of stimulus used on the extracted

heart, thus proving that nerve stimuli act through the production of specific chemical substances within the cell.

A new antidote, seemingly specific for acute mercury poisoning, has been discovered and successfully used by Dr. Rosenthal, senior pharmacologist, National Institute of Health, Washington, D. C. The experimental work was reported in the Journal of the American Medical Association, April 21, 1934. The drug, sodium formaldehyde sulphoxylate, is given intravenously in amounts (usually 10 to 15 grams) to confer on the blood, for several hours, the ability to reduce bichloride of mercury. Gastric lavage is done through a stomach tube with 5 percent solution of sulphoxylate and 200 to 300 cubic centimeters of this solution is left in the stomach.

The total ablation of the normal thyroid in congestive heart failure, and especially for the relief of angina pectoris has produced spectacular results in many cases. This therapeutic procedure is credited to Drs. Blumgart, Berlin, Davis, et al. The reports for the year 1934 seem to confirm their prophecy for therapeutic results.

A study of several thousand cases of malaria treated with atebrin by A. N. Kingsbury, director of the Institute for Medical Research of the Federated Malay States, reported in the Lancet for November 3, 1934, indicates that atebrin may in some instances cause a psychosis. There is a suggestion that the daily dose of atebrin be reduced to 0.2 gram daily in place of the 0.3 gram usually given at present.

SURGERY

Divinyl ether as a general anesthetic has been carefully studied by Leake, Goldschmidt, Ravdin, Beach, Lucke, Bourne, and Sparling. They believe it has certain advantages over ethyl ether, the most important of which are rapid narcosis (1½ to 2 minutes), and almost equally rapid recovery without postoperative vomiting. The anesthetic does not appear to cause appreciable damage to the liver or other internal organs.

A new treatment for osteomyelitis is described by Stewart in the January 1934 issue of Surgery, Gynecology, and Obstetrics, making use of picric acid and calcium carbonates. This is virtually a modification of the maggot treatment in that it is an attempt to discover the reason for the benefit resulting from the use of maggots. It was found that a biochemical effect from the excretion by the maggots of calcium carbonate in certain definite amounts into the wound led to stimulation of phagocytosis. It was believed also that the maggots absorbed the exotoxin of the infecting bacteria and rendered it nontoxic. It was found that when the calcium carbonate was combined with a specially prepared solution of picric acid the same result as with maggot infestation of the wound resulted. Clinical results

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were very gratifying and gave promise that this may be an improvement over the ordinary maggot treatment (more rapid healing, better control of treatment, and elimination of possible cross infection).

OBSTETRICS, GYNECOLOGY, AND PEDIATRICS

The most important event in the year is the general acceptance of the *Ogino-Knaus law as to the time in the menstrual cycle of ovulation.* Exact knowledge regarding this period is of great importance as furnishing a simple method of contraception requiring no chemical or mechanical methods and meeting with little opposition on religious or ethical grounds. It is also of paramount importance in the treatment of sterility and is probably the most important advance in the history of gynecology since the time of Sims and McDowell.

The effect of posture and voluntary muscular effort on intra-abdominal pressure in women and its value in the second stage of labor was studied and reported by Mengert and Murphy in the December 1933 number of Surgery, Gynecology, and Obstetrics. They find a 30 percent increase in pressure in the sitting position over the Sims position, due to visceral weight and better purchase for muscular action. They recommend "a more liberal use of the sitting posture during the second stage when it is desired to expedite labor." Dr. Kathleen Vaughan (work reported in the Lancet, Feb. 17, 1934), recently advocated the sitting or squatting posture as the result of its effect on increasing the antero posterior diameter of the pelvis. She found an increase of 1 to 1.5 centimeters gain in the true conjugate, an increase which, as she says, "would often suffice to turn a difficult labor into an easy one."

It is well known that the gonococcus does not readily attach the pavement type of epithelium of the adult vagina. By the *use of amniotin by mouth temporary change to this type of epithelium is brought about in the vagina of children with gonorrheal vaginitis, leading to remarkable curative results.*

One of the important events of the year in obstetrics and pediatrics *was the birth of the Dionne quintuplets.* This is the only authentic case of the delivery and survival of quintuplets. As the word "quintuplets" is now on many tongues, it may be pertinent to say that our standard dictionaries place the accent on the first syllable.

What is believed to be the youngest mother with a living child described in medical literature is reported by Dr. Hilda L. Keane of the Victoria Zenana Hospital of Delhi, India. The mother, seven years of age, was delivered by Cesarian section of a living baby girl weighing 4 pounds, 2 ounces, not premature. The mother weighed 48 pounds and had nearly all her deciduous teeth. The baby was nursed for 9 months and was perfectly healthy.

PUBLIC HEALTH AND PREVENTIVE MEDICINE

The Surgeon General of the United States Public Health Service in his report states that *the general death rate in the United States for the calendar year 1933 was the lowest ever recorded in the United States*, 10.5 per thousand. There were no wide-spread epidemics. Tuberculosis, typhoid, and diphtheria all showed the lowest recorded rates. There were two considerable local epidemics in 1934; one of encephalitis of a somewhat unusual clinical type in St. Louis and surrounding country, and an epidemic of poliomyelitis in the Pacific and northern Rocky Mountain States.

Dr. John A. Kolmer reports promising results with *a vaccine against infantile paralysis*. This is a sodium ricinoleate preparation from the spinal cords of infected monkeys. Animal and human subjects vaccinated with this vaccine showed the presence of large amounts of antibody, and a small series of school children has been vaccinated whose future medical histories will be carefully observed.

The cysts which transmit amebic dysentery can be filtered out of water by the usual filtration methods, according to the report made by the United States Public Health Service and chemists of the Chicago Department of Public Works, after experimental study of the problem.

EMBRYOLOGY

The discovery of giant chromosomes in the salivary gland cells of the yeast fly was made in 1930 by Heitz. Their size is 70 times that of the largest previously described chromosomes. Painter, Bridge, and Morgan, Nicolai and Koltzkoff were able to describe and map the gene bands of part of these chromosomes. Spoken of somewhat enthusiastically by the press as the "Rosetta Stone of Genetics", embryologists have expressed the belief that the knowledge of chromosome structure which will now become available holds the key to many important problems of heredity.

PATHOLOGY

Plans were made in 1934 whereby, in cooperation with the American Society of Clinical Pathologists, the United States Public Health Service will make *a careful study of the comparative value of serologic tests for the diagnosis of syphilis.*

PHYSIOLOGY

Dr. E. C. Kendall of the Mayo Foundation has succeeded in obtaining in pure crystalline form and establishing *the chemical formula of cortin*, the hormone of the adrenal gland cortex.

The fact that *growth and development is the result of a balance between the thymus and pineal* glands is practically established by the work of Rowntree, Clark, and Hanson.

What is probably a new vitamin has been found in seeds by H. Dam of the University of Copenhagen. This is believed to be an antihemmorrhage factor.

RADIOLOGY[1]

The outstanding development in radiology in 1934 is the *construction of a new 1,000,000-volt machine* capable of developing energy equal to 350 milligrams of radium—a triumph of electrical engineering as well as of radiology. This will be installed in the Soiland clinic in Los Angeles. With former high-voltage machines the tandem tube was used to raise the energy in the second tube. In practice it was found that the electron stream would not flow continuously into the second tube—some of the electrons flew off at a tangent puncturing the tube wall. In the new machine a porcelain cast tube is used.

The second outstanding feature of radiology is *the development of short-wave therapy* in the treatment of furuncles, abscesses, sinus, and joint disease. The new 16-meter machine is a marked advance over any previous machines in stability of output, compactness (weight about 40 pounds), and simplicity of operation and cost.

PROGRESS IN THE STUDY OF CANCER

The study of cancer producing substances such as 2 benzpyrene has led to inquiries into hormones such as oestrin, the molecular structure of which is similar to the cancer producing coal-tar fractions. The use of this hormone on the skin of male mice produced changes in the prostate suggestive of the early growth of a prostatic tumor. The prostate was greatly enlarged due to multiplication of cells and change in their character. Obstruction of urinary flow occurred. It has also been noted that there is an increase in the hormone content in tissues undergoing neoplastic change.

Another fundamental advance in the study of cancer is *the discovery of anticancer bodies* which have a lethal effect on cancer cells and not on normal cells. These antibodies adhere to the serum globulin fraction called englobulin. This fraction has been concentrated and when dried retained its potency for 3 or 4 months. Although no treatment except on experimental animals is possible as yet it is a promising field of cancer research.

[1] Supplied by Lt. Comdr. Otis B. Spalding, Medical Corps, U. S. Navy, Naval Hospital, San Diego.

It has long been known that the *mineral oils used in the textile industry may produce neoplasm in workers such as spinners of certain classes. Some of these oils are more carcinogenic than others* and this seems to be related to the geological strata from which they are obtained as well as the viscosity of the oil. Detoxification methods of these oils have been devised.

Assay of muscle tumors shows the presence of "cortin", neurogenic tumors "intermedin", and lipomas "prolan." One lipoma had 10,000 rat units per kilogram of prolan. There is a large amount of estrin in fibroadenomaous breast 6,000 rat units per kilogram.

THE DIVISION OF PREVENTIVE MEDICINE

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

A CONSIDERATION OF THE PROPHYLAXIS OF ACUTE CEREBROSPINAL FEVER

By O. J. Mink, Captain, Medical Corps, United States Navy

With the outbreak of the World War, epidemic cerebrospinal fever attacked the mobilized forces with a furor which was almost demoralizing. It became pitifully evident how little we knew about methods of preventing the development and spread of this disease. This appears most unusual when it is considered that the etiological organism had been established beyond a doubt for many years. Almost two decades have passed since the great test of 1917 and it seems appropriate to review the progress of the past years in the control of this form of meningitis and seriously to ask ourselves a few questions which would need to be answered if today we were compelled to mobilize for a great war. Are we better prepared in 1934 to prevent the appearance and limit the spread of epidemic cerebrospinal fever than we were in 1917? Have the lessons of the past years made diagnosis more accurate and treatment more efficient? The discussion of these two questions is of vital importance, for unless they can to a considerable extent be answered in the affirmative, the outlook in the solution of a future problem is not encouraging. To hope that meningitis will be absent as a mobilization problem is to deny the lessons of history and to indulge in an optimism which the facts do not warrant. Can a disease which is a cause of concern in the small commands of peace time be expected to become less of a problem when thousands of recruits are mobilized and hurried through the stress of training?

A review of the history of the disease leads us to define meningitis, from a military standpoint, as an acute infection which occurs in young recruits during the winter season. No facts were more undeniably demonstrated in 1917 than the influence of youth and season on the spread of the disease. The inexperienced youth of the twenties, under the influence of inclement weather, forms the food for the meningococcus. Many other factors contribute to season and

render this food more palatable to the causal organism. The combination of these factors, to which is added, of course, the meningococcus, produces the annual curve of incidence of cerebrospinal fever. The curve is usually in the form of an inverted V and covers the bad seasons of the year from October to March. Unseasonable weather in the spring may drag out the decline as far as June. The larger the numbers concerned the more regular the V and the greater the number of months covered by the curve. In the warm days of summer the disease disappears. Some authors deny this disappearance, but they do not refer to epidemics. Summer, spring, and winter are here used as terms to describe not seasons but weather. A spring with winter weather will not lessen meningitis as was so well demonstrated in the wintry June of 1917.

With the outbreak of meningitis in 1917, the authorities on this disease gave their approval to three measures which were in their opinion of particular importance in the prevention and control of this form of meningitis. These three measures were:

1. Extreme ventilation.
2. Increase in floor area per man.
3. The isolation and segregation of carriers.

It is proposed to consider these three measures in detail and to determine from past experience the value, if any, which each measure may have.

By ventilation we mean the maintenance of a supply of fresh air adequate to give a plentiful supply of oxygen and to remove the volatile waste products, particularly respiratory, of the human body. The influence of fresh air of proper temperature and moisture content delivered without the formation of a draft will be admitted by all as a sanitary measure of great importance in the maintenance of health. This was not the idea of the fresh air enthusiast of 1917. He demanded a maximum of air. It mattered not if the air came in a gale at a temperature of 30° or 100°. It might be laden with moisture or completely dried. The goal sought was always more fresh air. Observers state that at the height of this hysteria windows were removed from barracks and the occupants shivered and pulled their heads under the blankets. Here they breathed and rebreathed their three cubic feet of air, unappreciative of the advantages of ventilation about them. After shivering through a sleepless night they started the day's work exhausted and facing the prospect of a repetition of such nights and days. A respiratory infection followed and they became easy victims to the meningococcus or the pneumococcus. The conservative sanitarian said perhaps there was too much fresh air, but the enthusiast said, "The ventilation should be increased."

Under conditions similar to those described, one barracks rebelled, nailed their windows shut, and defied anyone to touch them. As an experiment, they were allowed their way, and no cases of meningitis developed. One cannot avoid the conclusion that ventilation which produces discomfort and prevents sleep because of chilling, excessive air currents, and dampness due to the entrance of snow or rain is not beneficial and may be more injurious than a room which is comfortable although close.

The question of floor space per person embraces not alone ventilation and the amount of air per man, but is of especial importance from the standpoint of mechanically separating the men from each other. The value of the latter phase of floor space as a means of preventing the spread of disease cannot be denied.

At Great Lakes in 1917 when cerebrospinal fever, pneumonia, and other infectious diseases were raging, an inspection party noted the great crowding in the Public Works Regiment and were surprised to learn that this regiment was practically free from contagion. This incident is not an argument in favor of less floor space, but shows that in the case of these men who were older a higher age group were able to withstand the bad effects of overcrowding.

If we consider extremes it must be evident that when men maintain at all times a separation of, for example, 100 feet, disease cannot be transmitted. On the other hand, if human beings are packed together as animals in a cattle car conditions must be optimum for transmitting disease. Chances of infection probably vary but little for short distances and at a certain distance begin to decline rapidly and quickly disappear. The actual point of disappearance is unknown but the distance is probably too great to be compatible with military efficiency. The necessities of a war-time mobilization will probably never allow men to have sufficient room to allow floor space to be a factor in preventing the spread of infection. An artificial separation and a relative increase in floor space may be produced by the use of screens and only by such means will it be possible to allow a degree of separation sufficient to influence appreciably the chances of infection.

Of all measures brought forth for the control of meningitis without doubt the most promising and the most spectacular was the idea of detecting and isolating the carrier of meningococcus. The plan provided that the carrier should not again associate with his fellow man until free from meningococcus. It was contended that a certain number of human beings harbored in their throats gram negative diplococci agglutinable by antimeningococcus serum; that these organisms were the meningococci and that the carriers thereof passed these organisms to nonimmune persons causing cerebrospinal fever. The organisms certainly appeared by all tests to be meningococci.

It appeared, if the early writers can be credited, that once the technical difficulties of the bacteriology involved were overcome a great advance in the control of meningitis was in sight. It was assumed though unproved that the organism carried was virulent and that the carrier owed his freedom from disease to a natural or acquired immunity. At Great Lakes the theory of the immunity of the carrier was disproved by the appearance of a liberal percentage of cases of the disease among the carriers. The virulence of the organism carried has not been proved, but it must be assumed unless the carrier theory is entirely emasculated. Assuming the organism to be virulent there remains the following proposition which is at least unusual in medicine, i. e., a virulent pathogenic organism lives and multiplies in the tissues of a nonimmune without producing disease. It is proposed later to consider the question "Is not the carrier actually suffering from a disease?"

In any consideration of the importance of the carrier theory it is necessary to determine the frequency and the laws, if any, which govern the creation and continued existence of the carrier. In 1919, Short published a chart covering these points and based upon observations of 60,000 cultures over a period of 15 months. This chart demonstrated the seasonal variation in the percentage of carriers and the relation of the percentage to such factors as rainfall, snowfall, high and low temperature, and particularly sudden changes in weather conditions. In the summer the percentage was as low as 2 percent while in the winter it often rose as high as 50 percent. Recruits on arrival at the mobilizing station showed as high as 25 percent carriers in the winter. Warm bright weather reduced the percentage and raw weather, particularly if combined with sudden changes from warm weather, caused a rapid rise. Carriers in an isolation camp cleared rapidly with the warm sunshine of spring and those who remained were usually the chronic cases who form the 2 percent of the summer time.

These variations in the carrier rate are of interest when studied in connection with the percentage incidence of meningitis. If the carrier causes the disease, cause should precede effect and the increase in percentage of carriers should precede the increase of cases. The two curves should be separated by an interval representing the incubation period of the disease. Short's work,[1] however, shows that the two curves almost coincide, with a little tendency for the carrier curve to lag. This would indicate that one condition is not dependent upon the other, but rather that both carrier rate and disease incidence are affected by the same factors. These factors have been considered in connection with the carrier rate and it is pro-

[1] Journal of Infectious Diseases, August 1919.

posed later to consider the same factors and additional ones at greater length in relation to disease incidence. Earlier writers believed they established the carrier theory by detecting carriers in groups of men among whom meningitis had developed. If this has value as positive evidence then similar negative evidence must be considered. Upon repeated occasions, groups of men showing over 50 percent of carriers were free from the disease while groups of men containing less than 10 percent carriers showed numerous cases of meningitis. In one instance a carrier giving positive cultures for 2 years lived with 75 men for 12 months and no cases of meningitis developed in the group. There can be little doubt that the carrier work failed to influence the curve of the disease and failed to control the spread of the infection.

Before dismissing this subject, it may be of advantage to call to the attention of those health authorities, to whom the theory of isolation of carriers may suggest brilliant possibilities and an easy solution of the meningitis problem, the seriousness of the additional problem which this theory may add to the meningitis question. A health officer once asked me about the best method of detecting carriers. My problem at the time was how to dispose of carriers already detected. Carriers can safely be estimated as 10 times as numerous as cases and their final disposition offers a problem incomparably more difficult than the handling of clinical cases of meningitis. Innumerable methods have been devised and recommended for ridding the carrier of his offending organism and permitting his restoration to the society of others. None offer results which are other than confusing and disappointing. Sunshine and warm weather will clear a carrier camp in a few weeks but the same factors will end a meningitis epidemic. However, the problem has not yet been solved. It has merely solved itself until the next season arrives. The routine isolation of large bodies of men in idleness each winter infuriates those involved, disgusts the authorities, and raises doubt in the minds of the friends of public health. Such a measure is only excusable when the efficiency of the method as a preventive measure can be demonstrated beyond a reasonable doubt.

A perusal of the preceding pages might lead one to the conclusion that the outlook for the future was indeed gloomy and that meningitis epidemics of the next mobilization must be allowed to take their course. While the studies of ventilation, floor space, and carriers have not a tendency to cause them to be recommended as a solution of the problem, nevertheless it is believed that much data have been obtained which if properly applied is of constructive value.

I feel that this subject cannot be introduced more clearly than by quoting Short's conclusions from his studies:

"It is not the intention in this paper, however, to assign the reason for the occurrence or nonoccurrence of cerebrospinal fever to meningococcus carriers, but rather to call attention to the multiplicity of factors which may be reasonably expected to operate alone or in combination to influence this incidence, to emphasize that the meningococcus carrier is only one of the factors of which crowding, over-training, immaturity, fatigue, exposure to weather, and other factors discussed in a previous report are probably equal, if not greater in importance, and that to concentrate our attention on any one factor to the exclusion of a proportionate regard for the others, is to neglect some of the opportunities for controlling the situation."

It is important to consider minutely some of the factors which Short mentions as influencing the health of the recruit and rendering him more susceptible to disease especially meningitis.

To more fully appreciate these factors and to understand the changes to which a young man is subject upon becoming a recruit, an attempt will be made to picture the life of the average young man of 18 to 20 years of age in civilian life and then to draw another picture of him after he enters recruit training, showing the numerous and often violent changes in habits of living to which he is subjected within a period of a few weeks.

The average young civilian of 18 is to a large extent his own lord and master. He may acknowledge the authority of his employer or his schoolmaster in that by them his activities during a portion of the day are somewhat limited. In his home he may see fit to obey his parents and govern his habits according to their wishes. It is probable, however, that he is not greatly influenced by the wishes or wisdom of his seniors and has learned to regulate his habits according to his own desires and without relation to the well-being or convenience of others. In the matter of clothing, his sensation of comfort causes him to adopt heavy or light garments at his pleasure. He judges the necessity for an overcoat, rubbers, gloves, or ear muffs. His throat and arms are covered or bare as he pleases, with only due regard for the conventions of dress. At night his bed is in a room which is heated and ventilated to suit his convenience and his bedding is suited to his individual ideas of the needs of the case and all are subject to such change as necessity may indicate is advisable.

In the morning the young man arises at an hour suitable to himself and subject only to the requirements of breakfast and work or school. Breakfast and other meals are what experience has trained his fancy to choose. Work or school only restrict his activities a few hours a day and he can quit either with little punishment or discomfort, covered by checkage of pay or loss of standing. His

duties completed, his time until he chooses to retire is his own. He may do as he pleases provided he avoids contact with the police.

How does this picture change when our care-free young man becomes a recruit? His clothing is no longer what he wants or thinks he needs but what years of experience have shown to be best suited for the Navy man in general. Experience has selected wisely but our young recruit has a hard time making the change. His neck is now bare while formerly he always protected it from the cold. All other articles of uniform are prescribed to cover the needs of the majority and not according to the needs of the individual. His duties cover long hours during the day, are severe for one who is not hardened physically, and if he feels indisposed and unenthusiastic he cannot avoid them by accepting a penalty of loss of pay or loss of job. Medically he is either sick enough to be excused from work or well enough to work. His status in this respect is decided by others and not by himself. His food is what hygienists have decided is best for large bodies of men. Experience has shown that it is high in nutritional value but it is not prescribed for the individual fancy but for the best interests of the many. At night he retires to a hammock which, to one unaccustomed, is not an ideal place for sleep. Blankets are according to estimated needs often estimated by one far away. Ventilation is figured scientifically but is not sufficiently variable to suit the needs of each individual of a large group. If figured by a ventilation enthusiast, it may not suit the desires or needs of any member of the group.

During his first 3 weeks while endeavoring to accustom himself to all these changed conditions of life the recruit is subjected to the special measures used in the prevention of disease. He is vaccinated against smallpox, receives three injections of typhoid vaccine, and perhaps is also immunized against diphtheria. All these procedures make the man feel below par and must result in a somewhat lowered resistance. To all the excitement and uncertainty must be added the effect of a certain amount of depression and homesickness which cannot help being factors influencing the sense of well-being. It is during this state of his Navy career that the beginner in the Navy usually develops the acute nasal troubles, respiratory diseases, and cerebrospinal fever. The veteran of several years suffers little from these diseases and in fact the tendency to these infections begins to decrease after several months of service.

We find that some of the conditions mentioned are present when students attend school and are housed in dormitories. Floor space is limited, ventilation is by rule, and carriers are without doubt present. Food, clothing, and the hours and conditions of sleep and work are standard for the entire group. However, there is in evi-

dence the factor of personal supervision which allows a variation of routine to meet emergencies and changing conditions. This personal care operates to correct errors and prevents the serious consequences which would otherwise follow. In the case of the recruit the medical officer can supply much of this supervision but the one most suited by the nature of his duties to watch these details is the company commander. Too often, however, the company commander of 1917 disregarded such matters and, without any intention of being negligent, assumed the "hard-boiled" attitude. There is little doubt that the period of training should be lengthened, the "breaking in" process should be "slowed up", and the recruit should be given an opportunity to "grow into" his new life and that during this critical period he should be under more careful supervision.

It is not the purpose of this article to discuss diagnosis and treatment except insofar as there may be factors of importance in controlling the incidence of the disease and limiting the spread of epidemics. Diagnosis is of special importance as epidemics are usually kept in motion by failures of diagnosis especially the diagnosis of mild atypical cases.

What constitutes a case of acute cerebrospinal fever or an infection due to the meningococcus? If the books on internal medicine of two decades ago are studied it would appear that infection with the meningococcus is synonymous with cerebrospinal fever. The textbooks of 1916 recognized no meningococcus infection except that characterized by inflammation of the meninges, that type of the disease which in 1904 gave a mortality of 80 percent and a mortality of about 20 percent after the introduction of antimeningococcus serum. At that time there appears to have been no conception of the possibility of a mild infection involving other tissues and not involving the meninges. The experiences during the war demonstrated a pure septicemic form, without meningeal involvement. It seems worth while inquiring if this conception of milder forms of infection may not be extended. Is there not in existence a still milder more localized form of meningococcus infection involving the walls of the nasal and pharyngeal cavities and the accessory sinuses? Is it not possible that the so-called "carrier" who increases in numbers so rapidly under unfavorable climatic conditions represents not a mere carrier condition but rather an individual with a local pathological condition who is suffering from the mildest local form of meningococcus infection? This conception of the carrier would explain some of the points observed in studying the incidence of both carriers and cases, namely, the increase in the carrier index at the same time and under the same conditions under which the meningitis index increases, and the practical disappearance of the carrier during

the seasons when meningitis is absent. The situation during an epidemic would resolve itself into a widespread infection with the meningococcus, the majority of the cases representing a local infection with few symptoms to distinguish the complaint from acute coryza, acute bronchitis, and especially that hazy clinical entity so common in bad-weather seasons, acute catarrhal fever or common winter influenza. A small percent of cases would show symptoms of septicemia with the meningococcus in the blood and a still smaller number would represent true acute cerebrospinal fever with cerebrospinal symptoms and bacteriological findings. Richmond (NAVAL MEDICAL BULLETIN, February 1926) reports 21 cases of which 14 were of meningeal type while 7 never passed beyond the septicemic stage. He calls attention to the very great importance of blood cultures and white cell counts in distinguishing the septicemic form from confusing diseases. In studying the same epidemic Minter in United States NAVAL MEDICAL BULLETIN, July 1926, isolated 31 carriers who apparently had sufficient disturbance in the naso-pharyngeal region to afford temporary lodging for the meningococcus. During this period he reports an outbreak of mild influenza (catarrhal fever, acute) totaling 148 cases. The epidemic of influenza, the cases of meningitis and continuation of the carrier stages cover about the same period and all conditions cleared up at about the same time.

It would seem advisable to consider a rise in the monthly rate of acute bronchitis, acute catarrhal fever, and acute tonsillitis as the first signal to be on the watch for cases of meningococcus infection. Instances of sinus and throat pathology especially if showing the meningococcus in the secretions should stimulate the watch for the septicemic form. The appearance of the septicemic type with a high white count and a positive blood culture makes it only a question of a short time until a true case of acute cerebrospinal fever will appear. In this chain of events, each suspicious circumstance will cause the search for the next step to be strengthened. Measures may be instituted to correct hygienic defects and faulty methods of living. Early recognition of the septicemic and meningeal forms will allow early diagnosis and early treatment and early treatment means low mortality.

CONCLUSIONS

1. In the future it does not seem advisable to place too much faith in the detection and isolation of carriers as a controlling measure. A study of the carrier situation is an instructive scientific study and impresses the public, both lay and medical, but it consumes much time better used for other measures of control. It also introduces the difficult problem of the final disposition of the carrier.

2. Adequate floor space and proper ventilation should be given due consideration but it is unwise to become hysterical on the subject and to assign to these measures an importance which may not be warranted and which may even produce harmful and dangerous results.

3. Spectacular procedures have a value in combating certain phases of the situation but they should never monopolize the field to the exclusion of tested and valued hygienic measures.

4. The recruit should be under constant supervision during the first months of his Navy life as it is during this period that he is hypersusceptible to infection and to faulty hygienic conditions.

5. Ample time should be allowed for "hardening" and "seasoning" the recruit and during this critical period he should receive maximum protection against inclement weather, overwork, and fatigue.

6. The importance of typhoid prophylaxis and smallpox vaccination as a means of undermining resistance should be kept constantly in mind. These measures are necessary but their accompanying bad effects should be minimized by special care and a lightening of other duties.

7. The value of increased monthly rates for other diseases, namely acute bronchitis, acute tonsillitis, and water influenza as a warning of the appearance of conditions favoring the advent of meningococcus infection should be kept constantly in mind.

8. Cases with cerebral symptoms represent only a certain percent of cases with meningococcus infection. The pure septicemic type is common and further study will doubtless show that a type involving the nasal and pharyngeal cavities and the accessory sinuses is quite common.

9. The medical officer should always keep in mind the possibility of meningococcus infection and the conditions under which it may appear, in order that the cases appearing may have the benefit of early diagnosis and early treatment with a resulting lowered mortality.

HEALTH OF THE NAVY

The general admission rate, based on returns for diseases, injuries, and poisonings in the third quarter of 1934, was 580 per 1,000 per annum as compared with 476, the rate for the corresponding quarter of 1933, and 530, the median for the third quarter for the preceding 5 years.

The admission rate for disease for the quarter was 510 per 1,000 per annum and the 5-year median for the corresponding 3 months was 470.

The admission rate for injuries was 65, compared with 59, the median or expected rate for the preceding 5 years.

Poisonings increased from 0.45 per 1,000 per annum for the second quarter to 4.28 per 1,000 for July, August, and September, due to an outbreak of food poisoning on board the U. S. S. *Fairfax* in July during Fleet Naval Reserve training. One hundred cases were transferred to the Norfolk Naval Hospital for treatment, and 6 or more mild cases were kept aboard ship. No complications were noted, and all men were returned to duty in a few days. Bologna was the suspected cause of the outbreak.

Acute infections of the respiratory type were less prevalent than had been the experience for the previous quarter. A total of 1,161 cases of these diseases was reported by all shore stations in the United States during the quarter, of which 646 were notified by the Naval Training Station, Norfolk, Va., and 174 by the Naval Training Station, San Diego, Calif. Acute catarrhal fever constituted 85 percent of these admissions. Only 7 cases of influenza were reported from all shore stations in the United States during the quarter.

The United States Naval Academy, Annapolis, Md., reported 13 cases of mumps among midshipmen, 1 in July, 6 in August, and 6 in September. The United States Marine Barracks, Quantico, Va., and the Navy Yard, Boston, Mass., each reported 1 case of chickenpox in July.

During the quarter four recruits with cerebrospinal fever were transferred from the Naval Training Station, Norfolk, Va., to the Norfolk Naval Hospital. Two of the three cases admitted in August terminated fatally, one on August 13 and the other on August 14. One other case of this disease developed in a recruit while on leave. He was admitted and discharged from the Naval Training Station, Norfolk, Va., after 39 sick days. The Naval Training Station, San Diego, Calif., reported one case in August.

A recruit with 7 days' service was transferred with typhoid fever from the Marine Barracks, Parris Island, S. C., to the United States Naval Hospital, Parris Island. This case was considered to have existed prior to enlistment.

There were 221 admissions for respiratory diseases reported by foreign shore stations, of which 81 were notified by the Fleet Air Base, Canal Zone, and 42 by the Fourth Marines, Shanghai, China. The Fourth Marines, Shanghai, China, also reported a moderately severe case of typhoid fever in August.

Reports from forces afloat indicate that the morbidity rate for all causes assumed a normal expectancy during the third quarter of 1934. The admission rate was 507 per 1,000 per annum, and the 5-year median for the corresponding 3 months was 498. Sixteen ships reported 540 cases of acute respiratory infections during the quarter.

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Three cases of cerebrospinal fever occurred among forces afloat during the quarter. A man attached to the U. S. S. *Humphreys* became ill in July while on leave and died in a civilian hospital; a member of the crew of the U. S. S. *Relief* died in the Norfolk Naval Hospital on October 11; and a member of the crew of the U. S. S. *Idaho*, who was transferred to the Norfolk Naval Hospital, recovered.

A case of malignant tertian malaria was admitted to the sick list on July 22 on board the U. S. S. *Hannibal* and returned "to duty under treatment" on August 8. While still undergoing ambulatory treatment for malaria on August 17 he complained of nausea and fainted. He died the following day without regaining consciousness.

Two cases of scarlet fever were reported in September, 1 from the U. S. S. *Barker* and 1 from the U. S. S. *Lexington*.

Two cases of typhoid fever were reported, 1 in August from the U. S. S. *Tulsa* and 1 in September from the U. S. S. *Luzon*, both of the Asiatic Fleet. Two cases of paratyphoid fever were notified by the U. S. S. *Tulsa*, 1 in August and 1 in September.

TABLE 1.—*Summary of morbidity in the United States Navy for the quarter ended Sept. 30, 1934*

Average strength	Forces afloat, 73,756		Forces ashore, 35,512		Entire Navy, 109,268	
	Admissions	Rate per 1,000	Admissions	Rate per 1,000	Admissions	Rate per 1,000
All causes	9,356	507.40	6,477	729.56	15,833	579.60
Diseases only	8,275	448.78	5,658	637.31	13,933	510.05
Injuries	976	52.93	807	90.90	1,783	65.27
Poisonings	105	5.69	12	1.35	117	4.28
Communicable diseases transmissible by oral and nasal discharges (class VIII):						
A	295	16.00	278	31.31	573	20.98
B	1,676	90.89	1,610	181.35	3,286	120.29
Venereal diseases	2,305	125.01	568	63.98	2,873	105.17

TABLE 2.—*Deaths reported, entire Navy, during the quarter ended Sept. 30, 1934*

Cause—Disease		Navy			Marine Corps		Nurse Corps	Total
Primary	Secondary or contributory	Officers	Midshipmen	Men	Officers	Men		
Average strength		9,581	1,676	80,551	1,183	15,940	337	109,268
Aneurysm, cerebral artery.	Hemorrhage, cerebral			1				1
Arteriosclerosis, general	Thrombosis, coronary			1				1
Carcinoma, stomach	None			2				2
Cellulitis, face	Septicemia			1				1
Cerebrospinal fever	None			3				3
Colitis, chronic	Peritonitis, general, acute			1				1
Dilatation, cardiac, acute.	None			1				1
Do	Status lymphaticus			1				1
Encephalitis, acute	None			1				1
Endocarditis, acute	Pneumonia, broncho-			1				1
Glioma, brain	None			1				1

TABLE 2.—*Deaths reported, entire Navy, during the quarter ended Sept. 30, 1934*—Continued

Cause—Disease		Navy			Marine Corps		Nurse Corps	Total
Primary	Secondary or contributory	Officers	Midshipmen	Men	Officers	Men		
Gonococcus infection, endocardium.	None					1		1
Hyperthyroidism	Status lymphaticus			1				1
Leukemia	None			1				1
Malaria, malignant, tertian.	None			1				1
Myocarditis, chronic	Thrombosis, coronary artery.			1				1
Nephritis, chronic	Hemorrhage, cerebral			1				1
Otitis, media, acute	Meningitis, cerebrospinal, acute.			1				1
Pneumonia, broncho-	None			1				1
Do	Cellulitis, neck and face					1		1
Pneumonia, lobar	None			1				1
Do	Edema, lung			1				1
Rupture, nontraumatic, diaphragm.	Shock					1		1
Septicemia	Dementia praecox					1		1
Sinusitis, ethmoidal, acute.	Abscess, brain			1				1
Syphilis	Dementia paralytica and malaria.			1				1
Tonsillitis, chronic (postoperative).	Pneumonia, broncho			1				1
Thrombosis, coronary artery.	None	3		1				4
Tuberculosis, pulmonary, chronic.	None			1				1
Tuberculosis, pulmonary, acute pneumonic.	Pleurisy, fibrinous, acute			1				1
Tuberculosis, pulmonary, acute general miliary.	Septicemia			1				1
Tumor, benign, brain	None			1				1
Ulcer, duodenum	Abscess, subphrenic			1				1
Do	Peritonitis, general, acute			1				1
Ulcer, stomach	Hemorrhage, stomach					1		1
Valvular heart disease, aortic insufficiency.	Endocarditis, ulcerative, acute (malignant).			1				1
Total for disease		3	0	34	1	4	0	42
INJURIES AND POISONINGS								
Asphyxiation, illuminating gas.	None					1		1
Burn, back and abdomen.	None			1				1
Contusion, abdomen	Peritonitis, general, acute			1				1
Crush, chest	None			1				1
Drowning	None	3		4		1		8
Embolism, air, due to submarine escape appliances	None			1				1
Fracture, compound, skull	None			5		1		6
Fracture, simple, skull	None			2		1		3
Fracture, simple, skull	Intracranial hemorrhage			1				1
Heat exhaustion	None			3				3
Intracranial injury	None					1		1
Injuries, multiple, extreme	None	1		5				6
Wound, gunshot, head	None	1		2				3
Poisoning, acute, cyanide				1				1
Total for injuries and poisonings		5		27		5		37
Grand total		8		61	1	9		79
Annual death rate per 1,000:								
All causes		3.34		3.03	3.38	2.26		2.89
Disease only		1.25		1.69	3.38	1.00		1.54
Drowning		1.25		.20		.25		.29
Injuries		.83		1.09		1.00		1.03
Poisonings				.05				.04

ADMISSIONS FOR INJURIES AND POISONINGS, THIRD QUARTER, 1934

The following table, indicating the frequency of occurrence of accidental injuries and poisonings in the Navy during the third quarter, 1934, is based upon all form F cards covering admission in those months which have reached the Bureau:

	Admissions, July, August, and September 1934	Admission rate per 100,000, per annum	Admission rate per 100,000, year 1933
INJURIES			
Connected with work or drill	753	2,756	2,237
Occurring within command but not associated with work	511	1,871	1,692
Incurred on leave or liberty or while absent without leave	519	1,900	1,757
All injuries	1,783	6,527	5,686
POISONINGS			
Industrial poisoning	3	11	26
Occurring within command but not connected with work	109	399	191
Associated with leave, liberty, or absence without leave	5	18	19
Poisonings, all forms	117	428	236
Total injuries and poisonings	1,900	6,955	5,922

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.	
	July, August, and September 1934	Year 1933	July, August, and September 1934	Year 1933	July, August, and September 1934	Year 1933
Percent of all injuries	42.2	39.3	28.7	29.8	29.1	30.9
Percent of all poisonings	2.6	10.9	93.1	80.9	4.3	8.2
Percent of total admissions, injury, and poisoning titles	39.8	38.2	32.6	31.8	27.6	30.0

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.

STATISTICS RELATIVE TO MENTAL AND PHYSICAL QUALIFICATIONS OF RECRUITS

The following statistics were taken from monthly sanitary reports submitted by naval training stations:

July, August, and September 1934	United States naval training station	
	Norfolk, Va.	San Diego, Calif.
Recruits received during the period	1,219	1,480
Recruits appearing before board of medical survey	8	0
Recruits recommended for discharge from the service	8	0
Recruits discharged by reason of medical survey	18	0
Recruits held over pending further observation	0	0
Recruits transferred to the hospital for treatment, operation, or further observation for conditions existing prior to enlistment	22	40

The following table was prepared from reports of medical surveys in which disabilities or disease causing the surveys were noted as existing prior to enlistment. With certain diseases, survey followed enlistment so rapidly that it would seem that many might have been eliminated in the recruiting office.

Cause of survey	Number of surveys	Cause of survey	Number of surveys
Absence, acquired, teeth	11	Enuresis	7
Adhesions, intestinal	1	Epilepsy	9
Ankylosis, right elbow	1	Flat foot	4
Anomaly of form (pronated feet)	1	Gastroptosis	1
Arterial hypertension	6	Gonococcus infection, urethra	7
Asthma	1	Gonococcus infection, prostate	1
Astigmatism	3	Hammer-toe both feet	1
Bursitis, chronic, right knee	2	Insufficiency, ocular muscle	3
Caries, teeth	1	Mastoiditis, chronic, left	1
Color blindness	1	Otitis, media, chronic	5
Constitutional psychopathic inferiority, without psychosis	1	Paralysis, ocular muscle	1
Constitutional psychopathic state, inadequate personality	1	Perforated nasal septum	1
Constitutional psychopathic state, emotional instability	1	Pes cavus	1
Deafness, unilateral	1	Pneumonitis, chronic, nontuberculous	1
Defective physical development	1	Psychoneurosis, hysteria	2
Deformity, acquired, cervical vertebra	1	Psychoneurosis, neurasthenia	2
Deformity, acquired, right elbow	1	Pyorrhea alveolaris	1
Dementia praecox	2	Rheumatic fever	1
Deviation, nasal septum	1	Sinusitis, frontal	1
Endocrinopathy (parathyroidism)	1	Syphilis	7
		Tuberculosis, pulmonary, chronic, active	1
		Valvular heart disease, mitral stenosis	3

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NAVY DEPARTMENT,
Washington, March 20, 1907.

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

TRUMAN H. NEWBERRY,
Acting Secretary.

Owing to the exhaustion of certain numbers of the BULLETIN and the frequent demands from libraries, etc., for copies to complete their files, the return of any of the following issues will be greatly appreciated.

Volume IX, no. 1, January 1915
Volume X, no. 2, April 1916
Volumr XI, no. 3, July 1917
Volume XII, no. 1, January 1918
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PREFACE

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

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

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

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

The Bureau does not 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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The greatest accuracy and fullness should be employed in all citations, as it has sometimes been necessary to decline articles otherwise desirable because it was impossible for the editor to understand or verify references, quotations, etc. The frequency of gross errors in orthography in many contributions is conclusive evidence that authors often fail to read over their manuscripts after they have been typewritten.

Contributions must be received at least 3 months prior to the date of the issue for which they are intended.

The editor is not responsible for the safe return of manuscripts and pictures. All materials supplied for illustrations, if not original, should be accompanied by reference to the source and a statement as to whether or not reproduction has been authorized.

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U. S. NAVAL MEDICAL BULLETIN

VOL. XXXIII JULY 1935 No. 3

SPECIAL ARTICLES

AMEBIASIS AND ITS SURGICAL COMPLICATIONS [1]

By J. J. A. McMullin, Commander, Medical Corps, United States Navy

Amebic dysentery, formerly considered a tropical disease, is now known to be endemic in the United States. Like the poor, it is always, but less frequently, with us. Interest in the subject of amebiasis was stimulated by the outbreak in Chicago in the summer of 1933.

Amebae were first noted by Lambl, the eminent Bohemian pathologist, in 1859 in stools of a child with diarrhea. In 1860 a Swede, Kjellberg, discovered a parasite which invaded the tissues of the bowel, and in 1870 and 1871 similar discoveries of this parasite were made by two Anglo-Indian medical officers, Lewis and Cunningham. In 1875 Losch in Russia first directed attention to amebae as the possible cause of dysentery. In 1878 Grassi found encysted amebae, but since they also were found in stools of normal persons, he denied their pathogenicity. The suggestion of Losch was not considered seriously because there were typical cases of dysentery without apparent amebic infection. Koch and Kartulis noted amebae in intestinal ulcers and Kartulis found them also in liver abscess. Councilman and Lafleur in 1891 noted a pathogenic species, found in the submucosa of intestinal ulcerations, and also reported a harmless species. The problem was greatly simplified when the etiology of bacillary dysentery was fully established in 1900, but the fact that amoebae were frequently found in stools of healthy persons was still a complicating factor. This complication was obviated when Fritz Schaudinn suggested that amebae belonged to two different species, naming the pathogenic one endamoeba histolytica on account of its ability to dissolve tissues, and the harmless species endameba coli. Endamoeba histolytica is now known to be the only pathogenic ameba. Among the nonpathogenic amebae there are two others besides the endameba coli. Walker and Sellards, who modified extensively the details of Schaudinn's work on the morphology of

[1] Read before the Brooklyn Surgical Society, Dec. 6, 1934.

endameba histolytica, fed encysted endameba histolytica to 20 volunteers in the Philippine Billibid prison. Four of the 20 contracted typical dystentery; 14 of the 20, who escaped clinical signs of dysentery, showed parasites in their feces. In other words, 20 percent of those parasitized contracted amebic dysentery, 70 percent became carriers of the disease, and only 10 percent of those exposed showed negative feces.

Amebic dysentery, endemic throughout the United States, thrives best in tropical countries: In Egypt, India, China, the Philippines, and Russia. The incidence of the disease is proportional to the effectiveness of sanitation. The Caucasian race is apparently more susceptible to the disease than the natives in tropical countries. The disease is less frequent in infancy and childhood, and in the female sex. Some observers maintain, however, that with equal chances of infection the incidence would be equal in the two sexes. The excessive use of alcohol producing as it does congestion and an increased functional burden on the hepatic parenchyma is a predisposing cause of abscess of the liver.

The incubation period, according to Walker and Sellards experiments, averaged 64 days. In the recent Chicago epidemic the shortest incubation period was 5 days and the longest 95 days.

Mode of transmission.—With the recent appearance in the United States of an increasing number of reported cases, it is well that we should have clearly in mind the method by which this malady is disseminated. Amebic dysentery is truly an endemic dysentery. Epidemics of diarrheas are only exceptionally amebic, for the reason that motile trophozoites, predominating in active cases, are not infectious. Any appreciable number of cases appearing at one time usually indicates that there has been an effective means of fecal contamination of water, milk, or food supply from some small focus or carrier. The healthy carrier, free of symptoms, or the convalescent from acute dysentery, who harbors encysted amebae, are the conveyors of the disease. Amebic infections will be with us until some simple and effective method is devised for the detection and treatment of these carriers of the disease. The encysted ameba demands moisture for its continued ability to reproduce the disease, desiccation rapidly rendering it harmless. It may live for several weeks in water, and longer in fecal discharges. Transmission by food and flies, while worthy of serious consideration, is of less importance than transmission by water. Plumbing installation where there is any cross connection between the sewage and fresh-water pipes should be replaced. Contamination of the fresh-water system by siphonage through a cross connection with sewage water was found to be responsible for the epidemic nature of the Chicago outbreak.

The incidence in some tropical countries bears a direct relationship to the amount of rainfall, and in Egypt the number of cases increases when the river Nile overflows. It may be of interest to mention a frequent post-mortem finding in amebic carriers as reported by several investigators. While the carrier may have had no symptoms of dysentery, at the autopsy table a considerable number of cases reveal ulcerative lesions or scars of ulcerative lesions in the colon. It is reasonable to assume that such lesions are the portals of entrance in the many cases of amebic abscess of the liver occurring in persons who have been carriers of the disease without ever having suffered from symptoms suggestive of amebic dysentery.

Pathological consideration.—Primary amebiasis belongs to the estate of the internist, but we shall consider briefly just how the endameba histolytica infects its human host. The motile amebae are destroyed and digested by the gastric juice. The encysted amebae enter through the mouth in water or on food, and pass through the stomach intact. Their envelops are dissolved in the small intestine, and there they discharge young amebae into the lumen of the terminal ilium and colon. Here they elaborate a cytolytic ferment, which destroys the epithelial layer of the lower intestinal tract, and penetrate to varying depths, occasionally producing a complete perforation. Usually the ulcer extends down to the muscularis, is variable in size with undermined edges, at which location lie the amebae in the products of their activity, but close to the surrounding viable tissue cells. Characteristic reactions on the part of the tissues occur. Capillary stasis appears with later complete thrombosis. An exudation of fluid follows from the thrombosed capillaries, producing edema followed by coagulation necrosis. In the amebic ulcer without secondary infection this gives rise to the gelatinous coagulum on its base with cells in all stages of degeneration. The amebae reach the liver by way of the portal vein. There are cases, also, of extension by way of perforation through the intestinal wall to other viscera. In the liver the initial lesion is an amebic hepatitis, in which the organ sometimes is able successfully to combat the invaders, but at other times succumbs to a coagulation necrosis similar to that which takes place in the intestinal ulceration. In the liver, however, it is usually a single, sometimes multiple, centrifugally enlarging cavity containing liver cells in all stages of degeneration, mixed with blood and bile. In some instances there is a mixed infection with the formation of true pus. The process as a rule is rapid, and no pyogenic membrane is formed, the cavity wall being a ragged edge of crumbling ameba-digested liver tissue. Progressive destruction is the rule until rupture or surgical drainage occurs, but some cases, with or without treatment, are arrested in their progress and undergo a process of encapsulation which ultimately may become calcified.

This brings to mind a patient from whom I removed a calcified amebic abscess of the liver in 1925, 7 years after he had been invalided home from the Philippines with amebic dysentery. The wall of the abscess was completely calcified, and the cyst cavity contained about 500 grams of a chalky white material with the consistence of putty. The termination of a liver abscess into a calcareous cyst is rare, but it does happen occasionally. This patient suffered from symptoms suggesting chronic cholecystitis, except that the gaseous indigestion was more distressing.

Surgical complications.—Surgeons rarely see patients suffering from primary uncomplicated amebiasis. Fulminating infection with perforation may occur. In intractable cases appendicostomy or coecostomy is sometimes done, but high colonic irrigation usually suffices for direct medication of the colon. Amoebicidal appendicitis is occasionally seen. Our chief concern as surgeons is with the secondary manifestations of the disease, the chief one being abscess of the liver. No definite figure can be quoted to indicate the percentage of primary amebiasis followed by liver abscess. The incidence varies from 5 to 20 percent. When the unknown number of cases of bowel infection without symptoms are kept in mind it may be seen how difficult it is to determine this figure. Certain it is that prompt diagnosis and effective treatment will either eliminate or markedly reduce the frequency of abscess of the liver.

Liver abscess usually occurs during the first 2 months after amoebic ulceration in the colon. The ulcer may have been of pin point size, and produced no symptoms, but it is believed that ulceration is necessary as the door through which the endameba histolytica enters. However, abscess of the liver may not eventuate until months or years have elapsed since the history of a frank attack of dysentery or diarrheaic symptoms.

Several observers of the disease, particularly Rogers, describe a presuppurative stage or an amoebic hepatitis, which lasts several weeks or months during which the patient suffers first from a dull dragging sensation over the liver area, and perhaps vague digestive upsets. This may be accentuated by the development of pain of a stabbing character, with irregular fever, profuse sweating and in some instances rigors or chilly sensations. Physical examination may reveal an enlarged and tender liver, and some immobility of the diaphragm. This period of hepatitis calls for active emetine treatment; otherwise the hepatitis may go on to abscess formation.

The dividing line between hepatitis and abscess cannot be drawn sharply. The symptom of discomfort progresses to a disconcerting pain, to a disabling pain, the patient finding greatest ease lying on the right side with the leg drawn up. This pain is constant, may radiate to the shoulder, neck, or arm, and is aggravated by jarring of the body, occasioned, for instance, by coughing or sneezing. The pain in amoe-

bic abscess of the liver is most variable in its intensity. Likewise are the other symptoms of this condition. They appear to depend upon the degree of virulence of the parasite, the amount of mixed infection in the abscess cavity upon the resistance of the patient, and most important of all upon the thoroughness of treatment.

The temperature ranges from a completely normal curve, in the chronic and slowly developing cases, to one which may be maintained with daily septic fluctuations to 102° or 103°, and again there may be periods of remission from fever, during which the patient may feel quite well. When the febrile reaction is septic, chills or chilly sensations and drenching and weakening sweats are frequently observed at night.

The patient may have a dry, hacking cough, depending upon the location of the abscess and the amount of diaphragmatic and pleural irritation. There may be episodes of vomiting. The appetite is poor, but copious quantities of cold liquids are relished. With chronic abscess of the liver the patient's facial expression is apathetic and at times extreme degrees of emaciation are seen. The skin and sclerae are slightly jaundiced, and the tongue is coated and the breath foul. In both acute and chronic abscess of the liver we find the respirations are shallow and thoracic, due to splinting of the diaphragm to keep the liver at rest. Slight pressure or tapping at various points over the liver aggravate the pain. An abscess in the usual site, the right lobe of the liver, sometimes produces a visible prominence of the right lower chest and will at times produce a definite increase in the right lower costal angle. If, as rarely happens, the abscess is in the left lobe, there may be a palpable mass in the epigastrium. Many times percussion will demonstrate a definite increase in the height of the dome of the liver. Less frequently the liver edge will be lower than normal. Upon auscultation crepitant rales and diminished breath sounds may be heard over the base of the right lung.

The leucocytic response is variable. It is said to depend upon the degree of secondary infection in the abscess cavity. Counts, however, average from 14,000 to 24,000, with 70 to 80 percent of polymorphonuclear cells. In some cases a normal count may be encountered. In chronic cases a hypochromic anemia is seen.

X-ray or fluoroscopic examination renders invaluable aid. It will be observed frequently that the diaphragm does not follow its normal excursion, and that the dome of the liver is elevated. The abscess cavity may be outlined, particularly in those which are of long standing with thickened walls.

Rarely a pulmonary abscess may occur independent of a liver abscess, but more often they are caused by rupture of an abscess of the right lobe of the liver into the right lung. The symptoms in either case are those characteristic of pulmonary abscess. There is a productive cough, at first muco-purulent which finally, as the products

of the activity of the ameba make their appearance, becomes the typical reddish-brown liver abscess material. This drainage phenomenon may be the means of spontaneous cure of the liver abscess, or it may lapse into a state of chronicity. The physical findings are those of an increased density of the base of the lung with diminished breath sounds.

The X-ray is likewise helpful in instances of pulmonary abscess and may point the way to a previously unsuspected involvement of the liver.

Cerebral abscess.—Cerebral abscess is an unusual complication of the disease, but has been observed after drainage of a liver abscess. The symptoms are those of any brain abscess, depending upon its location. There may be severe headache, evidence of increased intracranial pressure, hallucinations, epileptiform episodes, and projectile vomiting. The cerebrospinal fluid is normal in appearance, though it may be under increased pressure. The temperature more than likely will be close to normal, unless there be also an hepatic or pulmonary collection of pus.

The downward course to coma and death in amebic abscess of the brain is almost invariably rapid. Earlier diagnosis and operation might make the outlook of this complication less gloomy.

Infection in other organs.—It is probable that the endameba histolytica, reported as occurring in other organs, reaches them by extension from the bowel, liver, or lung. The liver abscess, if not evacuated surgically, ruptures in order of frequency, into the lung, peritoneum, pleura, stomach, duodenum, colon, illio-lumbar area, the vena cava, right kidney, and the pericardium. One of the patients in this hospital developed an extensive necrosis of the skin and subcutaneous tissue of the chest wall about an incision which drained an amebic liver abscess. This area continued to increase in size and exposed the ribs and intercostal muscles, and it may be particularly noted that active necrosis continued under treatment with carbarsone. When carbarsone was discontinued, and emetine administered, the local improvement was immediate and striking although the patient finally succumbed to the infection. Here was an instance of visible evidence of the specificity of emetine in skin amebiasis. In my opinion emetine was started too late in this case, and carbarsone was contra-indicated because of the liver damage.

Mayer describes the phagedenic nature of skin amebiasis resulting from draining amebic abscesses or intestinal and anal fistulae, and refers to a case of amebic ulceration of the glans penis, in a Chinaman, following coitus per anum. And so under such circumstances amebiasis may be a disease of venereal origin.

Diagnosis.—The diagnosis of amebic liver abscess is beset with difficulty. On one occasion I made a preoperative diagnosis of per-

forated duodenal ulcer, and found nothing abnormal within the abdomen except enlargement of the right hepatic lobe, which proved to be the site of an amebic abscess, and on another occasion I made a preoperative diagnosis of acute gangrenous cholecystitis and found a large abscess of the right lobe of the liver just about to rupture. Both of these abscesses were evacuated and drained, and were given one grain of emetine subcutaneously on 10 successive days, and went on to recovery. When a patient has symptoms indicating an acute abdominal emergency, and when abdominal exploration of such a case is negative, except for enlargement of the liver, the abdomen should not be closed until the liver has been explored with an aspirating needle. The diagnosis of amebic abscess of the liver has been confused also with subdiaphragmatic abscess, appendicitis, empyema, perinephritic abscess, and every other conceivable acute inflammatory condition in or adjacent to the abdomen. The principal reason for these mistakes is the simple fact that amebic abscess of the liver has not been given sufficient diagnositic consideration. Of prime importance in the diagnosis of amebiasis and its complications is the expert laboratory specialist who can recognize the endameba histolytica in its encysted and motile forms and differentiate it from the three other forms of nonpathogenic amebae. The clinical diagnosis is facilitated by a history of antecedent dysentery. When such a history is obtained, and fever, sepsis, and pain in the right upper abdomen are associated with demonstrable enlargement of the liver, a tentative diagnosis of amebic abscess of the liver should be made.

The symptoms of amebiasis vary from a severe prostrating dysentery, quickly fatal, to those of obstinate constipation or constipation alternating with diarrhea. This should be borne in mind always.

In collaboration with Commander E. A. Vickery, Medical Corps, United States Navy, I reported two cases of amebiasis with constipation from the Naval Hospital, Olangapo, P. I., in the October 1917 issue of the NAVAL MEDICAL BULLETIN. We commented as follows at that time:

> In connection with amebic infection in the tropics it is interesting to note a symptom-complex which has received very little attention in the literature. The symptom-complex so closely simulates that of chronic intestinal obstruction that a tentative diagnosis of intestinal stasis with auto-intoxication was made in the two cases herewith presented.
>
> The symptoms were practically identical in the two cases and were as follows: Marked and obstinate constipation, lasting for about a year, movement of the bowels being obtained only with a cathartic for variable periods lasting from 1 to 2 weeks; vague pains referred to the abdomen, with a little tenderness in the neighborhood of the caecum; loss of weight and appetite; lassitude; mild general malaise, with markedly sallow complexion. Both cases showed indicanuria. At no time was there any diarrhea.
>
> One case presented no history of acute attack, and symptoms were somewhat relieved by the administration of Russian paraffin. The other case had

a history of amebiasis 1 year prior to symptoms and a gastro-intestinal X-ray study showed no evidence of intestinal stasis.

It is interesting to note that amebic infection can occur in its chronic stage with absolutely no diarrhea, a point not generally considered in the diagnosis of the infection. The question of infectivity from such a carrier is an important one, as in one of the cases mentioned the amebae were found encysted, with but few motile forms. It seems probable that a symptom-complex of this kind in a patient returning from the tropics would easily escape diagnosis and might well be treated surgically as a case of mechanical intestinal stasis, particularly as encysted nonmotile amebae are not easily detected during a routine examination of the feces. We consider the administration of a saline purgative as a necessary preliminary to microscopic examination in these chronic cases, as amebae were only found after such a procedure.

The patient should take a saline purgative in the morning, followed by his usual breakfast, and within the next 2 or 3 hours a fresh warm liquid stool may be available for examination. The specimen should be taken immediately, while warm, to a slide on the warmed stage of a microscope, diluted with a drop of warm saline and quickly examined. A plea is also made for more frequent proctoscopic examinations in search for ulcers of the rectum. At times stool examinations will be negative, whereas the debris from a small ulcer will be positive. The encysted form of the parasite is found in stools not immediately examined or kept warm, but the element of error is multiplied many-fold unless the examiner has had considerable training and experience in the recognition of amebae. Concentration methods in the examination for cysts are practical, and the culture of the endameba histolytica from suspected feces further increases the chances of finding the parasite.

Amebic granulomas are not common by any means, and yet with their effective mimicry of carcinomatous lesions of the large bowel they should be borne in mind. Cases reported from time to time should make us pause before performing an extensive abdominal operation for a colonic mass without at least giving thought to this possibility. The presence of lymphogranuloma can be corroborated by the Frei specific antigen cutaneous test. Amoebic infection of the colon may cause benign poliposis, which not infrequently eventuates in malignancy. Likewise, in every case of chronic ulcerative colitis the stools should be examined at least six times for the presence of endameba histolytica.

It has been variously estimated that from 1 to 4 percent of the entire population of the United States is infested with the endamoeba histolytica. If we accept the lowest of these estimates it is sufficient reason for a careful search for this parasite in our routine hospital examinations, excepting perhaps patients acutely or severely ill from some other established cause.

TREATMENT

Treatment will be considered under the following captions:

I. The common type of amebiasis.

II. The severe type of amebiasis.

III. Hepatitis and abscess of the liver.

I. Common Type.—Chronic intestinal amebiasis:

(*a*) Carrier state,

(*b*) Recurrent, or

(*c*) Mildly persistent symptoms.

The contraindications to carbarsone or other arsenical compounds are: (1) Renal irritation revealed by urinalysis, (2) gross damage of liver, (3) optic nerve lesions, (4) history of arsenical dermatitis.

1. Give 0.25 gm carbarsone in gelatin capsules twice daily for 10 days.
2. Wait 10 days.
3. Repeat course at alternate 10-day intervals if ameba reappear in stools.
4. In resistant cases alternate course of carbarsone with courses of emetine or vioform. Emetine by hypodermic 1 grain daily until from 6 to 10 grains are administered, or vioform 0.25 gm in gelatin capsules twice a day for 10 days.
5. If gastrointestinal irritation develops, coat the vioform capsules with freshly melted phenyl salicylate.
6. Diet should be rich in vitamin B in all cases of amebiasis.

II. Severe Types.—

(*a*) Extensive ulceration of colon.

(*b*) Severe dysentery.

(*c*) Cases of acute, malignant onset and course.

General care: (1) Patient in bed; (2) soft diet, rich in vitamin B; (3) adequate rest by means of sodium amytal.

A. *Method.*—

1. Give cleansing sodium bicarbonate enema.
2. One hour later instill into rectum 200 cc of warm 2 percent sodium bicarbonate solution containing 1 percent carbarsone.
3. Give orally, well in advance of above, 0.2 gm sodium amytal to facilitate retention of the enema overnight.
4. If enema is expelled before morning, repeat it.
5. Five enemas on five successive nights must be retained overnight.

B. *Method.*—

1. Give hypodermically 65 mg of emetine hydrochloride subcutaneously once a day for 6 days.
2. This may be followed by 30 mg daily for 6 days.
3. The total dosage of emetine must never exceed 10 mg per kilogram of body weight.

Contraindications: (1) Never use emetine in presence of myocarditis; (2) when emetine is administered, an increase in the pulse rate and a fall in the blood pressure are danger signals.

Generally speaking the best results of treatment are obtained by alternating emetine and carbarsone when the patient's condition permits the use of both these remedies.

If there be severe colic, or diarrhea which cannot be checked with emetine, give equal parts of dicalcium phosphate or tribasic phosphate and bismuth subcarbonate in 4 g doses 3 to 6 times daily.

However, the best means of checking amebic diarrhea is by the use of emetine. Amebic diarrhea rarely persists after 2 or 3 grains of emetine have been administered.

To relieve colonic spasm.—Give atropin sulphate 1/150 gr hypodermically.

In cases with associated bacterial infection.—(1) Give bismuth subcarbonate, (2) also may give short courses of irrigation of 1 to 2,500 acriflavine hydrochloride each night for 6 nights.

III. For Hepatitis and Liver Abscess.—

1. Give emetine as detailed previously.
2. Do not give carbarsone or any arsenical compound.
3. Local treatment of amoebic abscess:
   (*a*) Aspiration.
   (*b*) Repeat aspiration if necessary.
   (*c*) Irrigate with 4 ounces 1 to 2,000 solution of emetine hydrochloride.
   (*d*) If fever disappears and there is no evidence of secondary infection, operation may be avoided.
4. Abscess of other organs, ulceration of skin, etc. (Emetine is the most useful remedy, but if contraindicated carbarsone should be ordered.)

The late Commander W. D. Owens, Medical Corps, United States Navy, successfully treated amebic dysentery with syrup of ipecac through a duodenal tube. To quote from his report in the July 1922 issue of the Naval Medical Bulletin: "An Einhorn tube is passed on a fasting stomach in the early morning, when the tube is well into the duodenum, 1 ounce of sirup of ipecac, containing 40 grains of the drug to the ounce, is passed through the tube by gravity." This treatment is given every morning for 10 days and might be useful in certain cases when the stomach will not tolerate the usual remedies administered by mouth. Again the duodenal tube might be found useful when the stomach is intolerant to the administration of other amoebicidal drugs and when intestinal irritability and diarrhoea preclude rectal medication.

Chatterji, quoted by Rear Admiral Stitt, states that there are two schools of thought on the question of operation versus aspiration:

One claims the following advantages for aspiration: (1) Whereas the shock to the already exhausted patient is great from an open operation, it is practically nil after aspiration; (2) while secondary infection is practically unavoidable in an open operation, with ordinary precautions it can be completely avoided in aspiration; (3) after open operation the convalescence is prolonged and the long stay in bed weakens the patient, whereas after aspiration the patient can get out of bed in a day or two; (4) prolonged suppuration and dressing after an open operation exhausts the patient; and lastly (5) statistics show that for the above reasons mortality is higher in cases of open operations than after aspiration.

The other school, advocating open operation, i. e., treating the abscess by incision and drainage, claims the following advantages: (1) That it allows free drainage; (2) that a more direct view can be obtained and the presence or absence of other contiguous abscesses can be detected by exploration.

They deprecate aspiration on the grounds: (1) That it is an unsurgical procedure; (2) that though the aspirating needle may evacuate one abscess, it may not evacuate a second one if present; (3) that it does not ensure complete evacuation; and lastly (4) that it may puncture venous trunks.

Brown in the Collected Papers of the Mayo Clinic, 1933, states that, "Practically all the deaths in the Chicago epidemic occurred after surgical intervention." This in my opinion is an indictment against routine open operation. The mortality from aspiration is about 2 percent. The mortality from open operation is about 30 percent.

When aspiration is used the area is anesthetized with 0.5 percent novocaine and a 3½-inch needle with a one-eighth-inch bore is introduced in the ninth or tenth interspace in the anterior axillary line. From this point the right lobe of the liver is explored and when pus is located a three-eighth-inch bore trocar and canula is introduced along side the needle into the abscess cavity. A catheter, fenestrated at the distal end is introduced through the canula, and the catheter is fixed to the skin with one silkworm gut suture. The abscess cavity should be irrigated once daily with 4 ounces of a 1:2,000 solution of emetine hydrochloride.

The accepted surgical treatment of liver abscess is incision and drainage. There are several methods of approach, depending upon the location of the abscess. The Ochsner operation is ideal when the abscess is located in the right hepatic lobe. The twelfth rib is removed subperiosteally, the lower reflection of the pleura, if encountered, is pushed upward and the dome of the liver explored extraperitoneally. If the pleura is incised it should be sewed to the intercostal muscles with a continuous no. 1 chronic catgut suture, and the operation completed. Another method is to resect the tenth rib posteriorly below the angle of the scapula. This route is applicable in certain cases when a large abscess is localized high on the dome of the liver and the diaphragm and pleura are pushed upward. A right rectus abdominal incision is used when the diagnosis is in doubt, and when more than one abscess is present. The liver may be indurated over

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the abscess if it be deeply dituated; if pointing near the surfaces fluctuation may be elicited. In either case the pus is first located with an aspirating needle, and the bulk of the contents evacuated with a trocar and canula. The abscess cavity is walled off with wet abdominal sponges and is then opened with a closed hemostatic forceps. The wall of the abscess is not disturbed, a drainage tube is introduced into the cavity, and the abdominal incision closed around the tube. In some instances it is advisable to bring the drainage tube out through a stab wound in the side, and close the larger operative incision without drainage.

CONCLUSION

In conclusion, I should like to repeat that this subject of amebiasis is one too long neglected by many, too long thought of as a tropical disease instead of being, as it is, always among us, playing a rather quiet but important role in the drama of living pathology. My final statement is a word of endorsement in favor of routine aspiration of amebic liver abscess with the realization that open operation is sometimes imperative.

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AMEBIASIS [1]

By H. M. Weber, Lieutenant, Medical Corps, United States Navy

I should like to present some interesting figures bearing upon the general prevalence of amebiasis in this country, a chart illustrating the influence of sanitation and education on the incidence of amebic deaths in Panama and the localization of amebic abscess in the liver, quote a few words of wisdom in connection with the problem of so-called amebic carriers, mention some facts in connection with reported cases of amebic granoloma and briefly speak of the retroperitoneal approach to collections of pus about the superior surface of the liver.

[1] Read before the Brooklyn Surgical Society Dec. 6, 1934.

Group of cases collected harboring the endameba histolytica having various numbers of stool examinations bear witness to the correctness of a statement made by James, which I quote:

Survey figures show that one examination uncovers about one-third of actual histolytic infections present in a given community; 3 examinations between one-half and two-thirds, and 6 examinations up to about 90 percent, an indefinite number of examinations may be required before the remaining 10 percent of infections are found.

The following figures are from California:

Investigators	Number of persons examined	Number of stools examined each person	Percent positive for entire history
Kessel and Mason	2,731	3	9.8
Kofoid	6,834	3.3	13.1
Reynolds	1,000	4	16.0

Craig is of the opinion that between 6,000 and 12,000 people in this country harbor the endameba histolytica. This amounts to roughly 1 in every 10 of us.

The Panama Canal Zone was created in 1904. In that year began the education of the people as to hygienic principles, effective treatment for disease and the improvement of the water supply. Let us see what it accomplished. The following figures are from Clark.

Cases of persons dead of amebic infections per 1,000 autopsies

1905 to 1907	50	1914 to 1917	4
1907 to 1910	61	1917 to 1920	9
1910 to 1912	30	1920 to 1923	3
1912 to 1914	29		

Note the sudden drop in cases when a strictly modern and efficient water system was installed at two population centers between 1914 and 1917.

The right lobe of the liver is known to be the area of predeliction for amebic abscesses, but let us hurriedly look at some figures. In 95 cases coming to autopsy localization was as follows:

	Percent
Right lobe	55.7
Right and left lobes	16.8
All lobes	15.7
Left lobe	8.4
Right and quadrate lobes	1.05
Not stated	2.1

A word as to the carrier problem. It is doubtful if there is such a thing as a "healthy" carrier. In a series of 65 known carriers never known to have a symptom during life, who have come to autopsy as a result of other conditions, all revealed the existence of intestinal lesions of various degree.

The amebic granuloma subject is of more than usual significance. It would be interesting to know how many amebic tumor masses involving the large intestine have been removed by operators, as carcinoma. Gunn and Howard in 1931 reported three most instructive cases, which I should like to refer to very briefly. Case 1 had a large mass believed to be carcinoma in the splenic flexure of the colon. An enlarged adjacent gland was removed, diagnosed as inflammatory, and the mass left in situ until later, obstructive complications developed. Then a hard and firm growth 8 cm long by 5 cm in diameter with a constricted bowel lumen was removed. It was a mass of inflammatory fibrous tissue and edema which on section revealed endameba histolytica. Case 2 had a tumor involving the cecum removed because of obstructive symptoms measuring 15 cm in length and 7.5 cm in diameter, the wall of the bowel being 4 cm in thickness. The mass was dense, the induration extending into the mesentery and pericecal fat. Areas of necrosis were seen in the bowel lumen over the growth. Amebae were found. Case 3 had a tumor mass the size of two fists involving the ileocecal junction, with obstructive features which was removed as a carcinoma. This growth also revealed amebic granulation tissue with amebae. Runyan and Herrick report three somewhat similar cases which involved the ileo-cecal region and which were removed because of obstructive features. Two of their cases were recognized as probably amebic in origin before operation. All of these cases, incidentally, revealed deep ulcerated areas superimposed upon the firm inflammatory fibrous and edematous mass. Amebae were demonstrated in the ulcer bases and edges, in sections. It is apropos at this point to remark that surgery may be indicated in these cases for obstructive symptoms, if response to medical treatment is not satisfactory.

Let us now consider the retroperitoneal approach to collections of pus about the dome of the right lobe of the liver as described by Ochsner. It is of course unnecessary to repeat that any method of approach to this region, which will not soil the peritoneal nor the pleural cavities, is well worth much thought as the method of choice. The mortality rate in which this method was employed in 31 cases was 9.7 percent. Paravertebral block anesthesia is used with the right subcostal region elevated as for an approach to the kidney. An incision is made over the twelfth rib, which is resected subperiosteally. At times it will be found that the lower edge of the pleura is in close proximity to the bed of the rib. The posterior mass of spinal muscles is retracted. Now if a transverse incision, and it is important that it be transverse, is made at the level of the spine of the first lumbar vertebra in the rib bed it will practically always miss the lower edge of the pleural angle. The fibers of the diaphragm are now encountered, sometimes well developed, at other times only

a few fibers of muscle separating us from the renal fascia. If the finger is now directed upwards the kidney and its fascia can be displaced downward and the peritoneal coat of the diaphragm quite easily separated and the dome of the liver explored. This method can be used to reach the posteriorinferior, right inferior, extraperitoneal and at times the right anterosuperior spaces. A similar peritoneal stripping approach anteriorly through an incision at the lower costal border in the anterior abdominal wall has been suggested by Clairmont, when the abscess is in the right anterosuperior space or the left anteriorinferior and superior spaces.

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A SUGGESTED CHANGE IN CALCULATING DECOMPRESSION TABLES FOR DIVING[1] [2]

By JAMES A. HAWKINS, D. Sc., CHARLES W. SHILLING, Lieutenant, Medical Corps, United States Navy, and RAYMOND A. HANSEN, Lieutenant, United States Navy

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Men who have been working in compressed air, either in diving suits or caissons, are liable upon their return to atmospheric pressure to a variety of symptoms variously classified as "diver's palsy", "compressed-air illness" or "caisson disease." Paul Bert (1878) showed that these symptoms were due to the fact that gas (chiefly nitrogen) which goes into solution in the blood and tissues during exposure to compressed air is liberated in the form of bubbles on too rapid decompression, and produces local or general blockage of the circulation or other injury. Subsequent investigators have confirmed and extended Paul Bert's conclusions. Paul Bert (1878) pointed out that

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[2] We wish to express our appreciation for the suggestions and advice given by the Advisory Committee—Capt. E. W. Brown, Medical Corps. U. S. Navy; Commander E. L. Gayhart, Construction Corps, U. S. Navy; and Commander H. E. Saunders, Construction Corps, U. S. Navy, formerly a member of the committee.

We also recognize the assistance rendered by Lt. L. E. Bibby, U. S. Navy, and Lt. R. W. Clark, U. S. Navy, former officers in charge of the Experimental Diving Unit.

by means of very slow decompression caisson disease could be avoided, but he did not furnish any definite method. V. Schrötter (1906) suggested a uniform decompression at the rate of 20 minutes an atmosphere. Boycott, Damant, and Haldane (1908), in a most comprehensive study, established the stage method of decompression which is the basis for the present method of calculating the decompression tables of the Diving Manual of the United States Navy.

The data to be presented in this paper were accumulated during experimental escapes from various depths using the submarine escape appliance—"lung"—in an attempt to determine how long a subject could remain at a given depth and then come to the surface without any decompression and yet not develop caisson disease.

Apparatus—Submarine escape appliance.—The submarine escape appliance, described by Mankin in 1930, is a bellows-type, collapsible, stockinette-covered rubber bag, 9½ inches wide and 12 inches long with a capacity of 5.5 liters. It is fitted with a special-type rubber mouthpiece which provides a watertight seal at the lips and is connected to the bag by two ¾-inch hollow metal arms joined by a metal cylinder containing two 1-way mica disk valves directing the gas flow. The exhaled gas passes through the exhaust valve and metal arm directly into the bag, and the inhaled gas is drawn through a soda-lime cannister located inside the bag and immediately below the intake valve. A manually controlled master valve is provided adjacent to the mouthpiece which completely cuts off the flow of gas at the top of the submarine escape appliance to allow filling of the bag. A Schraeder charging valve is provided at the top of the bag for inflating with oxygen. A rubber flutter valve is fitted into the bottom of the bag to permit exhaust of excess gas and thus maintain an even pressure in the appliance. It is provided with suitable straps and snaps for holding it in place while in use. A nose clip is worn to prevent nasal breathing.

Diving tank.—The experiments were carried out in a vertical cylindrical diving tank, 10 feet 1 inch in height and 9 feet 10 inches in diameter with walls of 2-inch steel, tested to a pressure of 400 pounds per square inch. When in use the tank is filled with water to a height of 8 feet to allow an air pocket above the water. It is fitted with an airtight hatch on the upper end which opens downward into the tank. There are six 4½-inch ports in the side for observation purposes. The tank on the inside is well lighted by electric lights, and is equipped with loudspeakers and telephone.

Recompression chamber.—A recompression chamber developed by the United States Navy was available for the treatment of caisson disease. This chamber is a horizontal cylinder of 2-inch steel, 14 feet 7 inches long with an inside diameter of 6 feet 6½ inches. It is divided into two compartments by a bulkhead and door of the same gage steel.

The inner compartment is 9 feet 8 inches in length while the outer compartment is 4 feet 11 inches in length. It is possible to enter or leave the inner compartment without changing the pressure on the subject. Each compartment is provided with four 3-inch observation ports. The internal working pressure is 500 pounds per square inch. The recompression chamber is also equipped with electric lights, loudspeaker, and telephone. Air pressure can be controlled from either inside or outside, and accurate gages are provided for determining the pressure. In these experiments, the recompression chamber was used only for the treatment of caisson disease.

Method.—Subjects were exposed in the diving tank to air pressures equal to various depths of sea water. During the exposure time they breathed in the air pocket and exercised by swimming and diving in the water. Prior to the end of the exposure, the subject put on the submarine escape appliance, charged it with oxygen or air, submerged completely in the water and breathed into the appliance for 2 minutes after which ascent was simulated at the rate of 50 feet per minute by reduction of the air pressure. This constituted the entire decompression received by the subjects. Each subject made daily dives 5 days a week until the series was completed. Usually eight subjects were used for each depth and time of exposure, and this was called a run. In each series the depth was kept constant while the time of exposure was increased either ½ or 1 minute for each succeeding run.

The end-point in each series was the production of caisson disease of severe enough nature to necessitate terminating the series. Competent personnel were on call at all hours to treat the subjects on the appearance of caisson disease, which was encountered in several subjects in each series except series 2.

Experimental results.—The basis for the suggested change in the decompression tables is the data obtained from 2,143 experimental dives made over a period of 3 years. They were divided into 12 series and were conducted at depths of 100, 150, 167, 185, and 200 feet. Table 1 gives the information concerning these experimental dives.

It is seen from table 1 that all subjects did not go the same length of exposure without incurring "bends." In the 4 series, run with different subjects at 100 feet the time when "bends" first appeared varied from 37½ to 43 minutes. In the same way the exposures following which caisson disease developed at 150 feet varied from 18½ to 28 minutes.

TABLE 1.—*Experimental results*

Series	Depth gage	Number of runs	Individual exposures	Initial exposure time	Increase of exposure time	Final exposure time	Caisson disease			
							First case exposure time	Number of cases	Number of exposures	
									Before first case	After and including first case
	Feet			*Minutes*	*Minutes*	*Minutes*	*Minutes*			
2	100	75	600	8½	1	34½		0	600	0
7	100	18	75	14½	1	39½	37½	3	56	19
9	100	50	127	32½	1	51½	40½	5	46	81
11	100	59	429	3	1	48	43	5	408	21
10	150	28	70	18½	½	24½	18½	5	4	66
3	150	34	117	10½	1	22½	19½	8	80	37
8	150	18	71	9½	1	21½	20½	2	56	15
16	150	18	54	18	½	27	26½	2	42	12
15	150	74	214	10	½	28	28	5	206	8
4	167	38	141	6½	1	22½	17½	6	95	46
5	185	9	72	6½	1	15½	14½	3	56	16
6	200	23	173	7½	½	16	13½	2	133	40
Total			2,143					46	1,782	361

Discussion—Historical.—In order to clearly understand the theory of decompression of men following high-pressure air exposure it is necessary to consider the fundamental facts of saturation and desaturation of the tissues. Haldane (1922) says:

> The formation of bubbles depends, evidently, on the existence of a state of supersaturation of the body fluids with nitrogen. Nevertheless there is abundant evidence that when the excess of atmospheric pressure does not exceed normal by more than 1¼ atmospheres there is complete immunity from symptoms due to bubbles, however long the exposure to the compressed air may have been, and however rapid the decompression. Thus, bubbles of nitrogen are not liberated within the body unless the supersaturation corresponds to a decompression from a total pressure of more than 2¼ atmospheres. Now the volume of nitrogen which would tend to be liberated is the same when the total pressure is halved, whether that pressure be high or low. Hence it seemed to me probable that it would be just as safe to diminish the pressure rapidly from 4 atmospheres to 2, or 6 atmospheres to 3, as from 2 atmospheres to 1.

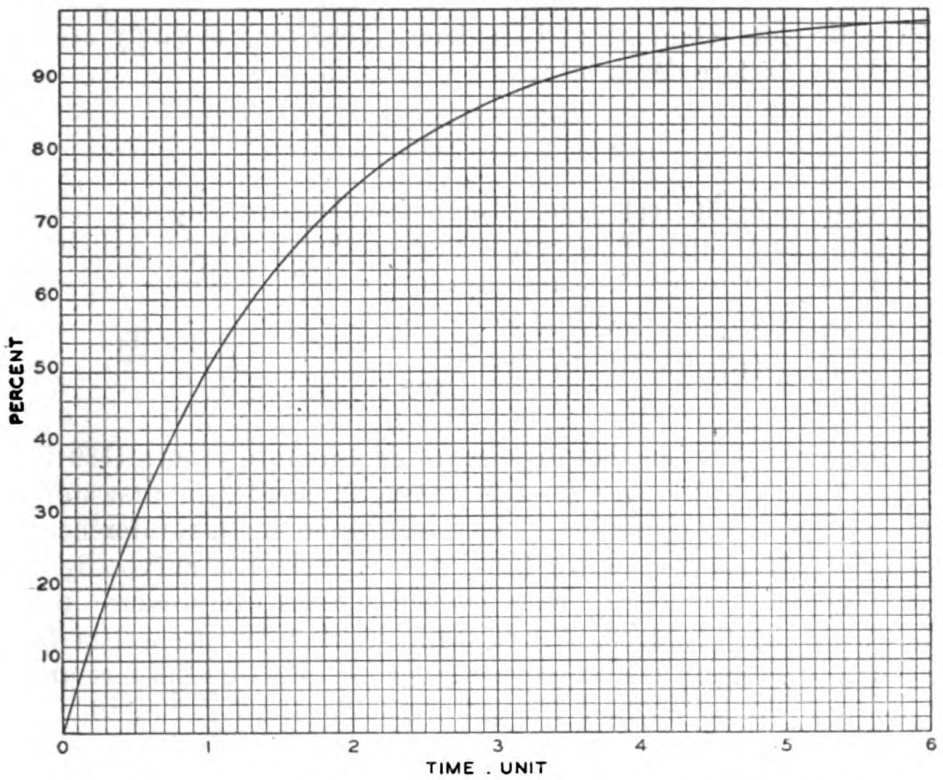
It was early shown that the degree of saturation of any part of the body depends not only on the pressure to which it is exposed and the duration of the exposure, but also upon its blood supply and its fat content. Thus, the blood itself is almost instantly saturated at the partial pressure of the gas in the lungs. Hill and Greenwood (1907) demonstrated that the kidney saturated and desaturated about 10 times as rapidly as the body as a whole. Vernon (1907) demonstrated that fat absorbed about six times as much nitrogen as blood; thus, fatty tissue would saturate and desaturate more slowly than non-fatty tissue. Haldane (1922) assumed that complete saturation of certain tissues is approached only after 5 hours exposure.

Whether or not the decompression is free from risks depends on the degree of supersaturation which can be borne with safety, the extent

to which the blood and tissues have had time or opportunity to become saturated, and the extent to which they have had time to become desaturated again during decompression.

On the basis of these facts, Boycott, Damant, and Haldane (1908) in calculating their stage decompression tables assumed the existence of 5-, 10-, 20-, 40-, and 75-minute tissues. In these tables they did not permit any of the tissues to exceed the ratio of 2.0 to 1 except at the last stage where the ratio might be as high as 2.3 to 1. This ratio was computed from the relative nitrogen saturation of the tissues in depth absolute to the nitrogen at normal atmospheric pressure.

FIGURE 1



Method of computation.—In order to permit a clearer understanding of the discussion to follow, the method of calculating a decompression table is given in detail. As an example, let us consider a diver at 108 feet (gage) for 30 minutes' exposure. Haldane (1922) states that the progress of saturation of the body with nitrogen is a logarithmic curve of the form shown in figure 1. From this figure the percentage saturation can be read off the curve, provided the duration of the exposure to pressure and the time required to produce half saturation of the tissues in question are both known. Thus the tissues which half saturate in 5 minutes (5-minute tissues) would in 30 minutes (6 time units) be 98 percent saturated. The 10-minute tissues would be 87 percent, the 20-minute tissues would be 65 percent, the

40-minute tissues would be 40 percent, and the 75-minute tissues would be 17 percent saturated.

According to Henry's law governing solubility of a gas, the tissues which are three-fourths saturated at 4 atmospheres absolute would contain as much nitrogen as if completely saturated at 3 atmospheres absolute. Thus in the present example the 5-minute tissues would contain as much nitrogen as if saturated at $0.98 \times 108 = 106$ feet gage. Similarly the other tissues are found to be saturated as if at 95, 70, 43, and 18 feet gage respectively.

We have found by use of the time unit and saturation curve that the most completely saturated tissues (5-minute tissues) are saturated to a pressure corresponding to 106 feet gage. We know from the work of Boycott, Damant, and Haldane (1908), we can safely ascend half of the absolute depth or to

$$\frac{106+33}{2}=69$$

feet absolute or 36 feet gage. But since desaturation also occurs during the 3 minutes of ascent, it is possible to bring the diver to 30 feet gage. During the 3 minutes of ascent he is subjected to an average pressure corresponding to

$$\frac{(108+33)+(30+33)}{2}=102 \text{ feet absolute.}$$

The difference between the pressure in his 5-minute tissues (139 feet absolute) and this average pressure during ascent to the first stop is $139-102=37$ feet absolute and he is subjected to this difference of pressure for 3 minutes or 0.6 time units. From the curve (fig. 1), we find that 0.6 time units is 34 percent. Thus the pressure in his tissues diminishes $0.34 \times 37 = 13$ feet and so when he reaches the first stop, the pressure in his 5-minute tissues is $139-13=126$ feet absolute. In a similar manner the saturations of the 10-, 20-, 40-, and 75-minute tissues are calculated. (See table 2.)

Since, in stage decompression, the diver is brought up in 10-foot stages, the next stop in this instance will be 20 feet (53 feet absolute). However, to maintain a 2-to-1 ratio, the diver has to remain at the 30-foot stop until the pressure in his most deeply saturated tissues (5-minute tissues at this stop) drops to twice the pressure of the 20-foot stop ($2 \times 53 = 106$). This drop will have to be $126-106=20$ feet. The difference in pressure to which the diver is subjected is $126-63=63$. If he remained long enough at this stop, the pressure in his body would drop to the pressure of the stop or 63 feet absolute, but he is to be desaturated only 20 feet at this point. So he will stay at this stop (30 feet) $\frac{20}{63}$ or 32 percent of the time required to desaturate

to the pressure of the stop. Applying this percentage to the curve (figure 1), we get 0.6 time unit and as 0.6×5 (minute tissues)=3 minutes, we know we keep the diver at 30 feet for 3 minutes in order not to exceed a 2-to-1 ratio. Actually we find when we calculate many of Haldane's tables (1922) that he often goes to a ratio of 2.1 or even 2.3 to 1, but this is well within the safety factor. As already shown, the pressure in his tissues drops 32 percent of the difference between the tissue tension at the start of the 3 minutes and pressure of the stop, i.e., in the case of the 5-minute tissues already figured 126−106 or 20 feet. By applying the time factor and percentage, all of the other tissues are calculated in the same manner.

By following the same method of calculating used to determine the length of stay at 30 feet, we find that it is necessary to stay 7 minutes at 20 feet. But in this case, it is the 10-minute tissues which are so deeply saturated as to be the governing tissues. That is, it is necessary to extend the time of this stop more than is required by the 5-minute tissues in order that the 10-minute tissues may not exceed a ratio of 2 to 1. In the same manner, it is the 20-minute tissues that determine the 10-minute stop required at 10 feet. All of these calculations are presented in table 2.

TABLE 2.—*Depth, 108; Time, 30—Decompression chart*

Tissue		Exposure	To first stop	First stop	Second stop	Third stop
	Depth	108	69	30	20	10
	Depth (absolute)	141	102	63	53	43
	Time	30	3	3	7	10
5	Time units	6.0	0.6	0.6	1.4	2.0
	Percent	0.985	0.34	0.34	0.621	0.75
	Difference in pressure	108	37	63	52	30
	Difference in saturation	+106	−13	−21	−32	−23
	Tissue saturation	139	126	105	73	50
10	Time units	3.0	0.3	0.3	0.7	1.0
	Percent	0.875	0.187	0.187	0.384	0.5
	Difference in pressure	108	26	60	59	46
	Difference in saturation	+95	−5	−11	−23	−23
	Tissue saturation	128	123	112	89	66
20	Time units	1.5	0.15	0.15	0.35	0.5
	Percent	0.646	0.098	0.098	0.215	0.293
	Difference in pressure	108	1	40	46	46
	Difference in saturation	+70	0	−4	−10	−13
	Tissue saturation	103	103	99	89	76
40	Time units	0.75	0.075	0.075	0.175	0.25
	Percent	0.405	0.05	0.05	0.113	0.158
	Difference in pressure	108	25	15	24	31
	Difference in saturation	+44	+1	−1	−3	−5
	Tissue saturation	77	78	77	74	69
75	Time units	0.4	0.04	0.04	0.093	0.133
	Percent	0.242	0.027	0.027	0.062	0.088
	Difference in pressure	108	43	3	7	17
	Difference in saturation	+26	+1	0	0	−1
	Tissue saturation	59	60	60	60	59

Interpretation of results.—Following the method just outlined, the saturation of each of the theoretical tissues upon reaching the surface was calculated for every case of caisson disease encountered during

the course of the experimental dives. These are all presented in figure 2 in which the abscissae represent the individual cases of caisson disease grouped according to the depth of exposure and the ordinates

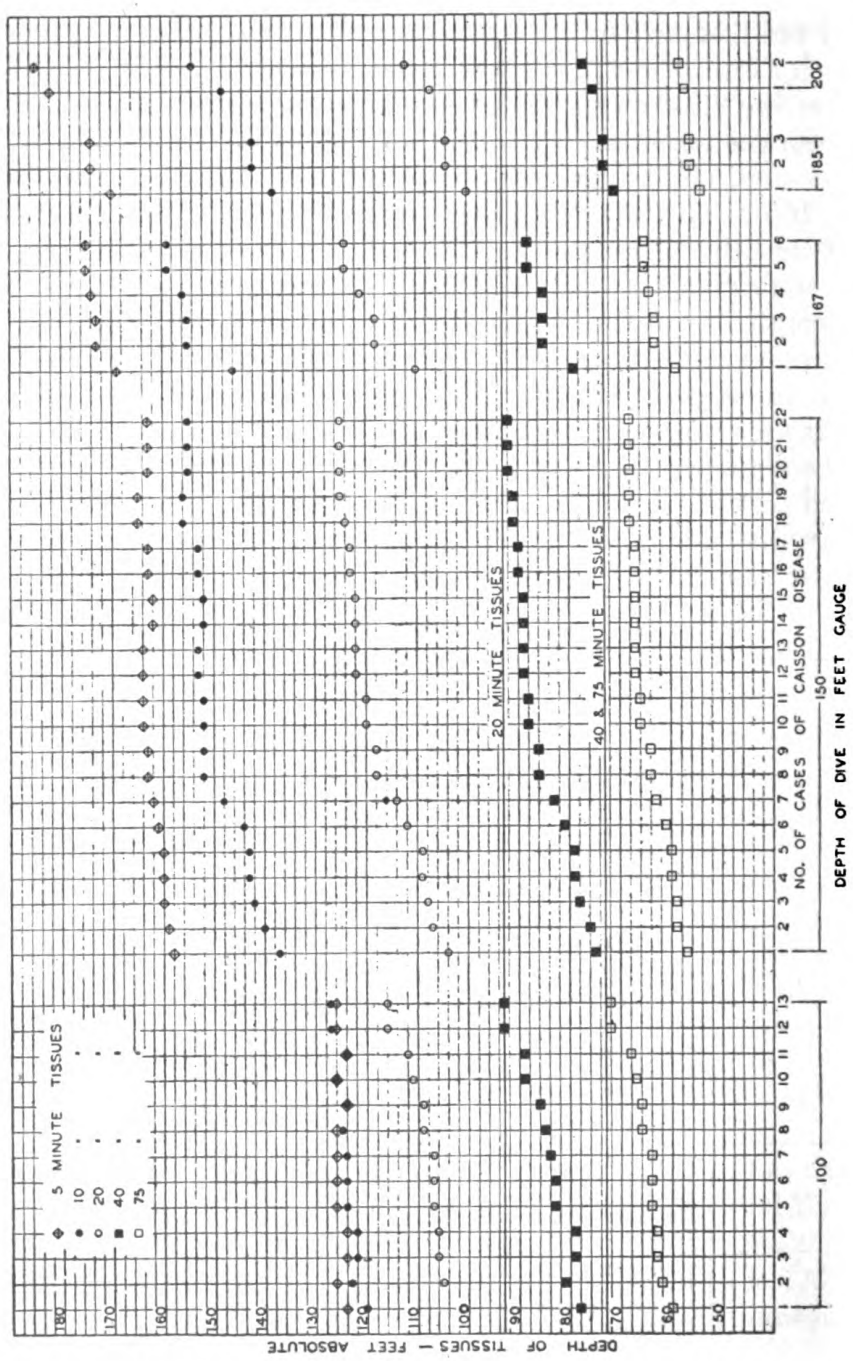


FIGURE 2

represent the saturation of each of the theoretical tissues in feet absolute upon reaching the surface. In order to determine the saturation ratio for any of the tissues thus plotted, it is only necessary to

divide the plotted depth in feet by 33. By doing this, we find that at a hundred-foot depth, the 5-minute tissues have a ratio of 3.8 to 1 while at 150 feet this ratio ranges from 4.8 to 1 to 5 to 1. At 200 feet, the ratio is as high as 5.6 to 1. As a matter of fact, the only tissues not exceeding a 2 to 1 ratio, i. e., plotted under the 66-foot level are the 75-minute tissues. The 10-minute tissues range from a ratio of 3.6 to 1 at 100 feet to 4.7 to 1 at 200 feet, the 20-minute tissues ranged from a saturation ration of 3.1 to 1 to 3.8 to 1, and the 40-minute tissues ranged from 2.3 to 1 up to 2.8 to 1. By looking only at figure 2, one would be forced to conclude that any of the tissues other than the 75-minute tissues might have caused the bubble retention which produced the caisson disease.

But by the same method of calculating, the saturation of each of the theoretical tissues was determined for the run prior to the caisson disease producing run, and again as shown by these results presented in table 3 the ratio for all of the tissues except the 75-minute tissues is high enough to have produced caisson disease. Yet we know that caisson disease did not develop until after a longer exposure, and thus a greater saturation than any reported in this table. In fact, in some instances diving operations were continued for from 10 to 14 runs after the development of the first case of caisson disease. Actually, as noted in table 1, of a total of 2,143 individual exposures, 1,782 were prior to the development of the first case of caisson disease in their respective runs while there were 361 including and following the first case. It is thus shown that in many individuals even greater ratios than those encountered in either table 3 or in figure 2 can be borne with impunity.

TABLE 3.—*Run prior to the run producing caisson disease in each series*

Series no.	Depth of dive feet gage	Number of subjects	Length of exposure	Depth of theoretical tissues in feet absolute (minute tissues)					Tissue saturation ratio (minute tissues)				
				5	10	20	40	75	5	10	20	40	75
			Minutes										
2	100	8	34½	126	121	102	78	60	3.8–1	3.7–1	3.1–1	2.4–1	1.8–1
7	100	8	36½	126	122	104	80	62	3.8–1	3.7–1	3.2–1	2.4–1	1.9–1
9	100	8	39½	126	124	107	83	64	3.8–1	3.8–1	3.2–1	2.5–1	1.9–1
11	100	8	42	124	124	109	85	65	3.8–1	3.8–1	3.3–1	2.6–1	2.0–1
10	150	8	18	158	136	103	74	57	4.8–1	4.1–1	3.1–1	2.2–1	1.7–1
3	150	8	18½	158	137	104	75	58	4.8–1	4.2–1	3.2–1	2.3–1	1.8–1
8	150	8	19½	159	140	107	77	59	4.8–1	4.2–1	3.2–1	2.3–1	1.8–1
16	150	6	26	162	151	121	88	67	4.9–1	4.6–1	3.7–1	2.7–1	2.0–1
15	150	5	27½	163	154	125	91	68	4.9–1	4.7–1	3.8–1	2.8–1	2.1–1
4	167	8	16½	168	143	107	75	58	5.1–1	4.3–1	3.2–1	2.3–1	1.8–1
5	185	8	13½	174	142	100	71	56	5.3–1	4.3–1	3.0–1	2.2–1	1.7–1
6	200	8	13	182	149	107	75	57	5.5–1	4.5–1	3.2–1	2.3–1	1.7–1

Depth (absolute) = depth (gage) + 33 feet.

It is conclusively shown by table 3 that all of the subjects were able to tolerate saturations in the 5- and the 10-minute tissues greatly in excess of the supposed safe ratio of 2 to 1. In fact, it is evident that the saturation of the 5- and the 10-minute tissues has no relationship to the production of caisson disease. These conclusions are based not only on the runs presented in table 3, for it should be borne in mind that in every series many of the runs preceding those presented in table 3 left the tissues saturated to a degree greatly exceeding the 2-to-1 ratio.

The 20-minute tissues appear to be the first ones it is necessary to consider in the production of caisson disease, for when these tissues reach a saturation of 63 feet (96 feet absolute) caisson disease begins to appear. By the same reasoning by which the 5- and 10-minute tissues were eliminated, the 20-minute tissues can be carried to a ratio of 3 to 1 without danger of caisson disease. Whether these tissues could go to a still higher ratio without trouble resulting cannot be proved from the dives made, as by the time the 20-minute tissues are left at 63 feet, the 40-minute tissues have reached a saturation of 41 to 43 feet, or a ratio of 2.3 to 1. Thus, it is impossible to determine whether the resulting attack of caisson disease was produced in the 20- or the 40-minute tissues.

Proposed change in tables.—From the foregoing results, it is seen to be safe to use a decompression table calculated without considering the desaturation rate of the 5- and 10-minute tissues and using a ratio of 3 to 1 for the 20-minute tissues, and 2.3 to 1 for the 40- and 75-minute tissues at the last stop. The time of decompression can thus be shortened considerably on dives of short duration where the 40- and 75-minute tissues do not become deeply saturated.

Tables have been calculated (see table 4) for certain depths and for various times of exposure in order to show the advantage in time saved in ascent, in which the 5- and 10-minute tissues are ignored, and carrying a ratio of 2.8 to 1 (3 to 1 would be safe) in the 20-minute tissues and 2.0 to 1 in the 40- and 75-minute tissues. These ratios are represented by the two horizontal lines labeled 30-minute tissues, and 40- and 75-minute tissues in figure 2. The saving in decompression time is large in many instances. For example, following a dive of from 20 to 30 minutes at 100 feet, the present tables require a decompression of 23 minutes while the proposed tables require but 4 minutes decompression. An exposure of from 30 to 40 minutes at 100 feet requires 33 minutes by the present tables and only 18 minutes by the proposed tables.

TABLE 4

Depth in feet	Time from surface to start of ascent	Diving-manual tables										Proposed tables				Total time of ascent	
		Time to first stop	Stops in feet									Time to first stop	Stops in feet			Diving-manual table	Proposed table
			90	80	70	60	50	40	30	20	10		30	20	10		
	Minutes	*Min.*										*Min.*				*Min.*	*Min.*
0 to 40	Over 180	1									5	1			5	6	6
41 to 50	0 to 60	2									5	1				7	1
	60 to 120	2									10	1			6	12	7
	120 to 180	2									10	1			10	12	11
51 to 60	0 to 60	2									10	1				12	1
	60 to 90	2									10	1			10	12	11
	90 to 120	2								5	15	1			17	22	18
	120 to 180	2								5	15	1		6	15	22	22
	Over 180	2								10	20	1		10	30	32	41
61 to 70	0 to 30	2								3	5	1				10	1
	30 to 60	2								5	12	1			7	19	8
	60 to 90	2								10	20	1			20	32	21
	90 to 120	2								10	20	1		12	20	32	33
71 to 80	0 to 30	2								5	15	2				22	2
	30 to 60	2								10	20	2			18	32	20
	60 to 90	2								15	30	2		14	19	47	35
	90 to 120	2								15	30	1		24	19	47	44
81 to 90	0 to 30	2								5	15	2				22	2
	30 to 40	2								5	15	2			9	22	11
	40 to 60	2							3	10	15	2			27	30	29
	60 to 90	2							5	15	25	2			27	47	29
91 to 100	0 to 10	3								3	5	2				11	2
	10 to 20	3							3	7	10	2				23	2
	20 to 30	3							3	7	10	2			2	23	4
	30 to 40	3							5	10	15	2			16	33	18
	40 to 50	2							10	15	20	2			18	47	20
	50 to 60	2							10	15	20	2		18	19	47	39
101 to 120	0 to 15	3							2	3	7	3				15	3
	15 to 30	3							5	10	15	2			14	33	16
121 to 140	0 to 15	3						2	5	10	12	3				32	3
	15 to 30	3					3	5	10	15	25	3		8	19	61	30
141 to 160	0 to 15	3					2	3	5	7	10	3			5	30	8
	15 to 30	3					3	5	10	15	20	3		19	19	56	41
161 to 180	0 to 15	3					3	3	7	10	15	4			8	41	12
	15 to 30	3				2	2	3	10	15	25	3	3	27	19	60	59
181 to 200	0 to 15	3				3	3	5	7	10	20	4			15	51	19
	15 to 30	3			3	3	3	5	10	20	20	4	12	27	19	67	62
201 to 225	0 to 15	8		3	5	7	7	10	15	20	20	4		5	20	95	25
226 to 250	0 to 15	4	2	3	5	7	10	10	15	20	30	5		16	19	106	40

Although these proposed changes in calculating decompression tables are based on the results of 2,143 experimental dives using the submarine escape appliance, yet it is hoped to further check them, both in suit dives at the experimental diving unit and in the open sea under service conditions.

SUMMARY

An analysis of 2,143 experimental dives has been made in which it is shown that the saturation of the 5- and 10-minute tissues has no bearing on the production of caisson disease. It is also shown that the 20-minute tissues may have a saturation ratio of 3 to 1 without the development of caisson disease.

The method of calculating a decompression table has been given, together with its relation to tissue saturation and desaturation in connection with the production of caisson disease.

The findings obtained from these experimental dives have been used as a basis for calculating decompression tables. These tables reduce the time of decompression following dives of short duration.

CONCLUSION

New decompression tables have been calculated without considering the desaturation of the 5- and 10-minute tissues, and allowing a ratio of 2.8 to 1 for the 20-minute tissues, and 2.0 to 1 for the 40- and 75-minute tissues.

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PROMOTION AND EXPERIENCE

By James C. Pryor, Rear Admiral, Medical Corps, United States Navy, retired

Promotion is worth working for.

The average young medical officer having just entered the Army or Navy may be assumed to have professional ambition and enthusiasm normal to his age, and scarcely would be human if he failed to avail himself of every opportunity for promotion in his corps.

Opportunity for promotion comes slowly, and when it does come it brings with it a demand that professional as well as mental, moral, and physical fitness for promotion shall be demonstrated by the aspirant.

Professional fitness for admission to Army or Navy medical corps or promotion therein requires that—(*a*) The candidate shall have absolved all of the requirements of a class A medical school for the degree of doctor of medicine, (*b*) shall have passed examination preliminary to admission to his corps or any earlier promotion therein, and (*c*) he shall have been a diligent student in the "University of Experience."

It is the third of these requirements, viz., that "He shall have been a diligent student in the University of Experience", to which attention is invited for a few moments.

Experience is either, (*a*) personal, as the term implies or, (*b*) vicarious, that which has been personal to others and communicated to us in some manner.

(*a*) Personal experience, that which falls under the immediate observation of an individual is essentially limited by opportunity and environment. The keener the mind and more retentive the memory the greater will be the value of personal experience, but personal experience is limited by personal opportunity and environment.

(*b*) Vicarious experience comes to us through the narrated, written, or printed experience of others.

Most commonly this experience reaches us through the printed word, hence it comes to us through magazines, brochures, books, reprints, or translations.

It is largely through this vicarious experience that the busy practitioner or specialist is able to keep informed concerning the rapid changes which are taking place in his profession.

The medical officer desirous of being prepared professionally for opportunity for promotion must follow the same path taken by the up-to-date practitioner in order to prepare himself for his duty to his fellow man and to himself.

And, further, being a specialist in military or naval medicine he must add to his own personal experience the vicarious experience which comes through the reading of military medical magazines, if he would be well qualified. These journals carry discussions of military medical matters for the military specialist whose duties are concerned with massed men in peace or in war.

Another method of profiting by the experience of others is attending meetings of medical men and hearing the discussion of live professional topics.

Among the most pleasant incidents of my professional life have been the contacts with distinguished physicians and surgeons who prize their membership in the Association of Military Surgeons and attend its meetings, often at a personal sacrifice.

As a result of long experience in the United States Naval Medical Corps the writer urges upon members of that corps the maintenance of membership in the Association of Military Surgeons of the United States which carries with it a subscription to the Military Surgeon at a total cost of $3 per annum.

The Association of Military Surgeons of the United States operates under a charter from Congress. It is composed of members of the several governmental medical services, and is the only organization of medico-military officers of the Government which is recognized officially.

Its aim is the furtherance of the professional and personal interests of the personnel of the medical officers of the Government services.

The Military Surgeon is published monthly, and brings to its readers much vicarious experience of value to a candidate for promotion.

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Naval medical officers, unless they have had personal experience with a landing force, do not realize how different is the problem when, with their landing force, they leave the sanitary surroundings of their ship with its pure drinking water, satisfactory facilities for food preparation, sanitary toilets, and comparative freedom from insect vectors, and go ashore to select a camp site and establish a camp.

Problems arise that are unexpected, perhaps—problems that cause the naval medical officer to realize that he has, with too little preparation, suddenly assumed the function of an Army medical officer.

In other words the naval medical officer who is properly prepared for promotion is the one who is ready and able to perform successfully the duties of the military medical officer on shore.

When the naval medical officer lands with troops, he becomes a military medical officer.

That he is expected to be prepared to perform these duties is indicated by consideration of such questions as: "How would you protect a company of marines on duty in Shanghai against cholera"? "Give the different types of latrines; uses and limitations", "Discuss the relation of soil pollution to disease", which questions very recently have been asked of candidates for promotion in the Naval Medical Corps. Such questions show need for knowledge of military as well as naval medicine.

In view of the foregoing, it is felt that such articles as "The sterilization of instruments in the field", by Col. G. P. Lawrence, Medical Reserve, United States Army; "Recent developments in medical-field equipment and transport at the Medical Department Equipment Laboratory, United States Army", by Col. G. L. McKinney, Medical Corps, United States Army; "Medical progress and the war", by Col. W. N. Bispham, Medical Corps, United States Army, which have appeared in recent issues of the Military Surgeon, cannot fail to interest the serious-minded naval medical officer who is mindful of possible duties ashore at any moment, and of fitness for promotion when opportunity comes to demonstrate that fitness.

Because promotion is worth working for, and because the Association of Military Surgeons of the United States and its official organ, the Military Surgeon, can aid the medical officer in preparation for promotion and for greater efficiency, membership in the Association of Military Surgeons of the United States is urged by one whose long service life has shown him the wisdom of this advice. The member who attends the meetings gains personal experience, and if he reads the Military Surgeon he is the better prepared by vicarious experience for his duty and selection for promotion in his corps.

THE TREATMENT OF VESICAL AND VASCULAR CONDITIONS BY OPERATIONS ON THE SYMPATHETIC NERVOUS SYSTEM [1]

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Knowledge of the sympathetic nervous system dates back to 1732, when Winslow recognized the difference between the central nervous system and what he called the sympathetic nervous system. The term he chose to designate this other nervous system was based on his assumption that it was through the ganglions and rami that certain functions of the body were coordinated by "sympathy." In 1764 Johnstone took exception to the term "sympathetic" and applied the term "involuntary", and in 1801 Bichat, thinking the sympathetic or involuntary nervous system was involved in the vegetative functions of the body, called it the "vegetative" nervous system. Further to confuse the terminology, in 1898 Langley, using Gaskell's differentiation of the sympathetic and parasympathetic nervous system, included them both under the term "autonomic." At the present time Langley's classification is the most acceptable and has proved of value in describing this nervous system.

Claude Bernard, in 1851, performed an experiment on a rabbit which founded the present therapeutic application of sympathetic ganglionectomy. He divided the nerves to the ear, following which he noticed that there was definite and persistent vasodilatation. He assumed that the vasoconstrictor fibers had been divided and that there was reflex vasodilatation. Surgery of the sympathetic nervous system probably goes back to 1889, when Alexander removed the cervical sympathetic ganglions in the treatment of epilepsy. In 1899 Jaboulay performed periarterial neurectomy for pain in the lower extremity. It was not until 1913, however, that Leriche performed periarterial neurectomy for vasospastic diseases. In 1916 Jonnesco removed a stellate ganglion by means of an anterior approach, and in 1923 Royle and Hunter divided the rami in the treatment of spastic paralysis. Adson and Brown removed the lumbar sympathetic ganglions for spastic paralysis in 1923, and in 1924 for Raynaud's disease. In the following year Davis and Kanavel, and Diez carried out the same procedure for relief of vasospastic diseases. In 1924 Cotte resected the presacral nerves for pelvic pain, and in 1926 Pieri used the same operation for relief of vesical pain. Learmonth and Braasch, in 1930, then developed a definite technic and analyzed the physiologic influence of the presacral nerves on the bladder.

The sympathetic nervous system is so associated with our everyday life that every observation regarding its various functions is of tremendous significance. The part that the sympathetic nervous system

[1] Read before the Sixth Medical Reserve Officers' Inactive Duty Training School, Rochester, Minn., Oct. 16, 1934.

plays in warfare is just beginning to be understood, as it probably influences certain reactions, such as shell shock and the development of certain chronic ailments. The two conditions, which will prove of interest to the Medical Departments of the Army and Navy, in which advances have been made, are treatment, by means of operations on the sympathetic nervous system, of atonic and irritative lesions of the bladder and vascular diseases of the extremities.

Anatomically, the sympathetic or autonomic nervous system is composed of series of ganglions, nerves, and plexuses from which the viscera, glands, heart, blood vessels, and smooth muscles in other situations are innervated. The most conspicuous feature of the system is a chain of sympathetic ganglions which extends vertically on each side of the spinal column through the neck, thorax, and abdomen; the spinal nerves are connected with the sympathetic trunk, on the same side, by one or more rami communicantes, through which they receive sympathetic fibers for the control of blood vessels, sweat glands, and smooth muscles situated within the territory of their distribution.

Sympathectomy in diseases of the bladder—Atonic and irritative lesions of the urinary bladder.—Urinary retention, resulting from congenital, neoplastic, traumatic, or inflammatory lesions of the spinal cord, that interfere with but do not destroy innervation of the bladder, has been partially or totally relieved by an operation described by Learmonth and Braasch. This operation consists of section of the presacral sympathetic fibers. The success of the operation, however, depends on the presence of some parasympathetic innervation; therefore, such conditions as tabes dorsalis and other degenerative lesions of the spinal cord are not amenable to treatment.

Vesical dysfunction.—Three distinct conditions of vesical dysfunction, paresis of the bladder, vesical pain, and spasmodic conditions of the vesical neck, respond to this type of operation. In considering the surgical relief of paresis of the bladder it should be borne in mind that the sympathetic nerves have been called the "filling nerves", and they increase the tonicity of the internal sphincter and inhibit contraction of the detrusor muscle, whereas the parasympathetic nerves are the "emptying nerves", and they relax the internal sphincter and contract the detrusor muscle. Normally these two systems coordinate in regulating the filling and emptying of the bladder. When it can be proved that paresis of the bladder is the result of imbalance between these two systems, and that a more nearly normal balance can be created by relieving the sympathetic influence, then resection of the presacral nerves is indicated.

Vesical pain.—Operations on the presacral nerves for vesical pain are followed by relief in cases of both inflammatory and neoplastic diseases. The mechanism by which the pain is relieved in these

conditions is still in dispute, inasmuch as some authorities insist that painful stimuli are carried by the sympathetic fibers, and others contend that the relief of pain results from the abolition of contracting impulses to smooth muscle. Whatever the mechanism, it has been established that removal of the presacral nerves, by interrupting the sympathetic fibers of the bladder, does relieve certain forms of vesical pain.

Malignancy of the bladder which has progressed to the inoperable stage presents a clinical problem. Resection of the presacral nerves may relieve the pain but a more extensive resection of nerves should be carried out when possible. Learmonth and Braasch have emphasized that part of the sensation of vesical pain is transmittee by the parasympathetic fibers and if complete denervation of the bladder is to be done, it is necessary to remove the hypogastric ganglions. These are the terminal ganglions for the hypogastric nerves and form the junction of the sympathetic and parasympathetic fibers before they are distributed to the bladder. This operation is followed by incontinence, and in the presence of an inoperable malignant growth or an extensive painful condition which is the result of inflammation, there is no other choice. In some cases the tissues through which the dissection for removal of the ganglions must be made have become so involved in the pathologic process that resection of the presacral nerve is all that can be done.

Resection of the presacral nerves or superior hypogastric plexus.—Because of their accessibility, the presacral nerves, or the superior hypogastric plexus of Hovelacque, have been resected for relief of disorders of the bladder, colon and rectum, uterus, and ovaries. The presacral nerves, in the majority of cases, are a plexus of nerves, which is situated just below the bifurcation of the aorta, beneath the posterior peritoneum, and which extends into the pelvis and terminates in two hypogastric nerves. The superior hypogastric plexus consists of two main bundles, which carry sympathetic rami from the lumbar sympathetic chain on either side, and a third group of contributing fibers, which descends from the intermesenteric plexus and carries fibers from the celiac and semilunar ganglions. These branches converge just below the bifurcation of the aorta and anteriorly to the lower lumbar vertebra.

The patient is placed in Trendelenburg's position. The abdomen is opened by means of an incision which is made low in the median line. After packing off the intestines, the posterior peritoneum, which covers the lower lumbar vertebrae and sacral promontory, can easily be exposed. If the patient is thin, the presacral nerves, or hypogastric plexus, can easily be visualized lying just beneath the peritoneum, in a fascial plane which is superior to the sacral vessels. A longitudinal incision is made in the posterior peritoneum; this

incision is begun above the bifurcation of the aorta, passes over the sacral promontory, and is continued into the pelvis. The presacral nerves can be dissected free and resected. The dissection should be carried upward until the contributing branches can be visualized, and downward until the nerves can be seen to form the two hypogastric nerves.

The wound is closed in the usual manner. The postoperative course is usually uneventful, and there are no untoward effects, except perhaps paralysis of the ejaculatory ducts. It is necessary to warn men that, after the operation, although they will be able to perform the sexual act and to experience a normal orgasm, ejaculation will not occur.

Vascular conditions of the extremities.—Diseases of the vessels of the extremities not only affect veterans who have been exposed to trench warfare and cold wet decks but also afflict civilians. Whether or not exposure, smoking, or hereditary tendencies prove to be predisposing factors, pain, gangrene, and ultimate amputation create a serious and disabling condition. The most prevalent vascular disease among men and, fortunately, the disease most amenable to sympathectomy is thrombo-angiitis obliterans.

Thrombo-angiitis obliterans occurs in adult life, has a predilection for men, and affects persons of all races in spite of the fact that it formerly was supposed to occur more commonly among Jews than among others. The underlying causes have not all been determined. The disease seems to progress after infection of the inner layers of the arterial wall and formation of a clot which occludes the vessels and decreases the blood supply to the extremity. The infection and the formation of clot vary in degree and distribution. The condition may affect the distal part of one principal artery, or it may include all of the principal arteries of all extremities at different periods. The usual course of this disease is rather slow; the main vessels of the feet and legs are involved early, and those of the upper extremities later. When it is economically possible, relief can be brought about by continuous rest in bed, by applying heat to the extremities, and by the intravenous administration of vaccines. In time, organization of the intravascular clot takes place and circulation will be partly restored. However, many patients are compelled to work, subjecting their hands and feet to trauma, and ulcers may develop sooner or later, and refuse to heal. These ulcers become infected, the infection spreads to adjacent tissues, and more thrombosis and gangrene appear which necessitate amputation of the extremity. By means of operation on the sympathetic system, it is possible to relieve the vasomotor spasm of the collateral vessels; this improves circulation, tends to prevent ulceration, infection, and gangrene, and hastens healing of existing ulcers and abrasions. In suitable cases, selected by means

of the "fever test", the pain usually is relieved, the circulation is improved, and the color of the skin changes from mottled reddish-blue to pink. Also, the temperature of the skin is increased by from 2° to 10° F. depending on the amount of vasoconstriction that was present before operation, the ulcers begin to heal, and the patient may be restored to his former status as a wage earner. Moreover, the operation may prevent further gangrene and extension of the process to the opposite extremity, which usually is involved to a slighter degree than is the extremity which causes the symptoms. The operation is not advised in the milder cases, in which the patients are not inconvenienced greatly, are free from ulcers, and are able to carry on regular work under symptomatic treatment. In a recent review of cases which were encountered in the last 2 to 5 years, and in which operation on the sympathetic system had not been performed, it was discovered that in 25 to 30 percent amputation of one or more extremities had been necessary. Less than 5 percent of the 70 patients who have been subjected to operation on the sympathetic nervous system have been obliged to undergo amputation. This comparison emphasizes the value of operations on the sympathetic system in protecting patients who have thrombo-angiitis obliterans from the loss of one or more extremities by amputation.

The other type of occlusive vascular disease, the underlying cause of which is arteriosclerotic changes in the vessels, is not accompanied by vasoconstrictor spasm of associated vessels, and therefore does not respond to operation.

Lumbar operation.—An abdominal incision is made from the symphysis pubis to a point 5 to 7 cm (2 to 3 inches) above the umbilicus, between the rectus abdominis muscles and to one side of the umbilicus. The Trendelenburg position is used, and the intestines are packed upward, as they are in pelvic operations. To expose the left lumbar sympathetic chain, it is necessary to mobilize the sigmoid and the lower portion of the descending colon by incising the peritoneum above and just lateral to the anterolateral border of the upper portion of the sigmoid and lateral to the attachment of the lower portion of the descending colon. After the large intestine has been elevated and retracted, with the peritoneum, there is exposed the retroperitoneum, the ureter, the left common iliac artery and vein, the lower end of the abdominal aorta, the genitofemoral nerve, the psoas muscles, the lumbar vertebrae, the lymph nodes, and the lumbar sympathetic ganglions, trunks, and rami, which lie on the lumbar vertebrae, just mesial to the psoas muscles. By retracting the abdominal aorta mesially, the sympathetic ganglions, trunks, and rami can be dissected free by beginning with the fourth lumbar ganglions and dividing all communicating rami.

The approach to the lumbar sympathetic ganglions on the right side is similar to that on the left, except that the peritoneal incision is made just lateral to the right lateral border of the inferior vena cava. It is carried downward over the right common iliac vein into the true pelvis, upward and mesially along the root of the mesentery of the small intestine, partially across the vena cava for a distance of 15 cm from the brim of the pelvis, and downward into the pelvis for a distance of 5 to 7 cm. The cecum and the small intestine are retracted outward and upward. The inferior vena cava is retracted mesially, and the common iliac vein is retracted downward and mesially. Further exposure and removal of the lumbar sympathetic ganglions, and division of all of the rami and of the sympathetic trunk are similar to the procedures employed on the left side. Closure consists in accurate apposition of both incisions in the posterior peritoneum to prevent retroperitoneal hernia, and accurate closure of the abdominal wall to prevent the more common type of post-operative hernia.

Thoracic operation.—The patient is placed prone on an operating table that is equipped with a cerebellar head rest. A posteromedian incision is made in the skin, from the tip of the spine of the fifth cervical vertebra to the tip of the spine of the fourth thoracic vertebra. The incision is carried down to the spinous processes, thus exposing the fascia over the trapezius muscle on both sides. The fascia and muscle are incised on each side; this incision is parallel with the spinous processes and extends from the sixth cervical vertebra to the third thoracic vertebra. The procedure at this point is carried to completion on the side to be operated on, before dissection of muscle on the opposite side (the operation is usually bilateral). The incision of fascia and muscle is made through the tendinous attachment of the trapezius, rhomboid, and serratus posterior muscles. The erector spinae group of muscles and the lower end of the splenius cervicis muscle are exposed. The transverse processes of the thoracic vertebrae can be palpated through these muscles. After the spinous process of the first thoracic vertebra and the tip of the transverse process of the first thoracic vertebra have been identified definitely, blunt dissection is made through the erector spinae group of muscles, parallel with the spinous processes. Muscular attachments to the transverse processes are now freed mesially, until the point at which the process fuses with the body and the laminae can be demonstrated. The periosteum of the first rib is incised on its dorsal aspect. This permits exposure of the rib lateral to the transverse process of the vertebra for a distance of 3 cm. The rib is cut at the outer border of this area of exposure, and the transverse process is cut where it joins the body of the vertebra. The pleura and lung are now gently dissected from the lateral side of the vertebra and are retracted anteriorly

and laterally. After exposure of the sympathetic trunk in the posterior mediastinum, the procedure consists in dissection and removal of the ganglions and of the intervening sympathetic trunk, and division of any gray rami that may run laterally from the thoracic ganglions to the first thoracic nerve. After the sympathetic trunk has been divided below, traction is made from above downward, thus exposing the cervicothoracic ganglions sufficiently to divide all of the rami ascending from the ganglions into the cervical region.

SUMMARY

Knowledge of the sympathetic nervous system has been increased by means of anatomic and physiologic studies, not only in the laboratories but also in the operating room and in the clinical wards. The finely balanced mechanism of this intricate nervous system plays a very important part in everyday life, and is being studied from the standpoint of its reaction to the rigors of warfare. Two outstanding clinical conditions, which have been observed in disabled veterans, and which are amenable to operations on the sympathetic nervous system, are conditions of the bladder and vascular diseases of the extremities. Selected cases of atonic and irritative lesions of the urinary bladder respond satisfactorily to resection of the presacral nerves, and the same is true following sympathetic ganglionectomy in selected cases of thromboangiitis obliterans involving the vessels of the extremities. The latter disease is not cured but the patient is definitely improved, and in the majority of cases amputation of digits and of limbs is prevented.

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TUMORS AND ASSOCIATED PROBLEMS

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Introduction

There are three main varieties of malignant tumor: the sarcomata, arising from connective tissues; the carcinomata, arising from epithelial tissues; the endotheliomata, arising from endothelium. It is customary to describe them as cancers.

The sarcomata are tumors with blood-containing spaces between the cells; consequently dissemination usually occurs by way of the blood stream, and the commonest site for the secondary deposits of sarcoma is the lung.

In carcinomata the blood vessels are well formed and the growth invades the small lymph channels by fine tendrils of cancer cells and the first metastases are in the lymph nodes. The frequency and rapidity of metastasis formation varies greatly.

Examples of endothelioma are growths involving proliferation of the endothelium lining blood and lymphatic channels. Since endothelial cells lie next one another without intervening substance, they resemble in this respect epithelial cells but embryologically they are mesodermal and therefore allied to the connective tissue group. The features of endothelial tumors, as expected, resemble now the one, now the other group (3).

There are no hard and fast rules in tumor growth. Virchow knew that sarcoma could originate in the stroma of an epithelial tumor, and Ehrlich showed, when transplanting carcinoma of a mouse into different animals, that the stroma may change so as eventually to replace the carcinoma and finally become sarcomatous (4).

The term "cancer" was formerly used to designate any malignant growth, especially one attended with great pain and ulceration, with cachexia and progressive emaciation. Now the term designates a malignant tumor having its origin in the epithelial tissue and invading any of the surrounding structures (1). It does not refer to a specific disease but to a group of diseases, the members of which are similar in many respects and quite different in others, especially as regards malignancy and the form of treatment. These diseases occur in man, animals, and plants and probably constitute biological reactions to various stimuli or irritants. This view is corroborated by the fact that in every case the disease is local at first. In the majority of cases secondary growths or metastases occur sooner or later in distant parts of the body.

The new growth commences by a proliferation of the existing cells of the epithelial tissues of the body. Very often these proliferating cells form solid columns, enclosed in a framework of connective tissue, and in this form invade the surrounding normal tissue. The cancer cells resemble the cells of the tissue from which the growth arises. The metastases resemble the primary growth in structure.

Locally, the growth extends by multiplication of the cells of which it is composed, destroying at the same time the surrounding tissues by pressure atrophy. Usually it degenerates and breaks down because the blood supply of the tumor is insufficient to supply the needs of the growing cells. The average duration of life in those afflicted with any one of these diseases is 2 to 3 years, the limits being roughly from a few months to about 6 years. Much depends on interference with important organs and structures. If a cancer runs its natural course it will extend until it reaches some free surface. Here, as a result of slight injury or because of local degeneration, putrefactive micro-organisms invade it, degeneration proceeds, and a foul ulcer with thickened edges results. Long before this has occurred, however, in most cases cancer cells from the periphery of the mass have grown along the lymphatics to the nearest group of lymph nodes, and perhaps to further groups, or minute portions of the primary growth have been carried by the blood stream to distant parts, particularly the liver or lungs. Wherever lodged, these transported cells multiply and form secondary growths or metastases. The total mass of metastases often greatly exceeds in size that of the primary mass (2).

Development of a Neoplasm

Consider several tissue cells. Assume that they are temporarily exposed to an injury. Some cells may be completely destroyed immediately by the injury, some will die as a result of injury, others may be so slightly affected that they continue their functions apparently uninjured. The injury will stimulate repair—that is, cells in the vicinity will begin to multiply in order to bridge the gap and produce healing. Repeat the same or similar injury at the same point. Again some cells will die, some survive, the survivors being stimulated into activity in order to bring about repair and healing. Let the injury be repeated over and over and over again. There must be a great many young cells, immature cells produced by attempted repair of previous injury, which if not destroyed by successive injuries, will be stimulated to multiply in order to bring about repair and healing. These cells will in due time have sustained so many injuries insufficient to destroy them that the sublethal injuries have become stimuli for the growth—i. e. multiplication of the cells. The cells have so often readjusted their vital processes to sublethal injuries that experience has endowed them with such proficiency in adjustment to injurious stimuli that they respond by multiplication to injuries, which at some time earlier in their career would have destroyed them. This acquired increased resistance to a certain type of injury enables the cells to compete with normal cells. They have so often responded by multiplication that they have acquired a specialized function and respond to any stimulus by multiplication if that stimulus is not strong enough to destroy them.

However, while well prepared by repeated readjustments to resist a certain injury, such cells are at a disadvantage when exposed to an entirely different type of injury, as compared with normal cells. They will perish when exposed to short X-rays or gamma rays of radium (if not resulting from such sublethal stimuli) where normal cells will survive even a greater injury by the same rays. But if the X-rays or gamma rays are of sublethal intensity for these rapidly multiplying cells they respond by the only way they can to any stimulus—by multiplication, and soon such repeated response endows them with resistance to increasing intensities of radiation—whereas the normal cells, hampered in their functions by repeated radiation, require less and less to be completely destroyed. Repeated inadequate dosage of radiation decreases sensitivity or increases resistance of malignant cells to successive radiations and increases sensitivity of normal cells (7). It is necessary to obtain the therapeutic effect desired from the first application or during the course of a first series of radiations following each other at short intervals (Roussy). Cells which have lost all properties except that of multiplication are also vulnerable when exposed to certain antibodies. For example, of 47 mice suffering

from spontaneously arising cancer, 34 or 74 percent were cured by inoculating refined and concentrated anti-cancer serum (euglobulin) into the tumors (41). In a high proportion of the cured mice (90 percent) it was possible to show that the mice had been rendered immune to cancer as the result of the treatment (41). It still remains to be seen whether autogenous vaccines can be developed of sufficient potency to destroy cancer cells in a living patient.

Biological Variation

Biologically all men are not created equal. Their forebears were subjected to various diseases, poisons, foods, or lack of foods, environments, privations, and stresses. It is common observation that the offspring of a diseased parent or a starving parent is not healthy and robust. It is also common observation that the effect of acute diseases, of injuries, and of environment upon the healthy and robust is not the same as upon the weak and sickly.

The pattern of response of the cells of the body to an injury, although similar, is not the same in any two individuals any more than the pattern of the ridges on their finger tips. In the animal kingdom, individuals can be produced under experimental conditions, whose tissue cells respond to every injury by aimless multiplication, i. e. neoplastic proliferation (21). In the human species individuals arise from interplay of factors of disease, malnutrition, starvation, overeating, overfatigue, environment, etc., throughout the course of several generations whose tissue cells respond to injury and especially repeated injury by aimless multiplication, i. e. neoplastic proliferation.

The cells have so frequently throughout several generations struggled for existence against adverse factors that one by one they have lost their various specialized properties until finally only the property of self reproduction remains. Such cells can respond to any stimulus only in one way, namely by reproduction. When they can no longer do that the line of individuals disappears from existence.

In the human species the individuals may be considered arranged in a series. At one end of the series are those whose tissue cells possess all the properties which enable them to respond to all stimuli innocuously and in a manner beneficial to the whole organism. At the other end of the series are those individuals whose tissue cells have lost all properties except the last one, that of self-perpetuation. In between these two extremes are individuals whose tissue cells possess properties enabling them to respond to stimuli in varying gradations. In those individuals fitting in near the lower end of the series minimal weakening by disease, environmental stresses, malnutrition, etc., will produce a state such that the slightest injury will produce neoplastic proliferation; as the individuals fit into the ascending series more and more repetition of an injury is necessary to

produce neoplastic proliferation under the same conditions of general poor health until the maximum repetition of an injury is reached in those who fit at the head of the series.

Effects of Disease and Age

In any one individual it is very unlikely that tissue cells respond to an injury exactly the same way at all times. On the contrary, experience indicates that they do not. In periods when the individual is suffering from a disease his tissue cells do not respond to stimuli injurious or innocuous as in periods of health. Every serious illness damages the individual. Tissue cells of an individual weakened by anemia or by toxins of a disease, or whose vitality has been sapped, be it by syphilis, tuberculosis, or any other infection, or bombarded by drugs, such as arsenic or mercury, or whose tissue cells have been subjected to unbalanced diet, to dietary deficiencies, to excessive carbohydrate or protein intake, do not respond to an injury with the vigor of a healthy cell. The summation of all the little stresses of a lifetime as age advances handicaps more and more the tissue cells in their response to injurious stimuli and occasionally perhaps even to functional normal stimuli. So it might be said that one by one the normal healthy properties of tissue cells are lost until only one remains—the last and most primitive of all the properties of a unicellular cell—the property of reproduction. Injuries evoke only this response—the cells can do only one thing and that is to reproduce themselves and when cells so respond to an injury or stimulus the result is neoplastic proliferation.

To prevent neoplastic proliferation we cannot control the past of our forebears or ourselves. We can avoid injuries, repeated irritation, and diseases often, and we can control our food and hygiene.

Types of Injury

Tissue cells may be injured by any one of numerous agents. Among such injurious agents whose effect on tissue cells has produced malignancy are: Tar, creosote, anthracene, pitch, soot, shale oil, petroleum, arsenic, aniline dyes, radium, Roentgen rays (22), mechanical trauma, extremes of heat and cold, parasitic irritants, such as Bilharzia (or distomiasis leading to primary cancer of liver in cows), spiroptera in gastric cancer of rats, trichinae in cancerous tongues, nematode worm in center of sarcoma and adenocarcinoma of rats, sarcomas growing around cysticerci, acari in lymphosarcoma of dogs, acari in tumor-cell nets of epitheliomas of face in man, acari and Demodex folliculorum in nipple in some cases of Paget's disease and cancer of breast, and worms in mouse tumors (these probably act through irritants which they secrete) (23). Syphilis has certain unusual capacity to excite

neoplastic growth (23). Tuberculosis is also closely linked with neoplastic development. "Sequels of both syphilis and tuberculosis have a definite relation to tumors, which reveals the microorganisms as indirect cancer parasites." The resulting tumor arises from the natural momentum of the disturbance originally excited by the parasite (23). Even the momentum of inflammatory processes may lead to tumor growth, especially in rats (23).

The injury produced by chemical products of stagnating milk following ligation of ducts produced cancer in mice (24).

Gastric carcinoma of rats was found to be dependent on presence of nematode worm (introduced by feeding rats with infested cockroaches). The epithelium gradually assumed power of independent growth apart from the irritant originally exciting its proliferation (23) (p. 143).

The irritation of retained secretions under prepuce in cases of phimosis has produced carcinoma (25). The irritation of tobacco, betel nut, jagged teeth, ill-fitting dentures have produced intra-oral carcinoma. The bolting of food and food too hot or too cold often are considered factors in cancer of stomach and esophagus (25).

Lacerations of cervix and collection of decomposing material in cervical canal are considered to be factors in cancer of cervix (25).

Prevention

It is generally conceded that the earlier a patient with cancer is properly treated, the more likely he is to recover from his disease. The disease being a local one, and early excision or destruction of the lesion constitutes a cure. If early treatment results in cure it does not appear unreasonable to expect even better results if more attention is focused on prevention of cancer.

It requires no proof to convince anyone that if he never acts as a chimney-sweep he will not develop the chimney-sweep's cancer, if he never straps a metal box containing charcoal to his waist he will not develop cancer at the site. It should require no proof to convince people that a sharp tooth, an ill-fitting denture, repeated injury by partial burn of hot pipe-stem or short cigarette stub may in some people bring about the development of cancer. Those who during their own life span or during that of their immediate ancestors have weathered the storms of disease, be it syphilis, tuberculosis or other, ought especially to guard against all and every form of injury, especially repeated injuries. Even the irritating smoke of cigar, cigarette, or pipe may prove an adequate cancer-producing stimulus in those whose tissue cells are not perfectly normal, robust, and healthy. Even the healthiest of men cannot afford to bolt down food, swallow drinks so hot or so cold that they cannot hold them in their mouths. Highly spiced food can be taken with impunity by some but it is

hardly wise. Certain diets, even in the healthy, are followed, often day after day, by sour stomach and heartburn. All these are repeated injuries to esophagus and stomach. How many of those who indulge in these habits will escape the penalty which repeated irritations or repeated injuries produce, cannot be guessed without knowing something of the make-up of the individual.

The irritation and injury produced by decomposing bodily secretions which are not removed, may lead to development of cancer. This is true of stagnating milk in the breast, of decomposing secretions of genital tract, of retained secretions under a prepuce in cases of phimosis. To those who want a formula by means of which to avoid cancer, it might be said: Avoid every injury, every infestation with parasites, every infection; be moderate in all things; maintain the body free from all decomposing materials; and make the slightest lesion a matter of prime importance until it heals.

Cellular Differentiation

The degree of anaplasia is the degree to which tumor cells have deviated in form from the mother cells from which they arose. Tumors are graded on the basis of relative degrees of cellular differentiation. This grading, which varies in different clinics because each group of workers has its own conception of what is meant by a certain grade, is, nevertheless, of considerable practical importance.

In general, the degree of radiosensitivity runs parallel to the degree of anaplasia of tumor cells (5). Radiation is less effective in the fully differentiated tumors and such are more suitable for surgical removal. The less differentiated the tumors, the more radiosensitive they are, and melt away rapidly under external radiation. However, this type of tumor, showing the minimal cellular differentiation, metastasizes early, widely, and usually through both lymphatics and the blood stream. Consequently, while the local process may be readily controlled, the tendency to generalization is such that the percentage of ultimate curability is very low. Real curability is greater in the more radioresistant types (6).

Knowing these individual differences, the doctor "will not attempt to destroy squamous carcinoma or neurogenic sarcoma with half erythema doses of external radiation, nor will he insert radium seeds into lymphosarcoma or lymphoepithelioma" (Ewing).

The order of radiosensitivity increases in the following order (Ewing):

1. Squamous carcinoma, malignancy of varying grades, radioresistant.
2. Transitional-cell carcinoma, malignant (highly), radiosensitive.
3. Schneiderian carcinoma, malignant (highly) (subvariety of transitional-cell carcinoma), radiosensitive.

4. Basal-cell carcinoma and adenoid cystic carcinoma, malignancy low, radiosensitive.

5. Lymphoepithelioma, malignancy high. Very radiosensitive (5).

The metastatic processes show a tendency toward cellular differentiation and are for that reason more radioresistant than the parent growth (6). Also, following repeated radiations, the cellular elements of cancers become less and less sensitive to successive radiations; while healthy tissues become more sensitive. This phenomenon, first noted by Pierre Delbet, was called by him "vaccination" against radiation. For this reason it is necessary to obtain the therapeutic effect desired from the first application or during the course of a first series of radiations (7). The degree of cellular differentiation cannot be known without a biopsy.

Biopsy

While a biopsy is essential, it is not free from risk. Cases have been made hopelessly inoperable by several biopsies on same patient—all giving positive cancer reports (8). Instead of making another biopsy, one of the original microscopic slides should be examined. In referring patients suffering from malignancy, any microscopic slide of the case or biopsy specimen should be sent with the patient to avoid delay in instituting treatment. There should never be a necessity for correspondence and requests. The biopsy remains property of the patient and refusal to forward it on grounds of it being the only section the hospital has, or because it is a rare museum specimen, will sooner or later meet with its deserts at the hands of the public. Repeated biopsies still continue to be necessary, due to delays of securing original biopsy specimen or obstinate reiteration of a description of the microscopic slide instead of forwarding the actual slide or biopsy specimen. Yet the treatment cannot be directed as effectively as when the cellular structure of the lesion is studied and the tumor graded.

Considering the dangers associated with biopsy it may be pointed out that forcible manipulation of a malignant growth during a clinical examination, especially repeated manipulations by a succession of physicians is far more dangerous than a simple clean incision; or preferably excision if at all practicable (9). In breast clinics, cases with ecchymosis due to previous examinations present themselves, living examples of a zeal which ignores the fundamental precept of a true physician "to do no harm."

There is no need for a biopsy in the breast. The growth should be removed by wide excision (9). The examination of the gross specimen will in a great number of cases establish diagnosis of malignancy if present. The material for frozen section is selected by the

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unaided eye and palpation. Frozen sections, unless unquestionable positive evidences of cancer are found are not dependable (8). Doubtful or negative reports must be verified by examination of paraffin-prepared sections as soon as practicable.

One of the surprises of cancer experience has been the lack of caution in attempting to make a diagnosis from unsatisfactory material. It is also a wise rule to examine microscopically all excised new growths no matter how small or benign in appearance (8).

Biopsy material removed by high frequency spark is so damaged by heat that diagnosis is difficult (9). A compromise plan is to use the knife to obtain the specimen and then to lightly cauterize the resultant surface to destroy loose cancer cells and seal the vessel openings. If the tumor is small, remove it entirely; if that is not practicable, take a fair-sized section from the edge, comprising a piece of the tumor and some of the adjacent tissue (8). Give the pathologist a diagram of the lesion, showing the site from which the piece was taken and if of assistance, indicate on diagram of removed segment in what plane sections should be cut. Remove secretion or blood from specimen at time of biopsy and send specimen to laboratory in formalin (8). In making a biopsy, strict asepsis is necessary and if possible the biopsy should be done at time of operation and the material immediately examined. Risk will thus be greatly diminished (9).

A careful study of enlarged lymph node distribution should be made and preparation should be such that exposure can be quite complete in order that, if necessary, several nodes may be removed. A fragment from a node should never be excised (9).

In all cases where a pathologist is called upon to make a diagnosis on surgical material on which the life of the patient may be put in jeopardy by an incorrect opinion, he should be present at the operation in order that he may be familiar with the region from which the tumor is removed and with its growth, aspect and connections (9).

Professor Roussy of Paris insists that biopsy must be taken by the same person who will view the section and make the diagnosis or at least in his presence. Many gross errors repeatedly result from the neglect of above principles (7).

As to the use of X-ray prior to biopsy, a justifiable dose of heavy X-ray exposures as preliminary measure cannot be expected to devitalize cancer cells (9).

Aspiration biopsy.—When the nature of a tumor must be ascertained, a useful method at times is the aspiration biopsy. The technique is as follows:

Anesthetize with a drop or two of novocaine. Nick skin with knife. Insert an 18-gage needle connected to aluer syringe and push needle into mass. Pull out plunger of syringe to create suction. Any cells

detached will enter needle as it is pushed forward, while suction is maintained. Change direction of needle once or twice. This will cut off column of cells within lumen of needle. Let plunger go lightly to equalize pressure. Withdraw needle and expel contents of needle on a clean slide. Use wire obturator in needle to expel contents if necessary. Spread smear by aid of another slide, pressing slides very firmly together—there is no danger of crushing cells. Dry over flame, fix and stain in 2 or 3 minutes. Examine under a microscope (10).

Punch biopsy.—Another means of obtaining a specimen from a tumor at some depth is the Hoffman punch (11). However, biopsies should be done only when treatment either by surgery or radiation can be instituted without delay.

Radiation therapeutic test.—The presence or absence of tumor tissue and its probable nature may also be determined without biopsy by means of a therapeutic test by radiation (5).

Primary Tumors of Bones

In order of sensitivity to radiation the primary tumors of bone are:

(1) Endothelial myeloma or Ewing's tumor.
(2) Giant-cell tumor.
(3) Multiple myeloma.
(4) Osteogenic sarcoma (36).

In bone tumors, biopsy only occasionally supplies knowledge which may not be obtained through other procedures and is not done routinely. It is always performed in the small group in which correct diagnosis cannot be made otherwise and should be performed only by the surgeon who is to have subsequent care of the patient and is prepared to carry out the best method of treatment.

If biopsy reveals endothelial myeloma, the treatment should be by Coley's toxins and radiation. If marked improvement does not occur at the end of 6 or 8 weeks, then amputation or resection, followed by prolonged prophylactic toxin (Coley's) treatment should be seriously considered. Further delay may result in metastases (36).

If biopsy reveals osteogenic sarcoma, especially with considerable new bone formation, the surgeon is dealing with a tumor highly resistant to Coley's toxins and radiation. From long and large experience it is known that preliminary radiation in such cases does not improve the prognosis of the later amputation. Immediate amputation is necessary (36).

In giant-cell tumors of bones, good results are obtained by X-radiation. A test dose is first applied and since these tumors are comparatively radiosensitive, massive high-voltage doses are seldom required. Treatment is controlled by repeated examinations of patient and periodic roentgenographs (37).

Another method of treatment is the surgical extensive curettage of giant-cell tumor of bone, swabbing out the cavity with zinc chloride or phenol, followed by a 2- or 3-months' period of Coley's toxin treatment (38).

Multiple myeloma is considered fatal. Four cases have been reported in which patients remained well long enough to justify a hope of permanent cure. The treatment is by Coley's toxins (i. e. toxins of erysipelas and B. prodigiosus) and by radiation (39).

The principles of treatment of primary bone tumors with pathological fracture are: (40).

In endothelial myeloma—immobilize the part. Use radiation—radium or high voltage X-rays to limit of skin tolerance. Later, if extremity is useless, may amputate (40).

In osteogenic sarcoma, amputate immediately above site of fracture if no pulmonary metastases exist. Give Coley's toxins intramuscularly and later intravenously as prophylactic measure (40).

Immobilization and X-radiation will unite fractures in the case of giant-cell tumors in most cases. Bivalved circular plaster cast will immobilize and is removable for X-ray treatments. Later observation will show any indication for surgery (40).

[To be continued]

CHORIO-EPITHELIOMA: SHOULD SERUM FROM THE FEMALE IN THE PUERPERIUM AND PREGNANCY BE GIVEN A THERAPEUTIC TRIAL?[1]

By James G. Dickson, Leutenant Commander, Medical Corps, United States Navy.

Chorio-epithelioma apparently presents a problem unlike that of any other malignant neoplasm. MacCallum (1) says that the "disease seems to offer an extremely interesting border-line condition * * *." The biological reactions of pregnancy indicated by gynecomastia (at times with secretion of colostrum) and the Aschheim-Zondek reaction are striking. Chorio-epithelioma of pregnancy, though one of the most malignant neoplasms known, undergoes spontaneous regression probably more frequently than any other tumor and at times there is extreme difficulty in determining the border line of malignancy. Kaufmann states that it is "permissible to assume that deported harmless materials (cells, villi) may become malignant at the situation where they are stopped * * *." He refers to cases of chorio-epitheliomatosis in which it was not possible to demonstrate the primary tumor and concludes that "The biological conditions, and not the histological pictures are therefore important in the true character of this tumor * * *" (2).

[1] Received for publication Oct. 12, 1934.

For years there has existed a belief that malignant neoplasia is analogous to the growth of embryonic tissue. It has not been stressed, however, that we have in chorio-epithelioma a well known tumor which is actually composed of embryonic tissue from one individual growing in the body of another.

Normal chorionic epithelium has properties which in any other situation would signify malignancy. It invades the wall of the uterus, erodes its blood vessels and at times forms emboli in the maternal blood stream. These features support the embryonic theory of tumor genesis. They have been well described elsewhere:

> Normal chorionic syncytium and cancer cells have much in common: extraordinary reproductive energy, rapidity of growth, invasive power coupled with destruction of the recipient tissue, and striking facility for hematogenous dissemination. When chorionic syncytial giant cells lodge in the lung, as they do with every childbirth, they soon die; but when carcinoma cell emboli lodge in the lung, they survive and grow only too frequently. One succumbs, apparently, to an antisyncytial hormone; against the other no such defensive mechanism seems to be at hand (3).

Chorio-epithelioma as a complication of pregnancy is rare. Its incidence cannot be given. Ewing (4) states that Pollasson and Violet collected 455 cases up to 1914. Considering the enormous number of pregnancies of the world, its relative incidence must be exceedingly small, possibly less than one in a million pregnancies.

The development of chorio-epithelioma in ovarian teratomas is so rare that its very existence may be questioned. Ewing (5) states, "While chorioma testis is comparatively frequent, the ovary has furnished only rare or uncertain examples of this type of atypical teratomas." He cites only four cases and admits that these are largely questionable. MacCallum (6) says that Risel accepts only one case, that of Pick. Two other possible cases have been recently reported (7, 8). It is very doubtful if either of these will be accepted by the authorities of the world.

Rare as is chorio-epithelioma as a development in teratomas of the ovary it may be relatively more frequent than is chorio-epithelioma as a development in pregnancy.

The conception that at least part of the group of teratomas arise from totipotent cells which have the inherent capacity to originate all the tissues of a new individual appears logical. It is strongly supported by increasing reports of the Aschheim-Zondek reaction in teratoma testis. This appears to indicate a close biological kinship between such processes and normal pregnancy. In both ovarian and testicular teratomas, chorionic epithelium, being formed early in the development of the embryo, apparently should be a frequent finding. Ewing (9) states that an important principle of the growth of teratomas is the tendency of one element to overgrow and suppress

the others. In teratoma of the testis, chorionic epithelium appears frequently to do this; in teratoma of the ovary it is almost unknown.

Why the rarity of chorio-epithelioma in pregnancy and in teratoma ovarii? The conception that chorionic epithelium per se is malignant seems reasonable. It is not unreasonable to postulate that, having been exposed normally through the ages to the possibility of malignant growth of chorionic epithelium, woman possesses an inherent capacity to combat such a development; that given the presence of this epithelium, the absence or occurrence of malignancy is determined solely by the presence or absence of a capacity for defense by the host.

So far as is known chorionic epithelium in the male is invariably malignant. It has not been described as a benign constituent of teratomas in that sex. Ewing (9) says that the adult tissues of teratomas give rise to benign growths and the embryonic tissues originate malignant growths. Obviously, the adult tissues develop from the embryonic. In the development of a teratoma from a totipotent cell, analogous to the development of the embryo, the embryonic tissues ordinarily should give rise to adult types. This cannot occur in the case of chorionic epithelium for it is a pure embryonic tissue and not destined to originate any adult type. Normally its destiny is to perform a definite function during gestation during which it is under control. It then dies. In the male its destiny is thwarted. Control is lacking. It exhibits, however, its physiological properties. The result apparently invariably is a malignant neoplasm. So we may say of chorionic epithelium: Physiologic and very rarely malignant in the female; accidental and invariably malignant in the male.

The idea of a hormone or antibody control of normal chorionic epithelium is not new. MacCallum (10) says that the complete disappearance of definite chorionic epitheliomata led Fleischmann (1905) to speculate as to

> "Whether there is some substance formed in the maternal blood at the end of pregnancy, which, like the experimentally produced syncytiolysin of Scholten and Veit, has the function of destroying the syncytial elements which remain buried in the uterine wall or lodge in distant organs. The failure of this substance might allow the unchecked development of the tissue into a destructive tumor, while its late formation might account for the disappearance of the tumor * * *.

Kaufmann (11) states that in pregnancy normal deported cells and villi are destroyed probably by cytolysins in the sense of Ehrlich and that it is believed that when such antibodies are wanting, the circumstances favor a rapid overgrowth or the formation of a malignant chorion epithelioma. He quotes Schmauch as believing that successful therapy of this tumor may be practiced by immunization against its specific cells.

Bell (12) in his "working hypothesis" of the genesis of malignancy has conceived that normal chorionic epithelium is controlled by some substance elaborated by the fetus. If chorio-epithelioma in the male is biologically identical with that of pregnancy, and it is generally conceded that it is, his theory is unacceptable in that it fails to explain the much greater frequency of chorio-epithelioma in teratomas in the male as compared with the female.

To the conception of a maternal control of chorionic epithelium might be raised the objection of the infrequency of chorio-epithelioma in teratomas in the male. What this incidence is cannot be given, as figures showing the incidence of teratomas are not available. However, it is not common as a reported finding. Heidrich, Fels, and Mathias (13) in a review of the universal literature in 1930 collected 140 cases of chorio-epithelioma in the male. They credited nine of these as being of extragenital origin. The genesis of teratomas is unknown. It is possible that only part of them originate from cells capable of giving rise to chorionic epithelium and that chorio-epithelioma develops in every teratoma in the male which comes from cells with this capacity. As has been pointed out (3) it would require serial sections of the entire growth to rule it out. The morphological picture of undifferentiated chorionic epithelium is not sufficiently characteristic to make the diagnosis infallible. If it be shown, as appears reasonable, that the Aschheim-Zondek pregnancy reaction may be obtained only in the presence of chorionic epithelium, this test may prove a great aid in making such diagnoses. For surely the biological reactions of a tissue must be more specific than any histological picture.

If the conception of a maternal hormone or antibody control of chorionic epithelium is correct, serum from the female in the puerperium and possibly also in the latter part of pregnancy when administered to one suffering with chorio-epithelioma might exert a retarding influence on the process. It is offered with the hope that others more fortunately situated as to clinical material will give it a trial.

Therefore, it is recommended that selected hopeless cases of chorio-epithelioma be treated by the intravenous administration of large doses of serum from the human female at various stages of the puerperium and the later stages of pregnancy.

If the reaction should be favorable, the possibility of the use of serum from one of the lower animals, such as the mare, should be investigated.

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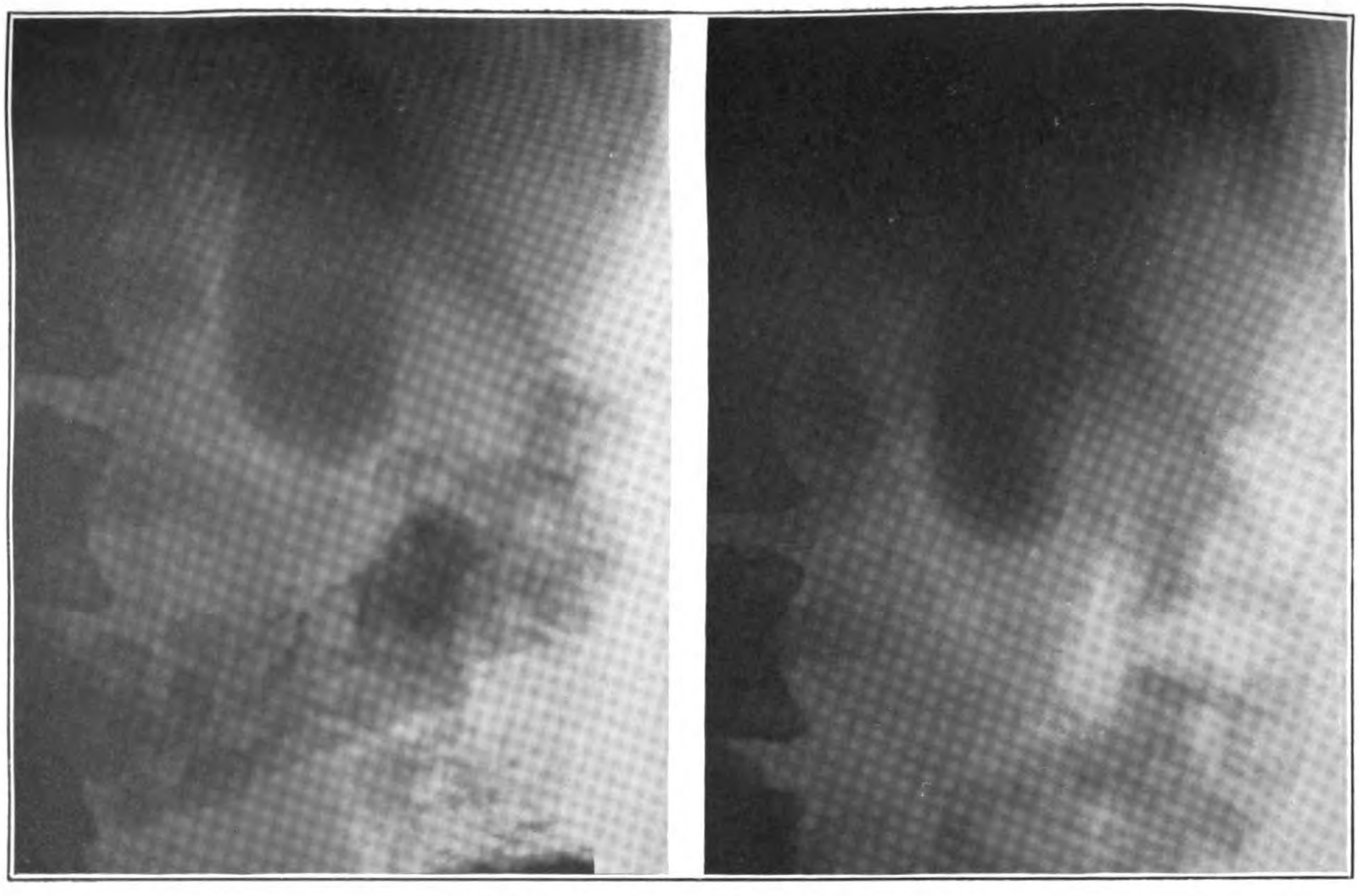
DOUBLE ORAL ADMINISTRATION OF DYE FOR CHOLECYSTOGRAPHY

By I. W. Jacobs, Commander, Medical Corps, United States Navy

As the oral method of dye administration for cholecystography has practically displaced the intravenous injection of sodium tetra-iodophenolphthalein, it seems appropriate to describe a double oral administration of the chemical which has given satisfactory results at the United States Naval Hospital, Brooklyn, N. Y., during the last 9 months.

Heretofore, the usual oral dose of dye was used with average results, necessitating repeat examinations at intervals of 1 month to 6 weeks in those cases where the gall bladder failed to visualize, or the giving of another dose of dye the following day prior to the completion of the test. This was never quite as satisfactory as the intravenous method; consequently, radiologists and surgeons began to doubt the specific value of this procedure.

After visiting various hospitals in New York and consulting their roentgenologists, it was the impression of the writer that there was little uniformity in this work. Naturally, results differed, depending upon the technique used, for one radiologist would report positive findings, where another would find a similar gall bladder shadow normal.



NORMAL FUNCTIONING GALL BLADDER.

1. Visualization at 20 hours.

2. After ingestion of a fatty meal.

Another impression was formed during my hospital contact which is worth noting. As most of these examinations were made in out-patient cases, the usual procedure was to give the patient a list of directions and the dye prior to the taking of the X-rays. To a certain degree Navy patients are similarly prepared, except that the nurses and corpsmen supervise the giving of the dye and the neccessary diets. It is believed that due to this laxity in the preparation of the patients, some of the poor results are obtained and that by a more thorough attention to specific details, the usefulness of this test is enhanced. Therefore, an outline of the method used in this hospital may be helpful in getting uniform results in other institutions.

1. The drug used here is Shadacol made by the Davies, Rose & Co., Boston, Mass., which can now be obtained from the supply depots of the United States Navy. After running a series of cases and comparing it with other preparations, it is believed that Shadacol gives very uniform results in the average case.

2. Great attention is devoted to the preparation of the patient. As this is an X-ray problem the administration of dye is done by members of the Radiological Service, only requiring cooperation of the nurses and corpsmen in the carrying out of some of the routine, such as the giving of diets and getting the patients to the X-ray department on time for their pictures.

3. A list of printed instructions are sent to the wards and orders are written in the nurse's order book by members of the X-ray department signed by the X-ray officer. The list of instructions are as follows:

(a) At noon on the date of admission the patient has a regular hospital diet.

(b) Immediately afterwards, 1½ drams of camphorated tincture of opium is given.

(c) Five minutes later a regular dose (one bottle of dye) is given in one-half glass of cold water. (Stir dye until it turns a milky white, stirring about 2 to 3 minutes.) Nothing by mouth is given until 6 p. m.

(d) At 5:45 p. m. an X-ray film is taken in order to diagnose cholesterol shadows which may be missed if this is not done.

(e) At 6 p. m. the patient has a carbohydrate meal of one-half grapefruit with sugar, tea and sugar, and two crackers. Immediately after this meal another regular dose of the dye is given, after which nothing is taken by mouth until so instructed by the X-ray department.

(f) The patient reports to the X-ray department at 8 o'clock the following day for the second X-ray film.

(g) The patient then receives a specially prepared fatty meal consisting of one glass of milk and cream, two eggs, toast and butter at 8:15 a. m. and then a series of 3 to 4 X-ray films are taken at intervals of 45 minutes.

Examples of this test are given below showing first a normal functioning gall bladder and another pathological one revealing a solitary gall stone.

Two pictures, each series, showing (1) Visualization of gall bladder at 20 hours and (2) Films after ingestion of a fatty meal.

METHYLENE BLUE AND OTHER AGENTS AS ANTIDOTES IN HYDROCYANIC ACID AND CARBON MONOXIDE POISONING

By George F. Cooper, Lieutenant, Medical Corps, United States Navy

In view of the controversial nature of various articles which have appeared in medical and industrial publications, in recent months, in regard to the value of methylene blue and other agents, in poisoning from the cyanides and from carbon monoxide, it is believed that a summary of the present status of such treatment is timely.

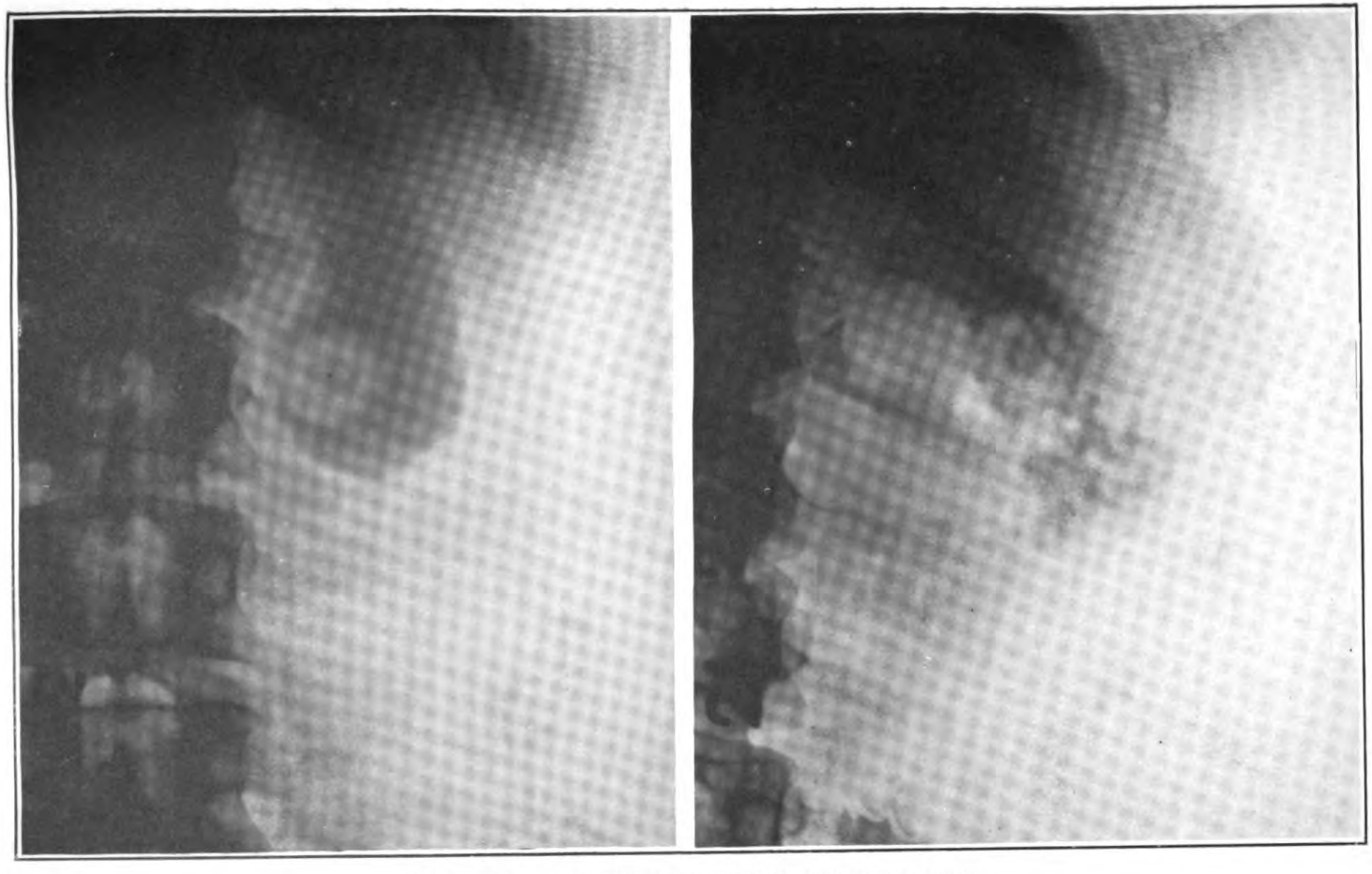
Recently, while talking to members of one of the classes at the Naval Medical School, it was found that most of the members of the class were under the impression that methylene blue is as efficacious in the treatment of carbon monoxide intoxication, as it is in that produced by the cyanides. Possibly, this erroneous belief is shared by other members of the Medical Corps.

Methylene blue (Methylthionine hydrochloride) is a dye, and has been advocated in medicine for many conditions, such as local application in diphtheria and tonsillitis, and internally in such conditions as malaria and neuralgia, and in the treatment of gonorrhea. Its common use, until recently, has been that of a laboratory dye, but since the report of an almost miraculous recovery from cyanide poisoning, due to its use intravenously, it has been suggested as an antidote for many poisons, usually without scientific basis for such suggestions.

The writer has reviewed the literature on the subject of cyanide and carbon monoxide poisoning, from 1933 to date, since most of the newer theories have developed during that period, and will attempt to present, in this article, the latest available data, and the most recent ideas and remedies that have been suggested for use in poisoning from hydrocyanic acid, and from carbon monoxide.

The first definite statement that the writer wishes to make, is to quote from the excellent paper of Haggard and Greenberg (1), in which they make this concluding statement:

> There is no valid basis, theoretical, experimental, or clinical, for the belief that methylene blue is an antidote for carbon monoxide asphyxia.



PATHOLOGICAL GALL BLADDER SHOWING A STONE.

1. Visualization at 20 hours.

2. After ingestion of a fatty meal.

Eddy (2) has shown the definite antagonism between methylene blue and sodium cyanide, and has demonstrated the power of methylene blue to decrease respiratory depth and rate which has been increased by the cyanide. He has shown, also, that the methylene blue alone will stimulate respiratory activity in rabbits and dogs, and this may be the manner in which apparently favorable results have been accomplished in cases of carbon monoxide intoxication. However, no comparable figures are available to show that it is of more value than inhalations of carbon dioxide as a respiratory stimulant and it is certainly more dangerous to use.

Eddy studied the effects on anesthetized dogs, of response of submaxillary gland to agents which affect oxidations and acid base equilibrium.

Salivary secretion stimulated by continuous administration of pilocarpine, submaxillary blood volume flow, blood pressure, pulmonary ventilation and rate of oxygen consumption, were recorded simultaneously.

It was noted that repeated doses at 10- to 30-minute intervals, of sodium cyanide, 0.5 cc per kilogram of M/100 solution, produced like effects—stimulation of respiration followed by depression, a rise and then a fall in blood pressure, increase in submaxillary blood volume flow, and slowing of secretion.

The same dose of sodium cyanide given 10 minutes after the administration of methylene blue, 1 cc per kilogram of 1 percent solution, usually had no effect on any of these functions, or at most produced only slight respiratory stimulation. Given 45 minutes after the methylene blue, the sodium cyanide effects reappeared but were still less than before administration of the dye.

He studied the effects of M/100 solution at a rate of 5 cc per minute and found that if a small dose of methylene blue was given during the injection of sodium cyanide, the respiration was decreased in rate and depth, though by itself the methylene blue in the same dosage produced persistent stimulation of respiration.

He states in his summary that methylene blue either abolished or reduced markedly the apparent effects of a single small dose of sodium cyanide upon respiration, blood pressure, salivary secretion, and submaxillary blood volume flow. The antagonism persisted in part for more than 45 minutes.

Methylene blue alone produced persistent respiratory stimulation. It stimulated respiration that was depressed by sodium cyanide to a point enabling the animal to survive an otherwise fatal dose of cyanide. It reduced respiratory activity when that was increased by previous cyanide administration.

Hanzlik (3) has reviewed the literature on the subject, up to 1933, and the reader is referred to his paper for the earlier history of meth-

ylene blue in the treatment of poisoning. He concludes that the evidence shows that there is definite antagonism between methylene blue and cyanide, and that this definite antagonistic property was demonstrated as early as 1926.

The original theories of the mode of action of both hydrogen cyanide and carbon monoxide were based on the action of these substances on isolated tissue, and in some instances on animals not of the same type as man or other red blooded animals. The original supposition that methylene blue might be of value in carbon monoxide poisoning was based on the theory that carbon monoxide acted in the same manner as hydrocyanic acid, in interfering with the oxidative ferment of the tissues. Warburg, as quoted by Haggard and Greenberg, has shown that even small amounts of hydrocyanic acid will do this, and prevent tissue utilization of oxygen. He has shown, also, that carbon monoxide may combine with the tissue ferment and inhibit its action, but not in the concentrations which are immediately fatal to men and animals.

Mechanism of carbon monoxide poisoning.—Since carbon monoxide unites with hemoglobin to form methemoglobin, and thus prevents the carriage of oxygen to the tissues, the asphyxiation in carbon monoxide poisoning is not because of inability of the tissues to utilize oxygen, but because of deficiency of oxygen in the blood. Methylene blue does not supply oxygen.

Then, since methemoglobin cannot carry oxygen to the tissues, the conversion of more oxyhemoglobin into methemoglobin, by the injection of methylene blue, only serves to remove that much more oxygen-carrying power from the blood, and, instead of preventing anoxemia, actually promotes such a condition. Carbon monoxide has an affinity for hemoglobin many times that of oxygen.

Action of methylene blue in cyanide poisoning.—The mode of action of methylene blue, in cyanide poisoning of the tissues, is not primarily on the oxidative ferment, but as follows: The methylene blue, when injected intravenously, reacts with the hemoglobin to form methemoglobin. Cyanide will combine with methemoglobin to form cyanmethemoglobin (which is very stable) and thus reduce the amount of cyanide free to act as a poison on the tissue ferment.

In other words, it bridges the gap, so that, by removing the free cyanide and combining it into cyanmethemoglobin, the cells and ferment of the tissues have an opportunity to recover their functions. The fate of the cyanmethemoglobin is problematical. It may release the cyanide, very slowly, in amounts which the body is capable of detoxifying, or eliminating, and the methemoglobin, by reduction, may be reconverted into oxyhemoglobin, although it is known that methemoglobin remains in the blood for a long time after its formation, and it has been found by spectroscopic examination as long as 3 weeks after formation.

Wendel (4) quotes the works of Brooks, Geiger, Hanzlik, Eddy, Hug, and others, individually, and points out some pertinent facts in support of the method of action of methylene blue in cyanide poisoning outlined above. He shows by reference to the work done by himself, Warburg, Combemale, Shaffer, Kubowitz, Christian, and others, that the dye does not act, in overcoming cyanide poisoning, by providing a substitute for the normal cellular catalysts of respiration which are inactivated by the cyanide, but that methylene blue catalytically converts hemoglobin into methemoglobin, and that the methemoglobin forms, with hydrocyanic acid, an unusually stable compound, cyanmethemoglobin. The union of the cyanide with the methemoglobin is so firm that it can be removed with difficulty, if at all, by a vacuum.

Wendel (5) reasons that, since methemoglobin may be used as an effective agent for absorbing hydrocyanic acid from exceedingly dilute solutions of gases, the formation of methemoglobin by the injected methylene blue leads to fixation of the hydrocyanic acid, as cyanmethemoglobin, within the blood cells, whether the HCN enters the blood stream by absorption from the lungs, alimentary tract, or other tissues. He states that, if the above explanation is correct, like or better protection should be afforded by injecting methemoglobin alone, or by other agents which form methemoglobin more rapidly.

Hug (6, 7) records the same findings as Wendel, and comes to the same conclusions, so that a summary of the work of both can be given in abstract.

They found that the injection, into dogs, of the cells from 20 cubic centimeters of blood, per kilogram of body weight, the hemoglobin of which had been converted into methemoglobin by amyl nitrite (the excess nitrite being removed) effectively protected the animals from the toxic effects of a lethal dose of hydrocyanic acid. They found, also, that the same volume of such cells would revive animals approaching death from lethal doses of HCN.

Hug found that sodium nitrite (which is known to form methemoglobin) to be much more effective than methemoglobin in protecting from cyanides. He found, also, that certain other agents such as pyrogall, pyrocatecol, and phenylhydrazine have a similar effect.

Hug used dilute HCN intravenously, injected slowly and continuously—and found the following to be true. Rabbits are able to detoxify and survive HCN injected at the rate of 1 mg/kg of body weight per hour—continuously. More rapid administration kills.

If sodium thiosulphate 1 gram per kg has been previously injected at the proper time the animals can survive much greater quantities of HCN. Results with dogs were similar.

He found that when HCN was injected continuously into dogs at the rate of 1 mg/kg, per minute, they died after about 16 minutes.

The previous injection of methylene blue or sodium nitrite (5–10 mg/kg body weight) increased their survival time threefold or more.

Sodium sulphide and colloidal sulphur had no antidote effect whatever.

Since the amount of protection that can be gained in this manner depends upon the amount of hemoglobin that the animal can spare for conversion into methemoglobin, Hug found that not more than one-half to two-thirds of the hemoglobin can be spared, without leading to asphyxia, and he estimated that about four lethal doses of hydrocyanic acid would inactivate about 60 percent of the hemoglobin. This was the maximum amount for which he found the nitrite to give protection (four lethal doses of HCN).

Trautman (8), in studying the effect of methylene blue in the treatment of animals exposed to inhalations of hydrocyanic acid gas, came to the conclusion that the treatment was valueless for animals absorbing the gas by inhalation. However, since the human nervous system, and the human reaction to this gas may vary considerably from that of rats and guinea pigs, his evidence is not conclusive enough to preclude the use of methylene blue in human beings poisoned by hydrocyanic acid gas, even through inhalations. Further work on that phase of the subject should be carried on.

It should be understood, however, that hydrocyanic acid gas is what might be called an "all, or none" gas. That is, if sufficient of the gas is inhaled to cause death, nothing is of avail, and if a sublethal amount is inhaled, fresh air and artificial respiration will cause immediate recovery.

Death from the inhalation of hydrocyanic acid gas is so rapid that it is doubtful if any treatment other than artifical respiration is of value, and that, only in the border line case, in which sufficient gas was inhaled to inhibit respiration, but not enough to stop other body processes.

Geiger (9) reported a case with recovery from cyanide poisoning, and that report, more than anything else in recent years, served to stir up interest in the search for antidotes to hydrocyanic acid. In a later report (10) he cites 2 cases of hydrocyanic acid poisoning, and 2 which were, probably, carbon monoxide poisoning, but it would be difficult to credit methylene blue with saving either of the carbon monoxide cases, in as much as both were administered artificial respiration, and one, certainly, had the characteristic relapse which is not uncommon in carbon monoxide intoxication.

Bell (11) reports a case in which favorable results were attributed to the injection of methylene blue, although the patient received inhalations of 95 percent oxygen with 5 percent carbon dioxide, a recognized treatment for carbon monoxide poisoning, and in addition, caffein sodium benzoate, 3¾ grains.

Chen, Rose, and Clowes (12) studied both methylene blue and amyl nitrite in cyanide poisoning and found the latter to be more efficient. They found that methylene blue would antagonize, or protect against 2 lethal doses of sodium cyanide, while by the inhalation of amyl nitrite dogs could tolerate 4 lethal doses of sodium cyanide, and that those that died from larger doses, survived longer if treated with the amyl nitrite. The amyl nitrite also had the advantage of ease of administration and was useful in the control of muscular rigidity and convulsions. They state that the frequency of the dose of amyl nitrite should be reduced rapidly to 1 every 30 minutes, and finally, to every 2 to 5 hours, as respiratory and pulse rates approach normal.

Foresti (13) suggested the use of sodium tetrathionate in the treatment of cyanide poisoning. Draize (14) found it to be somewhat more effective than methylene blue in cyanide poisoning, and considerably more potent in carbon monoxide intoxication than methylene blue.

Brooks (15, 16, 17) advocated methylene blue as an antidote in both hydrocyanic acid and carbon monoxide poisoning, and was responsible for its trial by Dr. Geiger in 1932, on the case that has been publicized so widely. She advocates its use, also, in carbon monoxide poisoning and makes the statement (15) in part, that,

* * * the injection of methylene blue, which has the power to carry oxygen catalytically to the tissues, as soon as it is injected, enables the tissues to carry on their normal function, even while the monoxide is still in the blood, so that these degenerative processes are thereby avoided.

The facts included in the above statement have not been proved, experimentally or clinically.

CONCLUSIONS

1. In view of the fact that the antagonistic action of methylene blue for cyanide has been demonstrated by many noted research workers, it should be tried in every case of cyanide poisoning where it is available, and it should be kept available in all places where patients poisoned with cyanide may require treatment.

2. Amyl nitrite and sodium tetrathionate may be used also, either alone, or in the case of the nitrite, in conjunction with the dye. They may be of value in carbon monoxide intoxication.

3. The preponderance of evidence is against the value of methylene blue in carbon monoxide intoxication, and in view of the fact that both the monoxide and the dye unite with the hemoglobin in the blood to form methemoglobin, it does not seem reasonable that a treatment which further lowers the already deficient oxyhemoglobin could be of value to a patient suffering from carbon monoxide intoxication. The use of oxygen-carbon dioxide mixture (oxygen 95,

carbon dioxide 5) with prolonged artificial respiration, seems to be the treatment of choice.

4. While it appears that methylene blue, administered intravenously, may act as a respiratory stimulant, there is no evidence that it is more efficient than carbon dioxide, alone, or when inhaled with oxygen.

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ORAL PROPHYLAXIS

By E. B. Howell, Lieutenant Commander, Dental Corps, United States Navy

In naval dentistry, the year 1934 was marked by the inception of a systematic and methodical administration of oral prophylaxis aboard ship. The institution of this branch of preventive dentistry afloat was made possible by policies adopted by the Bureau of Medicine and Surgery whereby necessary facilities were added to ship's dental departments.

In most instances additional space was needed. Fortunately, it is no longer necessary to "sell" modern dentistry to the lay mind. Recognizing the many benefits to be derived from such an undertaking, these spaces were readily obtained through the hearty cooperation of commanding officers who truly had the interest and welfare of the crew at heart.

Although oral prophylaxis is primarily instituted for the prevention of dental caries and gingival disease, the results obtained are far more important when we embrace all the measures that the term implies. Our present realization of the interdependence of mouth health and general health, makes it difficult to estimate the far-reaching effects of this branch of dentistry.

It is also a notable fact that dental prophylaxis accomplishes far more than the mere maintenance of health. Statistics from educational centers throughout the country prove conclusively that a person whose mouth is placed in an hygienic condition takes more pride in himself and his surroundings and acquires in general a higher standard of living.

Aboard the U. S. S. *Nevada* for a 12-month period oral prophylaxis was undertaken in a routine manner so that every man on the ship had received treatment at the end of the first 6-month period. At the end of the second 6-month period each man had again been treated and his condition recorded and checked against the first examination. Many of the significant results obtained from this treatment cannot be shown by figures. However, it was most gratifying to note the marked improvement in mouth hygiene and the exultant attitude of the average man to this service.

Prior to their visit to the dental clinic, these men had received instruction in oral hygiene given by the dental officer to several assembled divisions at commanding officer's inspections Saturday mornings. General observance of the laws of health was stressed, giving particular attention to the proper use of the toothbrush, the necessity of guarding against infectious diseases, and the importance of diet in relation to teeth and to general health.

Prophylaxis treatments were likewise rendered to divisions in their numerical or alphabetical order in such a manner that the divisions were never deprived of the services of more than two men at a time. Yet a constant flow of personnel to the dental clinic was assured. Upon completion of treatment by the technician, an examination was made by the dental officer, defects noted and recorded. Acute conditions such as diseased roots, extensive caries, acute gingivitis, Vincent's infection, were treated immediately. The more chronic cases were either given an appointment or placed on a waiting list.

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At the end of the first 6-month period, men treated numbered 1,175; incidence of caries, 40 percent.

At the end of the second 6-month period, men treated numbered 1,207; incidence of caries, 30 percent.

During this period the ship's complement averaged 950 men. Consequently there was a 50-percent turnover in personnel. Therefore these figures are, of necessity, approximate.

The most outstanding result is evidenced by the total absence of Vincent's infection in the second group. The occasional presentation of a case since completion of the second group has been in each instance a recent arrival aboard ship.

It is highly essential in fitting out an office of this kind that an additional dental technician be provided. Dental technicians in the naval service occupy a position comparable to that of dental hygienists in civilian practice. In most States legislation has been enacted limiting the activities of dental hygienists "to the removal of lime deposits, accretions, and stains from exposed surfaces of the teeth and directly beneath the free margin of the gums"; but "prohibit the performance of any other operation on the teeth or mouth or any diseased tissues of the mouth." Naval dental technicians are limited in a similar manner and are not permitted to attempt any other procedures in the mouth. In performing these duties in which they have been trained, and therefore most capable of performing, they render an invaluable service to naval personnel.

Furthermore the advantages of having an extra chair are many and varied. By utilizing it for X-ray work, both dental and medical, the dental officer is saved many an interruption. A sudden influx of acute cases, particularly extractions, may be placed in the second chair, there to be anesthetized. Examinations and charting, the polishing of fillings, may be accomplished during spare moments. In the absence of the dental officer, both chairs may be utilized for prophylaxis treatments, the final examinations being given at a later date. In fact, it is believed that, by the addition of this new equipment, efficiency and output have been so increased that in the not too distant future an office not so equipped will be considered a relic of the dark ages.

It is the consensus of opinion aboard this ship that oral prophylaxis, in addition to a marked improvement in mouth hygiene, has resulted in increased efficiency on the part of the individual both in personal appearance and performance of duty, and in a general improvement of morale.

PSYCHOLOGY OF THE SICK

By B. W. Hogan, Lieutenant, Medical Corps, United States Navy

In the study of the sick individual it is well to have an idea into which classification he belonged before his illness—heretofore most students of man have used an arbitrary division, based on two basic types, the "long thins" and the "short thicks"—natuarlly there being no absolutism in nature, blendings of these two opposites occur in great number and variety of expression. It is not always easy to place a given individual definitely, either among the longs or the dense (ulcers—"long thins," gall bladder—"short thick"). The "long thin" or the asthenic is pale, scrawny, long-limbed with narrow head and face, long, narrow straight nose, small, often receding chin, narrow chest and abdomen, deficient development of fat and musculature, reduced pilosity on the body, but often with abundant cranial thatch, abstemious, dyspeptic, with a tendency to tuberculosis, and when insane, schizophremic, i. e., prone to fixed ideas, ideas of persecution, etc. This type is active, intense, intellectual, self-centered (introverted), often deficient in a sense of humor, fond of reforming, dogmatic or fanatical, and not infrequently detestable when claiming a too intimate knowledge of the Almighty's plans for making the world safe for democracy. The "short-thick" or pyknic—so-called, not because he likes picnics, though no other type is so fond of them, but from the Greek work πokvos, meaning compact or thick-set, is rubicund, rotund, large bodied, short limbed, broad through the chest, but broader through the abdomen, with round or pentagonal face, pug or thick nose, moderately pilose, fond of eating and drinking, eupetpic, with a tendency to apoplexy and arteriosclerosis, on the mental side, cyclothymic, i. e., predisposed to the recurring circular or manic depressive form of insanity, such as melancholia, extroverted, socially easy-going, tolerant in morals and religion, and often very lovable, because claiming no inside information in regard to Almighty's designs.

Now with this classification of types in mind, the "long thins" and the "short thicks," it will help us in sizing up the individual, and usually when sickness comes, these characteristics are in some degree exaggerated, although this classification is not infallible. While there is little doubt in anyone's mind that at present the most sensitive instrument for measuring personality is our intuitive sense, yet the intellectual faculty demands one by which the processes of reason can reach the same end. For this purpose there exist in general, two methods of rational approach to the study of the psychic panel of a human being: One of these is by direct objective observation of personality traits as demonstrated in gestures, attitudes and other forms of response to questions and problem situations, the other is by an analysis of the conscious unconscious mechanism. Personality cannot be

measured by instruments of precision or encompassed by a mathematical formula. Yet personality is a force so powerful and definite that all the world is acutely aware of its distinguishing presence at every human contact. It is the most difficult quality of man to comprehend and in most descriptions its subtle essence is quickly lost. The tang of personality changes continuously from infancy to old age, through five successive epochs of development, the prepuberty, pubescence, complete adult, climateric, and senescence. We can view the patient in a perfectly common-sense way and fit the facts of his personality into the objective and practical data of clinical medicine. There is nothing as scientific as common sense in our dealings and handling with the patient.

The experience we call disease is just like a quarrel. Its signs and symptoms are likewise evidence of conflict between a particular set of qualities of a human being and an equally particular set of adverse forces in the environment. These forces may come from any of the four corners of the universe, riding upon the black wings of those four grim and powerful dragons, physical violence, chemical violence, bacterial violence, and psychological violence. Attacked thus from every angle by the legions of adversity armed with bewildering variety and completeness, how shall mere man hope to live without suffering, or indeed survive? But after all it is not so hopeless as it sounds for Nature is the great magician, the mighty balancer of forces. For if indeed, with a turn of one hand, she hurls engines of destruction, she is able by a cunning burst of the other to set up bulwarks of defense within the living organism.

Disease tends to bring about a certain amount of regression. The sick person tends to become dependent and childlike and to wish for and demand the care and treatment accorded a child. His language, therefore, characteristically displays this infantile dependent quality as one of its components.

The irritability, unreasonableness, fault-finding, complaining, apprehension, doubt, peculiar mannerisms, and many other characteristics of the physically sick need to be interpreted to be understood, for they cannot be taken at their face value, they do not mean what they appear on superficial observation to mean. These patients are talking in a different dimension, their language is conditioned by motives which we know little or nothing of in ourselves, because we do not need them or have not been thrown back to the necessity of their use. With our sick patients we must remember that they are trying to express how they feel and nothing is more difficult as witness the extreme paucity of the language of the emotions. There are no words that adequately convey the meaning in this sphere of human experience.

That the physical or emotional condition has much influence on patients generally must be evident to anyone who gives it consideration. In the daily life it all plays its part in affecting the outlook. We have only to observe the effect of disappointment, fear, and joy, to make plain the fact. Is it not well illustrated in the child who is relieved by the "kiss to make well" and "we are but children of larger growth." As a matter of fact the unrelieved, disturbed emotional condition may and often has serious effect on both mental and physical health, which may lead to dire consequences, strikingly illustrated as shown in the many cases resulting from the World War. Patients through their complaints express hidden motives that must be recognized if rapport and cooperation are to be assured. They have emotional or life problems that result in attitudes seriously influencing a plan of treatment. Often the unexpressed problems of the patient are more serious than the physical problems complained of. It is possible by taking time and allowing the patient to talk to reveal hidden motives and emotional problems and the use of these facts in handling the patient is a big factor in the art of medicine. Nervous breakdown is a very common complaint and many take a real pride in telling that they have had it, especially if they have had several attacks. They roll the term under their tongues like a sweet morsel—the emotional element is perhaps the sole cause. There is no doubt, however, that by far the most difficult problem of all for a patient to face is that which leads to the necessity of dealing with the emotional life. In all acute diseases, pneumonia, typhoid fever, etc., the emotions have their part; the people reported as "making a brave fight" are those whose emotions are under good control and do not, therefore, lessen the physical resistance, preserving the emotional calm.

Physical disease usually involves variations in mood with consequent changes in general mental content and a tendency toward repersonalization. The psychological symptoms in fever are particularly marked, these often include flight of ideas and psychomotor excitement. In extreme cases there also is clouding of consciousness, hallucinations, and perhaps delusions. Common observation that patients with tuberculosis are optimistic and even mildly uephoric, while patients suffering from diseases of the liver are pessimistic, melancholy, and hypochondrical. Influenza may also be followed by lethargy and depression. It is likewise commonly supposed that dyspepsia produces irritability and that cardiac diseases are associated with nervous anxiety. Southard suggested a rough correlation between the location of a disease and the accompanying feelings. He pointed out that diseases above the diaphragm are frequently accompanied by pleasant feelings, while those below are more likely to be accompanied by unpleasant feelings.

Why does a patient go to the doctor anyway?

I presume his motive is that he has some problem on his mind that he cannot settle himself. He has some conception of something that he expresses in terms of defect, or maladjustment, or disease, or lack of function in some part of his body which interferes with his accomplishments, what he wishes to do in life.

It is quite characteristic for patients to present their complaints to the doctor as though they were holding up for his observation an unpleasant burden, of which they would like to be relieved. "Lift it off, doctor"; "do something for me"; "get rid of this package of misery." Lifted to the desk the package is carefully unwrapped while the patient sits at a distance. The process of unwrapping so strangely wrapped a parcel requires many elaborate instruments and much practice and becomes a fascinating occupation, often unknown folds and knots are met with, which in themselves are so intriguing that before long not only is the waiting expectant bearer of the package forgotten but the contents of the parcel itself never reached. There follows an elaborate description of the package and finally a diagnostic label is fastened to it and it is handed back to the bearer. For a time the patient is interested in the label and shows it with as much pride as discomfort will permit to his friends, and the physician for a time gains credit as a labeler of packages. The ball of responsibility having been tossed to the doctor, the patient sits back and expects the physician to remove the package of misery, the patient is persuaded that when the symptoms are diminished the package is removed. Thus the reason why quacks and cults and charlatans are so successful.

No matter how much faith the patient may possess, the figure of the physician expresses a dual symbolism. On the one hand he represents hope and health and on the other must likewise signify disease and death. This latter and foreboding representation is not altogether easy to overcome, because, like all other symbolic influences, it exerts its effects, despite reason, upon the emotional levels of the subconscious mind. It menaces the instinct of self-preservation. Mutual confidence destroys this fearsome element.

There are two qualities which the sick individual shows to a great or less degree—fear and desire for attention.

He is fearful of the unknown, of the consequences of the disease, that the physician has not made a correct diagnosis or does not understand the trouble for which he is giving treatment, that the medicine will not act as it should and that it will not successfully combat the developing disorder of untoward results, of chronicity, of disabling effects, and that death is impending. Most fears evaporate when they are squarely looked at.

The desire of the patient for tangible attention is also a marked characteristic. He likes the medical visitor or social worker to stop and examine or talk with him at each visit. He is not satisfied with the physician who looks in hastily, glances over his chart, gives a few orders, and departs. He is sick. He is important to himself if not to anyone else. His mind is beset with doubt and fear and anxiety. He wants to be fussed over, and he welcomes the kind of attention of which this is considered a necessary part. There is a sense of martyrdom or hereoism in certain patients, and they wish to be fully appreciated.

The patient comes to a hospital, he runs the gauntlet of admission procedures, he is given a bed amongst unfamiliar faces, he submits himself to numerous and various laboratory tests, he makes a tour of the X-ray department, all this time he usually is handed a slip and informed to report at such a department, his opinion is very seldom asked and there is nothing explained to him; in the meantime he is attempting to become accustomed to other phases of hospital existence—the meal hour, for instance, an important event in every man's life—the house diet, which is apparently planned for its calories and vitamines, but with little regard, if any, for individual taste, much less national appeal.

The hospital smells may next engage the patient's attention; perhaps the so-called "institutional odor" is present—that fetid stench compounded out of human sweat, carious teeth, infected tonsils, kitchen smells, neglected bed pans, and stagnant atmosphere.

He must accustom himself to the hospital noise, the rackets, reverberations and echoes issuing from bathrooms, serving kitchens and corridors during 24 hours of the day. So we find our sufferer trying in vain to formulate a satisfactory answer to his question, "Why do sick folks leave home?"

Perhaps by now the patient has learned that, after the first flurry immediately succeeding admission subsides and familiarity has led to the usual reactions, both of the sufferer and of the hospital personnel, the so-called "convalescent period" is inclined to be a bit drab and tedious. This feeling will be emphasized if the hospital does not maintain a library service and has not seen fit to foster any educational plan for patients, be it book work or handwork, or both.

Again, even well people find the pangs of homesickness unendurable; during the World War, mere absence from home and friends, coupled with the ever-present fear of danger or possible death, disabled thousands of well individuals, who represented the flower of American manhood. These were the so-called "shell-shocked" cases. The situation confronting sick persons who enter a hospital for the first time is exactly parallel—many are on the verge of a nervous break-

down—some are actually in danger and know that death is within the range of probability. Pain and distress may be accompanying features; surely such patients find small comfort in the coldness and indifference often encountered in places that claim to be organized for the purpose of promoting health and consequent happiness.

Hospital attendants' attitude toward the sick.—Hospital workers are prone to accept the unreasonable demands of a patient as personal affronts. Daily contact makes it easy to forget that a body diseased often means a mind disabled. Hospital attendants are seldom positively cruel; more often they exhibit this tendency in a very negative sense, expressed by inattention and incivility. Hospital workers should maintain an inexhaustible supply of kindness, consideration, and forbearance, ready for use. An aptitude for debate or a natural or cultivated taste for recrimination have no place in the armamentarium of a physician or a hospital worker.

The Biblical injunction "A soft answer turneth away wrath", should always be kept in mind. Politeness and consideration, its effect on a patient is usually swift and sure. Insolence never yet cured insolence, unreasonableness is not an antidote for unreasonableness. The individual lacking enough in the fundamentals of psychology to interpret the peevish or unreasonable complaints of a sick person as a personal insult is totally unfitted to practice the healing art or care for those distressed both in mind and in body.

During the convalescent period the work of the vocational and occupational therapist is one of the greatest therapy measures and one often neglected. It takes the mind off of one's disabled state. It improves morale and awakens or renews interest in creating something and gives promise of future usefulness to one whose future seemed hopeless. Thus it can be made diversional or purposeful.

The vocational work replaces the idleness, the reading of cheap novels, the card games, and the gossiping and complaining of food and nurses, which make up the life of the average ward patient in the average hospital. It is difficult to make the hospital cheerful, hope-inspiring, without the aid of vocational workers. Daily visiting with all convalescent patients for a fairly extended period is not only practical, but desirable—it consoles the sick person.

For the internist, the surgeon, and the out-patient doctor, the knowledge that the patient has a mind as well as a body, and the cultivating of the psychiatric point of view will enable the physician to treat not only the physical symptoms of the patient, but also his whims and peculiarities, his personality, his mind, and in fact the entire patient, with greater efficiency, greater success, and greater service to mankind.

THE SURGICAL TREATMENT OF RETINAL DETACHMENT

By Grover C. Wilson, Lieutenant Commander, Medical Corps, United States Navy

Great strides have been made in the treatment of retinal detachment in the past decade. In this article an attempt will be made to briefly describe and evaluate the most commonly accepted operations for this condition.

According to Vogt a retinal tear was first seen by Coccius in 1853; De Wecker, in 1870, was the first to recognize that a tear was the cause of detachment; Martin, De Wecker, and de Luca, in 1881, were the first to employ ignipuncture in the treatment of retinal detachment, and Galezowski in 1902 and 1903 was the first to use ignipuncture successfully and systematically in the treatment of retinal tear.

Galezowski's method was, for reasons not definitely known, allowed to fall into disuse and until 1923, at which time Gonin rediscovered, perfected, and employed ignipuncture, the prognosis of detachment of the retina was extremely unfavorable. Treatment was of little value and an improperly treated detachment tends to enlarge and usually becomes total. Spontaneous reattachment is rare, and when it does occur relapses are the rule, and complete blindness is the usual final result. According to Leber spontaneous reattachment with restoration of even moderately useful vision occurs in only 3 to 6 percent of cases. Moreover, excepting those cases due to some local cause, as trauma, there is a strong probability that both eyes will become involved.

The Gonin operation.—After locating the tear the pupil is dilated, the eye anaesthetized, and a conjunctival incision parallel with and just back of the limbus but in front of the tear is made. The conjunctiva is now separated and retracted backwards exposing the sclera overlying the tear at which point an incision 2 to 3 mm long is made with a Grafe knife through the sclera, choroid, and retina in a meridianal direction. Through this incision the tip of a white hot cautery is introduced for a distance of 3 to 4 mm into the eyeball and kept there for 2 to 10 seconds and then slowly removed.

The operation is varied to suit the case. For example, a multiplicity of tears would require more than one puncture.

If the technique is properly carried out the edges of the tear are seared and made to adhere to the choroid thus closing the hole and effecting a cure in a certain number of cases by preventing further seepage of fluid beneath the retina through the tear.

The operation still has its advocates although it is by many considered too harsh a procedure for the delicate structures involved. Furthermore its applicability is limited to those cases in which a tear can be definitely located and we cannot get away from the fact that it is not always possible to find a tear. Linder was able to demonstrate

a tear in only 84 out of 118 cases. According to Schoenberg, Gonin with his treatment by ignipuncture obtained cures in 40 percent of cases of less than 1 year's duration and 55 percent of cases under 3 weeks duration, but others, including his assistant, were unable to duplicate his good results.

The Linder-Guist operation.—Realizing the disadvantages of the Gonin operation Guist and Linder, in 1930, began to employ an operation devised by Guist and reported by him at that time. A conjunctival flap is turned back exposing the sclera over the detached portion of the retina. The tear and borderline between the detached and nondetached areas of the retina are located. The tear is then circumscribed by a number of trephinings 1.7 mm in diameter and, if possible, a second line of trephinings is made along the line of demarcation between the detached and nondetached areas of the retina. The trephine openings are placed not more than 1.25 to 1.5 mm apart since the effect of the chemical cauterization to be used in the next step does not spread over the surrounding area more than 0.5 mm. The trephine openings do not go through the last thin layer of the sclera on account of the danger of perforating the choroid which, if it occurs, may cause collapse of the eyeball and dangerous hemorrhage. This last thin scleral layer is removed with the point of a sharp keratome. After the trephinings have been completed the exposed choroid in each trephine opening is first treated with cocaine crystals and then with the tip of a potassium hydroxide pencil for not more than 1 second. Neutralization is then immediately carried out with a 0.5 percent solution of acetic acid followed by irrigation with salt solution. Finally a small blunt pointed probe is passed through the choroid at each trephine opening to allow drainage of subretinal fluid.

The operation is tedious and may required 2 to 3 hours for its completion and there is always the danger of escape of a small amount of the escharotic into the interior of the globe with resultant damage to the retina and vitreous. However, it can be used in cases not suitable for the Gonin operation, such as those with large tears or cases in which no tear can be located, and is still done by a certain number of men. According to Linder and Guist a larger number of cures are obtainable with this operation than is possible with the Gonin method.

To avoid the necessity of numerous trephinings and the danger of a strong escharotic Linder devised his so-called "undermining operation" in which the area of detachment is delimited by only a few trephine openings, the sclera is separated from the choroid by a spatula passed through these openings, a dilute solution of potassium hydroxide is injected between the sclera and choroid, and the subretinal fluid finally drawn off by perforating the choroid. This

operation is said to have the disadvantage of abolishing the function of that part of the retina fastened down. There is also danger of the caustic liquid's getting into the vitreous through premature perforation of the choroid with the trephine or spatula.

Weve, Larsson, and Safar, in 1930, advocated the treatment of retinal detachment by surgical diathermy.

At this point it may not be amiss to digress for a moment to refresh our memories on the nature of the diathermic current and the difference between the heat produced by diathermy and that produced by the electric cautery. An alternating current with a frequency up to 10,000 oscillations per second causes muscular contractions and what is familiarly known as electrical shock but, as demonstrated by D'Arsonval, when the frequency becomes greater than this figure, muscular contractions cease and the tissues become heated. The frequency of the current used in medical and surgical diathermy varies from 740,000 to 3,000,000 cycles per second. A positive and negative electrode are used, the current passing from the former to the latter through the tissues. The tissues form part of the circuit and by acting as a resistor create the heat. This heat spreads fanwise in all directions but is greatest at the point of contact with the positive electrode. A small pointed positive electrode produces sufficient heat to coagulate or charr the tissues, while if a large flat electrode is substituted and the same amount of current is used the current is spread out and a sense of warmth only is produced.

The electric cautery differs from the diathermic current in that it is a complete circuit in itself, the heat being generated by a resistor in the cautery tip.

The nature of the current is the same whether we are using medical diathermy, the coagulating current, or the cutting current, these terms simply indicating the quantity of current used and the resistance encountered in the tissues. For example, the coagulating current can be changed into a cutting current by using more amperage and retaining the same electrode.

The Safar operation.—Electrocoagulation with the Safar needle electrodes is used. These needles are 1.8 mm long and are made in several forms, the comb type consisting of three needles in series being most commonly used. Other forms are the single short needle on a stalk, and the brush type consisting of a collection of six or seven needles on one stalk. Current sufficient to produce easy perforation of the sclera and choroid with moderate coagulation is used.

For peripheral tears or detachments in the periphery where a tear is suspected the zone of the tear is delimited by making an arch of punctures from ora serrata to ora serrata, the arch passing behind the tear or detachment, and the comblike electrodes being used. All needles are left in place until the operation is finished in order to

prevent the subretinal fluid from escaping prematurely, which would render the bulb soft and allow the retina to come in contact with the sclera, where it would be injured by the next puncture. The area of detachment, which is now enclosed by the arch of needle punctures posteriorly and the ora serrata anteriorly, is then punctured several times with the single short needle on a stalk to allow further escape of subretinal fluid. All needles are now removed and the conjunctiva sutured.

For posterior tears the brush-type electrodes are used, several punctures being made simultaneously with one brush over the area of detachment.

With his method Safar states that in 1932 he was able to obtain reattachment with a good functional result in 57.5 percent of 40 unselected cases; in 1933 he obtained complete reattachment in 85 percent of 40 unselected cases; and that in uncomplicated cases of not more than 5 months' duration he has been able to obtain permanent improvement in as high as 90 percent of the cases treated.

The Safar operation presents certain disadvantages. The needle brushes cannot be cleaned by flaming but must be cleaned by scraping and other time-consuming methods. Unless the needles are perfectly clean and of the same diameter the dosage delivered to each point will vary. Each pin when inserted in the sclera may stick with a pull of 6 ounces so that the total pull required to remove one of the larger needle brushes may be as high as 2 pounds. Approximately 50 milliamperes of current is needed for each needle point. The 150–300 milliamperes required for the larger brushes may be sufficient to cause damage to the macula. If the sclera is wet or the needles become bent toward each other the six-point brush may act practically as a flat electrode and coagulate the sclera severely.

In 1933 Clifford Walker, of Los Angeles, described his technique and instruments for treatment of retinal detachment. Instead of using a multiple needle electrode, such as the Safar brush, he designed and uses single pins with an insulated bakelite holder for inserting these pins one at a time. He also designed his own diathermy machine, but good work is being done by other men with the Walker pins and holder used in conjunction with diathermy machines of other standard makes. He recommends a current with a frequency of 750,000 oscillations per second. The Walker microtips are manufactured from a 15- to 25-percent iridium-platinum wire, which material can be cleaned and sterilized by flaming in a bunsen burner. The microtips are made in two sizes, a 0.3 mm and a 0.5 mm wire diameter being most commonly used. In form the pins consist of a small coil about 1 mm in outside diameter on one side of which there is an eyelet for insertion of a thread and on the other side the pin, the whole being composed of one continuous piece of wire. The coil is made slightly oval to fit without

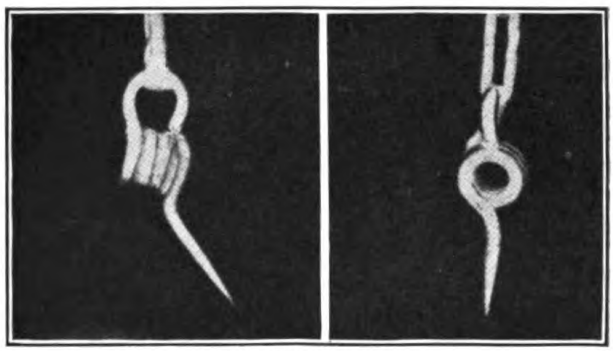

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FIGURE 1.—THE WALKER MICROPIN.

Magnified 10 diameters.

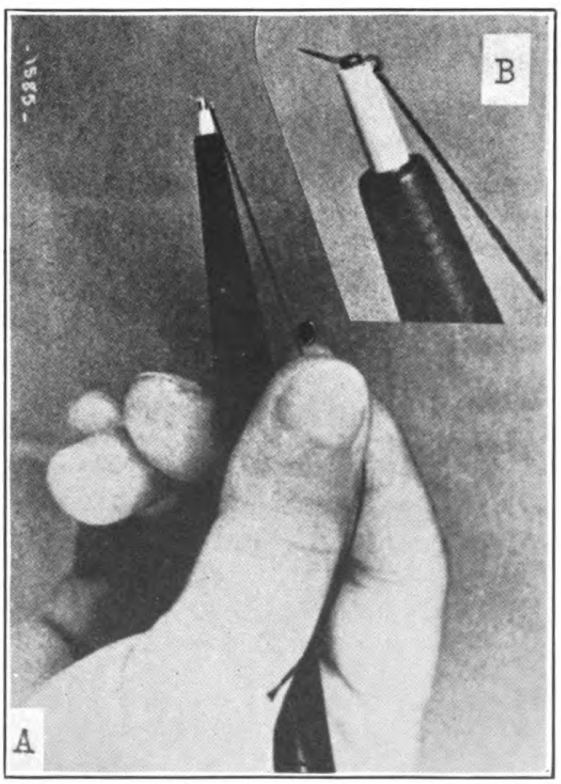

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FIGURE 2.—MICROPIN SHOWN IN FIGURE 1 MOUNTED ON THE WALKER INSULATED BAKELITE HANDLE READY FOR INSERTION INTO SCLERA.

A, Approximately natural size; *B*, magnified about 3 diameters.

rotation the correspondingly flattened stylus of the bakelite handle. The point of the micropin is set at almost a right angle to the coil but may be bent to any desired position (fig. 1).

To prevent loss of the pins in the orbit during operation and to facilitate removal, silk threads are passed through the eyelet and the ends of the threads tied together. For easy handling and sterilization of the micropins a dozen or so may be mounted on a piece of gauze or a wood or cork slab and sterilized by wet or dry sterilization. The threads must be thoroughly dried before use.

The insulated bakelite handle is about 6 inches long and about the size of an ordinary lead pencil. A metal center pin or stylus ending in a point oval in cross section to fit the oval coil of the micropin projects from the tip of this handle. A small rubber cuff insulates the stylus between the handle and the micropin (fig. 2, *A* and *B*).

The Walker operation.—After the area of detachment, and, if possible, the tear, has been located with the ophthalmoscope, the patient's vision and fields are taken, a cycloplegic is instilled, and the eye is anaesthetized, local anaesthesia being used in adults. A conjunctival flap overlying the area of detachment is then turned back. It is usually necessary to sever one or two extraocular muscles which are cut near their attachment to the globe and retracted during the operation by means of muscle forceps or traction sutures. The exposed sclera is then mopped dry and the micropins of smaller size diameter are inserted up to the hilt (coil), which carries them through the sclera and choroid into the fluid in the subretinal space. The pins are placed about 2 mm apart. If the tear can be located a row of these smaller size pins are inserted along the margins of and completely surrounding the tear. A second row staggered with the first may be used although this is not routinely done.

If the tear cannot be located the area of detachment is surrounded by a barrage of the small pins. In peripheral areas this row of pins arches from ora serrata to ora serrata, passing back of the detached area. It is not necessary to close this barrage by placing pins along the ora serrata region, as the firm attachment of the retina in this vicinity acts as a natural barrier. The barrage line will, therefore, in the average case, form an open semicircle involving 100° to 180° of the ocular circumference. The anterior ends of this barrage line should extend to within but not closer than 2 mm of the ora serrata.

In surrounding an area of detachment the barrage of pins should be so placed that they are just within the margin of the detached portion of the retina. In this position, as previously noted, the pins pass through the sclera and choroid but not through the retina, as this structure when separated is pushed up some distance from the choroid by the subretinal fluid. If a pin is accidentally placed outside the

area of detachment it will pass through the retina but does no appreciable harm.

The pins must be inserted one at a time and all must be left in position until the last pin has been placed. If a pin is pulled out or accidentally drops out before they are all in place there is danger of the retina, which is being held up by the subretinal fluid, dropping back into place due to loss of this fluid, in which case it may be pierced by all succeeding pins.

The macular area must be scrupulously avoided in inserting the micropins as the small amount of charring and choroiditis produced by even one pin would be sufficient to destroy the entire area.

After the detached portion of the retina has been delimited by a barrage of pins we next turn our attention to drainage. While the openings left by the pins composing the barrage may allow sufficient subretinal fluid to escape, it is the usual practice to provide additional drainage by inserting 2 or 3 or more of the pins of the larger diameter in the center of the detached area. However, no large openings should be made directly over a tear on account of the possibility of vitreous loss by herniation through the tear.

After all pins, including those in the center for drainage, are placed, they are then removed, preferably one at a time, by means of the attached silk threads. A small quantity of serous fluid is usually seen to escape at this time.

In inserting the pins an amount of current sufficient to cut readily and produce a slight amount of coagulation immediately surrounding the pin should be used. This small amount of coagulum will cause the pin to stick in position until the operation can be completed. If too strong a current is used, charring and not coagulation around the pin takes place, there is no coagulum to hold the pin, and it may drop out with resultant premature loss of subretinal fluid. If too little current is used, coagulation only is produced and the coagulum will plug the hole, interfering with drainage. From 25 to 50 milliamperes for each pin is the proper amount of current. The cutting current is usually employed, in which case the current-control switch, with which several machines now on the market are equipped, is set at about 2, although the coagulation current may be used, in which case the current control switch must be advanced to around 5.

A charred area with wandering of pigment at the site of insertion of each pin can be seen in the fundus with the ophthalmoscope, an adhesive choroiditis being poduced which is sufficient to cause the retina to permanently adhere to the choroid along the barrage line, thus sealing off the detached area so that the retina, which now lies flat against the choroid, is in a favorable position for reattachment over the entire separated area.

After completion of the operation any detached muscles are reattached in their original position, the sutures being loosely tied in approximating the cut ends, as these muscles, being of normal length, exert no great tension on the sutures such as is seen in squint operations. The conjunctival flap is then sutured into position, the eye dressed, both eyes bandaged for 1 to 2 weeks, and the patient is kept in bed for 2 weeks.

While requiring a little more time for its performance than the Gonin or Safar operation it is believed that the Walker operation offers a greater refinement of technic, with all steps of the operation at all times under better control, than any other surgical procedure now being used for retinal detachment.

Several operators using the Walker method report reattachment with good functional results in approximately 50 percent of unselected cases. The percentage of cures in selected cases, by which term is meant uncomplicated cases of not more than 2 months' duration, would of course be much greater.

The author has recently had the privilege of seeing several cases of retinal detachment in which the Walker operation was performed. Following is a report of one of these cases:

Mrs. F. H., age 52, reported for treatment on June 27, 1933, complaining of defective vision in the right eye of about 8 hours' duration.

Past history essentially negative except for a gallstone operation in 1926. The hospital records show that patient's vision at time of this operation was 20/40 with glasses in the right eye and 20/20 without glasses in the left eye.

General physical examination essentially negative. Blood pressure 165/110. Urine negative.

Eyes: Vision in right eye reduced to ability to count fingers at 5 feet. Vision in left eye normal.

Fundi: In the right eye a large bulging separation of the retina in the upper and nasal portions is seen. There are also a moderate amount of vitreous opacities in this eye.

The left fundus is negative except for a moderate sclerosis of the retinal vessels.

Visual fields, right eye: Entire field blind except a very small area above and nasally. Left eye: Field not taken. Probably normal.

June 30, 1933: Walker operation done on right eye. A small hemorrhage into vitreous occurred shortly after this operation.

July 20, 1933: Vitreous hemorrhage about absorbed.

August 29, 1933: Retina in good position. Field normal above, nasally, and temporally. Slight constriction below (reduced to 35°).

September 12, 1933: Vision in right eye 20/50 with glasses.

It is desired to invite attention to the following valuable diagnostic point: In retinal detachment the separation takes place between the pigment layer and the layer of rods and cones. The pigment layer remains attached to the choroid and is not elevated with the detached portion of the retina. If choroidal structure such as pigment or choroidal vessels can be definitely recognized with the ophthalmoscope

in the suspected area the condition is not a detachment of the retina but is either a detachment of the choroid, which is extremely rare, or a sarcoma.

Should there be an error in diagnosis and one of the various operations for retinal detachment be performed on a patient suffering with a sarcoma of the choroid the result would be disastrous as growth of the tumor would be stimulated by the trauma and metastasis would take place through the scleral openings.

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PROPHYLACTIC USE OF ARSENICALS IN SYPHILIS

By Joseph W. Kimbrough, Lieutenant, Medical Corps, United States Navy

The use of the specific arsenicals for prevention of syphilis, following known exposure, has been thoroughly discussed in recent medical literature. These discussions are naturally of a rather theoretical nature since the modus operandi of the arsenic itself in syphilis is not thoroughly understood. Also, unfortunately, this reasoning is largely from a negative point of view; perhaps the patient would not have developed syphilis even if he had not received the arsenical. Another factor is the difficulty of securing accurate data. The patient receives his prophylactic medication, and if no infection follows there is no need for him to return to the clinic. If a luetic infection appears the medicine, doctor, or clinic was no good, according to the patients reasoning, and he goes elsewhere for his treatment. In either case there is a strong tendency for contact to be lost. The same difficulty has been experienced by birth-control clinics in assembling data.

The statement has been made that a certain rather sizeable amount of the arsenical is the minimum with which to secure prophylaxis. This seems difficult to understand when it is remembered that a varying period of time is required in which to give this amount, and all the time the infection is, at least theoretically, making rapid headway in its invasion. In other words a smaller amount of medication should be as effective in the earlier and less-extensive stages of the invasion as a larger amount when a more-extensive infection is present.

It is also generally admitted that, following such prophylactic treatment, an atypical form of the disease may manifest itself at a later date.

Last, but not least, is the ever-present danger from the intravenous use of an arsenical preparation.

The writer has always avoided this form of prophylaxis whenever possible, and, as a result, has not had an extensive experience with it. However, it is felt that the single-case record here reported, consisting as it does of positive evidence, is of more actual value than many cases with negative results from which only theoretical points can be worked out.

J. J. P. C. C. Std.

On October 15, 1932, patient was exposed to venereal infection. Later he was told by the woman that she was receiving treatment for syphilis. None of the details concerning her infection or treatment could be ascertained. On October 18, 1932, patient received, at his own request, neosalvarsan gm 0.45. On October 22, 1932, gm 0.60 of the same preparation was given. A small abrasion was noticed on the penis on November 9, 1932, and a darkfield examination was negative for Treponema Pallidum. On November 15, 1932, the lesion was diagnosed as a chancroid. The darkfield examination was again negative on November 19, 1932. On November 29, 1932, a reddish macular rash was noted about the trunk and forehead. The diagnosis was changed to syphilis on December 31, 1932, and the patient transferred to the hospital where he was first seen by the writer. Here his blood Kahn was 4 plus. Placed on routine antiluetic treatment his course was uneventful.

An interesting point in this case consists of the fact that the patient was infected by a woman who was receiving antiluetic treatment, thus raising the point of whether or not the invading organism was resistant to arsenic. The author called attention to the possibility of this condition in an article several years ago (NAVAL MEDICAL BULLETIN, April 1928).

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CLINICAL NOTES

ARTERIO VENOUS ANEURYSM OF THE INTERNAL CAROTID ARTERY AND THE CAVERNOUS SINUS [1]

Report of a Case

By J. F. Riordan, Commander, and O. R. Nees, Lieutenant, Medical Corps, United States Navy

A carotid cavernous sinus aneurysm is a condition which results from a fistula between the internal carotid artery and the cavernous sinus. This condition is usually discussed under the subject, pulsating exopthalmos. Pulsating exothalmos may be due to other conditions, but by far the most frequent cause is a carotid cavernous fistula. That this condition is not one of the extreme medical varieties is shown by Locke's (1) list of 588 cases. Nevertheless, it is seldom that any one person sees more than 1 or 2 of these cases in a lifetime of busy practice; therefore, it is important that every case should be reported.

Carotid cavernous aneurysm may be classified as traumatic and spontaneous in origin. The trauma may be direct, such as a gunshot wound or indirect as in skull fracture. The spontaneous type results from a rupture of the diseased arterial wall. Locke (1) states that three-fourths of the traumatic cases occur in men and three-fourths of the spontaneous ones occur in women, also that three-fourths of all cases are of the traumatic type. De Schweinitz and Holloway (2) found that the onset occurred during pregnancy in 14 out of 49 women suffering from pulsating exopthalmos. The average age for the traumatic group is between 35 and 40 and for the spontaneous group the average is toward the end of the fifth decade.

The cavernous sinus lies at the side of the body of the sphenoid bone. It extends from the medial end of the superior orbital fissure anteriorly to the apex of the petrous portion of the temporal bone posteriorly where it divides into the superior and inferior petrosal sinuses. Its cavity is irregular in size and shape and is divided by numerous fibrous strands, thus causing it to assume the appearance of cavernous tissue. In its lateral wall are embedded the internal carotid artery with its sympathetic plexus, the occulo motor, the trochlear, the ophthalmic and maxillary divisions of the trigeminal, and the abducent nerves. The superior ophthalmic vein and inferior

[1] Read before the Medical Society of the Territory of Hawaii, Kaual, Apr. 28, 1934.

ophthalmic vein empty into it separately or they may unite before doing so. It communicates with the opposite cavernous sinus by means of the anterior and posterior intercavernous sinuses, often called the circular sinuses. It also communicates with the pterygoid plexus, with the internal jugular vein by small venous channels which accompany the internal carotid artery through the carotid canal and by the inferior petrosal sinus, with the transverse sinus via the superior petrosal sinus and through the superior ophthalmic vein with the angular vein. It should be noted that the superior and inferior petrosal sinuses which carry most of the blood from the cavernous sinus are closed in by rigid structures, namely, bone and dura mater; thus the effects of the arterio venous aneurysm are thrown chiefly on the veins of the orbit. These veins are thin walled and are surrounded by loose aerolar tissues so they readily dilate under the increased pressure. The optic nerve lies close to the anterior end of the cavernous sinus and is accompanied through the optic foremen by the ophthalmic artery. The ophthalmic vein or veins, together with the third, fourth, and sixth nerves, and the branches of the ophthalmic division of the fifth, pass through the sphenoidal fissure. It should be noted that the cavernous sinus is the only place in the body that an artery passes directly through a venous channel, so that the rupture of the artery wall in this locality leads directly to an arterio-venous aneurysm.

The signs and symptoms vary greatly and their severity apparently depends upon the size of the fistula and whether or not thrombosis has occurred in the sinus. As soon as a rupture occurs in the arterial wall the blood pressure in the cavernous sinus rises and, of course, may become as great as the arterial pressure. This reverses the flow of the blood current in the ophthalmic veins. If the communication between the cavernous sinuses is very large the symptoms will be bilateral and less marked than when unilateral.

The bruit is usually the first symptom of which the patient is aware. It has been variously described by patients as buzzing, pounding, roaring, rushing. It may be continuous or synchronous with the systolic pulse. It may change with changes of posture. It may be so annoying that the patient is unable to get sufficient sleep. It is most frequently heard by the patient in the frontal region of the affected side although the patient may feel that it is located in any part of the head or the sides of the neck. On auscultation the bruit is usually heard loudest over the frontal area of the affected side but it may be so loud that it can be heard over any part of the skull or the great vessels of the neck. Cases have been reported in which the sound was audible to the observer although standing some distance from the patient. Compression of the carotid artery on the affected side usually stops the bruit, or in some cases

it may require compression of both sides. Occasionally pressure on the eye ball or the angular vein of the affected side will stop it. In traumatic cases it may be heard immediately after the patient regains consciousness but the usual interval is about 21 days, according to Rhodes. However, its onset may be delayed for months or even years in which case the condition probably is of the spontaneous type. The spontaneous cases are usually sudden in onset.

The next most common symptom is pulsating exopthalmos. Both the proptosis and the pulsation may vary greatly in degrees. The pulsation may be absent or so slight as to be barely perceptible to the finger tips or it may be so marked as to be easily seen on inspection. The proptosis may be so marked as to prevent closure of the eye lids and thus lead to corneal ulceration and loss of the eye. As stated above, the effect of the increased blood pressure is felt mostly by the orbital veins. This leads to great dilation and the veins may become several times their normal size. There is usually a definite thrill to be felt over the region of the superorbital notch. The conjunctive becomes very vascular and the edema may be tremendous. The opthalmoscope usually shows the retinal veins to be greatly dilated, tortuous, and possibly pulsating. There is usually more or less papilloedema.

Various nerve lesions may be included in the picture. These may be due to direct injury since it is found that most basal skull fracture lines involve the superior orbital fissure. The lesion may be caused by pressure and the optic nerve is injured by stretching if proptosis is marked. Any of the following symptoms may result: Reduced vision or blindness, paresis or paralysis of the eye movements, ptosis, dilated and fixed pupil, and loss of corneal sensation. De Schweinitz and Holloway (2) state that 20.4 percent of cases of pulsating exopthalmos terminate in blindness and not more than 11.1 percent retain normal vision. The sixth nerve is the one most commonly involved. Injury to the fifth nerve will result in pain or numbness of the parts supplied by it. The symptoms described above are typical but they may not all be present. In rare cases the eye signs have appeared first on the side opposite the lesion. This is difficult to explain unless it is possible that the rupture in the artery is just opposite the intercavernous sinus, thus transmitting the stream of arterial blood directly to the opposite side.

Another finding which may be of value in differential diagnosis is the oxygen saturation of the blood in the internal jugular vein of the affected side. According to Lennox (3) the saturation in normal subjects is 62 percent, while the oxygen saturation of blood from the internal jugular vein of patients suffering from intracranial arteriovenous fistula is much higher, usually from 91 to 94 percent.

Carotid cavernous sinus aneurysm must be differentiated from (1) malignant tumors or cirsoid aneurysm of the orbit, (2) aneurysm of the orbital vessels (3) orbital cellulitis (4) cavernous sinus thrombosis (5) true aneurysm of the internal carotid artery within the cavernous sinus (6) internal hydrocephalus, and (7) exopthalmic goiter.

A few cases have been reported which have typical signs of carotid cavernous sinus fistula but an autopsy showed no signs of a communication between the artery and the sinus.

The aim of all treatment is to stop the leakage of arterial blood from the internal carotid artery into the cavernous sinus. Direct attack by surgery as has been suggested is too difficult so the usual attack is ligation of the internal carotid or of the common carotid artery on the side of the leak, if the side of the leak is known, or ligation of one of the arteries of side suspected and of one of the carotids on the opposite side if necessary.

Dandy (4) states that up to 35 years the total ligation of the internal carotid artery can be done without harm but thereafter total ligation causes too much risk and that partial occlusion of the artery by a band of fascia should be done. He suggests another method of gradual occulsion of the artery by using rubber tubing which constricts the artery for a periof of 8 or 10 days. The tubing is removed at the end of this time and the contracting scar tissue reduces the lumen of the vessel. The same authority states that after the fiftieth year the total occlusion of the internal carotid is too dangerous to the integrity of the cerebral hemisphere. All observers suggest that before ligation, partial or total, the common carotid be occluded daily for several minutes at first to 1 or 2 hours later. This is done by pressure on the artery over the transverse process of the sixth cervical vertebra. Keegan (5) has devised a collar to put pressure on the carotids preliminary to ligation. This same author believes that a few months should be allowed between injury and ligation to permit collateral circulation to develop.

Dandy (4) believes ligation of the internal carotid on the side of the injury is the best procedure. Huddy (6) suggests ligation of the common carotid on the injured side, believing that anastomoses between the internal and external arteries will continue the fistula if only the internal carotid is ligated. Brooks (7) thinks that by opening the internal carotid artery after a clamp is applied proximally and packing the internal carotid with a strip of muscle obtained from the sternocliedo mastoid muscle and then closing the wound in the artery in the best method. After suturing the artery the proximal clamp is removed and the muscle strip forced toward the rent in the artery by arterial pressure. He has done 3 cases in this manner with good results in 2. The muscle is to increase clotting as well as to plug the rent in the artery. The formation of the thrombus is con-

sidered too dangerous by some observers who feel that this should be avoided. Kerr (8) and Huddy (6) suggest partial ligation of the artery with a second operation several months later in which total occlusion is done, the interval giving the brain an opportunity to accommodate itself to the changed blood supply. Huddy (6) even suggests ligating one common carotid and several months later ligating the other if the symptoms persist. Kolodny (9) suggests ligating the common carotid and the external carotid of the same side because of the anastomoses of the external carotid.

Locke (1) reports from the literature 231 ligations of common carotid with a mortality of 8.98 percent, 38 ligations of internal carotids with a mortality of 7.98 percent, 21 bilateral ligations with a mortality of 14.28 percent, an average for all ligations of 10.38 percent. Huddy (6) and De Schweinitz (2) are in agreement that ligation of the common carotid gives a cure or improvement of 65 percent, failure in about 25 percent, and deaths in about 10 percent. In Locke's (1) series, ligation of the internal carotid gave cures or improvement. There is always danger from immediate ischemia of the brain or later embolism that may cause hemiplegia or aphasia. It has been stated that there is a dimunition of cerebral functions after ligation of both carotids but Huddy (6) states this has not been confirmed. One must always remember that at times spontaneous cures of the fistula occur but the course is usually progressive and blindness is the result.

The case we are reporting is that of R. F. P., EM1c, U. S. C. G., a white male, 29 years of age, who was admitted to the naval hospital, Pearl Harbor, Territory of Hawaii, January 8, 1933. While sitting on the rail of the lanai of his home, he fell backward to the street below, a distance of 15 or 20 feet. He was received at the naval hospital, Pearl Harbor, about 10 hours after the injury. He was stuporous, but could be aroused, when he would answer questions and obey commands. Temperature was 97.6, pulse 58, blood pressure 120/50. Pupils were equal, regular, and reacted to light well. All the tendon reflexes were present and moderately active. There were no pathological reflexes. There was blood in the left auditory canal but no spinal fluid was seen there. In addition to the intracranial injury, he had compound fractures of mandible and a fracture of left radius. At this time the X-ray did not demonstrate the fracture of the base of the skull which was found later. During the first 24 hours the pulse remained between 60 and 70, the blood pressure 120/50, and the respiration 20. During this time he had severe vomiting spells. The second day the patient was clear mentally but drowsy. In the morning there was ptosis of the left upper eye lid and paralysis of the left external rectus muscle and some impairment of the left internal rectus muscle. The left pupil was slightly larger than the right, regular, reacted to light but more sluggishly than did the right one.

The vision of the left eye was blurred. There was a suggestion of protrusion of the left eyeball.

The third day the left pupil was fully dilated and reacted to light. Reaction of the right pupil was normal. The elevator muscle of the left upper eyelid and all the extrinsic muscles were paralyzed. Vision of the left eye was restricted to counting fingers. The exophthalmos was marked. The optic nerves showed no change. The reflexes and mental state had not changed and the blood pressure, respiration, and pulse were normal.

EXOPTHALMOS AND EDEMA OF LOWER LID, LEFT EYE.

The left pupil remained dilated and became fixed. On the sixth day edema of the lower conjunctiva developed. The patient continued to be clear mentally during the day but was irrational at night. On the sixth day a spinal puncture was done. The pressure was 250 mm of water, cells 7; globulin, no increase; Kahn, negative; colloidal gold, no curve. The pressure fell to 150 mm after removal of 35 cc of fluid. The exophthalmos gradually increased as did the edema of the lower portion of the conjunctiva. In addition there was noticed at about 7 weeks after injury a distinct pulsation of the eyeball. The supra orbital vein on the left was dilated and had a distinct pulsation synchronous with the heart beat. There was a thrill over this vein and a loud bruit over the vein and over adjacent portions of the forehead.

About 9 weeks after injury it was noted that there was a slight return of the functions of the extrinsic muscles of the left eye. The vision at this time was about one-third normal. The pulsation of the left eyeball, the thrill and systolic bruit over the left supra orbital notch remained as before. The diagnosis was considered to be arteriovenous aneurism between the left internal carotid artery and the cavernous sinus, but there was a possibility of its being an aneurysm of the ophthalmic artery and superior ophthalmic vein. On April 26, under local anesthetic, an incision was made through the left eyebrow and the supra orbital vessels were dissected out. The superior ophthalmic vein was found to be greatly dilated and continuous with the angular vein. The supra orbital artery was normal in size and there was no communication between it and the vein. The vein contained arterial blood, pulsated, and the blood current was reversed. The vessels were ligated after a section of the vein had been removed. The vision was reduced to counting fingers by this operation. The edema of the conjunctiva increased. The retinal vessels became engorged and the disk chocked. We now feel that the ligation of the superior ophthalmic vein should not have been done as all symptoms increased afterwards. Due to the increase of symptoms, 13 days after the first operation, under local anesthetic, the left carotid sheath was exposed and the common carotid, internal and external carotids, jugular vein, and vagus nerve were dissected free. A rubber shielded clamp was placed on the internal carotid artery. As soon as this was done the pounding and noises in the head ceased. When the clamp was released the pounding and noises returned within a few seconds. The clamp was left on the internal carotid for 10 minutes and as the patient had no untoward symptoms such as hemiplegia or marked vertigo it was considered safe to proceed. A small incision was made in the internal carotid artery above the clamp. There was a spurt of blood proving that there was considerable back pressure in the artery from intracranial anastomoses. Two small strips of muscle were packed upward in the lumen of the artery, though it was thought that these strips did not go as high as the carotid canal. The artery was sutured with silk. When the proximal clamp was released and full pressure restored to the artery there was some oozing. We did not think it safe to depend on this suture so the artery was doubly ligated with silk. The patient showed no reaction after operation; the edema of the conjunctiva remained stationary for about 3 weeks at which time the lower lid was sutured to the skin of the forehead. When the suture was removed, in 2 days the edema was gone and did not return. The exophthalmos slowly receded but never entirely left. The patient was seen 1 year after carotid ligation. The vision in left eye is about 15/20 for distance but there is a paralysis of accommodation and the pupil is somewhat dilated. There is a paresis of the external and superior recti muscles. The proptosis is moderate. There is so far no return of the pulsation or bruit. The patient has been working every day and has no complaint except for occasional headache. We believe it remains to be seen whether a cure has been effected.

SUMMARY

I. There is reported a case of aneurysm of left internal carotid artery and cavernous sinus the result of fracture of base of skull.

II. About 72 hours after injury there developed a left exophthalmos with marked edema of lower portions of conjunctiva of this eye, paralysis of some of the extrinsic muscles of this eye, dilatation of left pupil, marked reduction of vision in left eye, and later a bruit in skull synchronous with heart beat.

III. Ligation of left internal carotid artery improved the exophthalmos and edema of conjunctiva; restored the vision of left eye to 15/20 and stopped the bruit in skull.

IV. One year after ligation of the internal carotid artery the condition had not changed.

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AN INSTANCE OF CIRCULATORY COLLAPSE ATTRIBUTED TO DINITROPHENOL

By Roy J. Leutsker, Lieutenant Commander, Medical Corps, United States Navy

B. C., a machinist's mate in the United States Coast Guard, was given about 6 weeks in which to reduce 25 pounds, the goal set by the examining physician, before he could qualify for reenlistment.

Physical examination on admission:

Weight 206 pounds. Height 67½ inches. Age 32.

Blood pressure 140/90. Exercise tolerance excellent.

No cardiac pathology found.

Urine: All routine specimens negative for albumin, casts, and erythrocytes. Mosenthal test showed SpG. variation of 1.010 to 1.030 with satisfactory day to night ratio.

Blood count normal.

Conclusion: No pathology except obesity.

He was placed on a standard obesity diet of approximately 1,500 calories daily and dinitrophenol grains 4½ daily. After 1 week, having had no trouble with this dosage, it was increased to 6 grains daily. On the morning of the third day following the increase in dosage he suddenly felt weak and faint, collapsed, and was carried to his bed. He was seen almost immediately by a medical officer. He was extremely pale, the skin was cold and clammy, the radial pulse could not be felt. The blood pressure was 70/30. He was totally unconscious at first but regained consciousness within a few minutes.

He was given adrenalin chloride, 1:1000, m 10 and caffeine and sodium benzoate, 2 grains subcutaneously and, after regaining consciousness, several cups of black coffee. Within 10 minutes the radial pulse returned and the blood pressure gradually rose to 120/80 in 30 minutes. He complained of a feeling of lassitude and remained in bed for the rest of the day; the next morning he felt as well as ever.

Dinitrophenol was discontinued and the urine examined frequently during the next few days by Derrien's method. Derrien's test can be made roughly quantitative by judging the intensity of the color reaction produced.

Last dose of dinitrophenol, 1½ grains, at 7 a. m.
First day, 12 m., 4 plus; 4 p. m., 4 plus; 8 a. m., 3 plus.
Second day, 8 a. m., 2 plus; 12 m., 3 plus; 4 p. m., 1 plus.
Third day, 8 a. m., 1 plus; 4 p. m., 1 plus.
Fourth day, 8 a. m., 1 plus; 4 p. m., trace.
Fifth day, 8 a. m., negative.

The following tests were made within 2 hours after the reaction:

Blood sugar, milligrams per 100 cc	158
Chlorides, milligrams per 100 cc	511
Icterus index	8
Blood count:	
RBC	4,980,000
WBC	10,350
Bands	8
Segmented	61
Lymphocytes	27
Eosinophiles	4

One week from the day of the reaction he was again placed on dinitrophenol, 4½ grains daily, and has made a satisfactory reduction in weight of 21 pounds without any further disturbing reactions.

CONCLUSIONS

1. This appears to have been one of those cases described as quantitative intolerance.

2. Cardiac stimulants were effective.

3. Persons susceptible to this type of reaction cannot be detected prior to administration of the drug. In comparing results with other physicians who are prescribing dinitrophenol in the treatment of obesity it appears that in those persons who have a quantitative intolerance, 6 grains daily or thereabouts is the critical dose. Increase in the dosage up to or beyond 6 grains daily should be made gradually and with the patient under close observation.

NAVAL RESERVE

MEDICAL CORPS

Promotions, first quarter, 1935:

John Jerome Goller, MC–F, U. S. N. R., from Lieutenant (junior grade) MC–F, U. S. N. R., January 28, 1935.

Sheldon Albert Jacobson, MC–V (G), U. S. N. R., from Lieutenant (junior grade) MC–V (G), U. S. N. R., March 8, 1935.

REFERENCE LIBRARY FOR RESERVE UNITS

In the offices of the various district medical officers a shelf of books for reference is being provided for the use of medical officers of the Naval Reserve units. It is planned to have on these shelves the following books:

Lovette—Naval Customs and Traditions and Usage.

United States Navy Regulations.

United States Naval Reserve Regulations.

Manual of the Medical Department.

Duties of a Medical Officer Afloat in the United States Navy.

It is believed that this reference shelf will be very useful to the Reserve officers and materially assist them in obtaining knowledge of Navy ways and methods.

POISON GAS IN THE NEXT WAR

At a recent meeting of the Harlem Medical Association in New York City, the president of the Association, Lt. Comdr. Albert S. Hyman, Medical Corps, United States Naval Reserve, arranged and conducted a meeting at which the problems connected with the use of poison gas and the readiness of the civilian population to meet it were discussed by medical officers of the Army and Navy. He drew many of the features to be expected in future wars to the attention of the civilian medical men of the Association.

J. M. FOLTZ.
1871–1872.
Chief of the Bureau of Medicine and Surgery.

NOTES AND COMMENTS

JONATHAN M. FOLTZ, SURGEON GENERAL OF THE NAVY, 1871–72

Jonathan Messersmith Foltz, the sixth in succession to hold the office of Chief of the Bureau of Medicine and Surgery, and the second to be Surgeon General of the Navy, was born in Lancaster, Pa., April 25, 1810. April 25 was also the birth date of Oliver Cromwell and St. Mark. He studied medicine under a preceptor, as was then a common practice, and at Jefferson Medical College, Philadelphia. One of his instructors was Dr. William P. C. Barton, of the Navy, the distinguished surgeon and botanist who afterward became the first chief of the Bureau of Medicine and Surgery. He made a most favorable impression on his pupil and undoubtedly influenced him in choosing a naval career. Foltz was commissioned an assistant surgeon April 4, 1834, his commission being signed by President Andrew Jackson. His first duty was in the new frigate *Potomac*, bound for the East Indies to protect American commerce there and to punish Malay pirates for attacks on American merchantmen. He took part in the celebrated battle of Quallah Batoo, February 6, 1832, when the piratical stronghold of that name on the west coast of Sumatra was captured and destroyed by the landing force from the *Potomac*. He thus began a service which carried him through the Mexican and Civil Wars. In the latter he was one of those who fought with Farragut and was his fleet surgeon in the attack on the forts at the entrance to the Mississippi, the capture of New Orleans, and the subsequent campaign in the western rivers that led to the fall of Vicksburg. He received the highest praise from Admiral Farragut for his courage, ability, and devotion to duty during this arduous period of service; and after the Civil War when the admiral was sent with a specially selected squadron to European waters, he asked Dr. Foltz to accompany him again as fleet surgeon. Following his return he served as president of the board of medical examiners at Philadelphia. In June 1871 he was made a medical director and in October of that year was appointed by President Grant as Surgeon General of the Navy. He retired for age April 25, 1872. His death occurred April 12, 1877.

Dr. Foltz was a man who possessed high administrative talents, and great professional knowledge. Industry and conscientious devotion to duty were among his outstanding characteristics. He had

been the warm personal friend of President Buchanan and his trusted professional adviser. Admiral Farragut placed implicit confidence in his ability and integrity. His own most marked characteristic was summed up by himself in his tribute to the sailor, "The sailor's greatest ambition is to do his duty. May I ever do mine."

VACCINATION AGAINST COMMUNICABLE DISEASE

The status of protective vaccination is a timely matter for every medical man. From a fairly comprehensive survey of the situation made at the end of 1934, by Dr. John A. Kolmer, it would appear that the diseases in which protective vaccination may be said to have been tried and proved of the greatest value, are smallpox, diphtheria, and typhoid fever. The result of vaccination against scarlet fever is not so satisfactory as for diphtheria but is nevertheless of value. The duration of immunity is unknown but believed to be several years. From 50 to 75 percent of children under 10 years of age give positive Dick reactions (are susceptible). In adults it is from 15 to 30 percent. Severe local and general reactions may occur and a satisfactory toxoid has not yet been developed. Vaccinations against Asiatic cholera, bacillary dysentery, and bubonic plague are all believed to be of value both in reducing incidence and mortality. Diseases in which vaccination as a protective means is not well established but new work is showing very promising results, are pertussis (new Sauer vaccine), acute anterior poliomyelitis, psittocosis, tetanus, and pneumonia. Vaccination against rabies used ordinarily only after a bite by an infected or possibly infected animal, has long been a successful mode of preventing the disease. The incidence under Pasteur treatment is less than 1 percent. Diseases in which attempts to obtain a satisfactory vaccine show results that are extremely conflicting or else are attended with danger, are tuberculosis, common colds, influenza, and epidemic meningitis.

DENTAL PROPHYLAXIS IN GENERAL ANAESTHESIA

The journal of the Americal Dental Association states that at the Lenox Hill Hospital, New York City, a careful dental survey of each patient made before general anesthesia and accompanied by appropriate prophylactic treatment was followed by a lessening of the postoperative pneumonic rate. When time would permit, the dental prophylaxis carried out consisted of cleaning the teeth, use of antiseptic mouth wash, and if practicable, removal of infected teeth. Despite the extensive use of local and spinal anesthesia, general anesthetics still have an important place and it would appear that a careful dental study of patients before general anesthesia might be of distinct value when opportunity and time permit it.

FILM STRIP COPIES OF SCIENTIFIC PUBLICATIONS

The difficulty experienced by students and research workers in many fields of obtaining books and articles for use from libraries will be overcome by recent efforts to develop a method of copying the printed page by moving-picture camera and reproducing it with a projector. The organization required in loaning books, the wear of books, the messenger and mail service required, and the fact that with many old, valuable, or irreplaceable books they cannot be permitted to leave the library, a method by which their contents or those of periodicals can be made available through the use of film strips would be a great service and would result in a great economy in the procedure of libraries.

A naval medical officer, Lt. R. H. Draeger, now on duty at the Naval Medical School, Washington, D. C., has by long and painstaking study and work overcome the many technical difficulties that have hitherto prevented the development of an apparatus that for a very small price is of practical use. It is now being used by the United States Department of Agriculture and the librarian has organized a public film service for research workers in the agricultural sciences.

SHORT COURSE ON THE VENEREAL DISEASES AT THE NAVAL MEDICAL SCHOOL

The study of the venereal diseases in the Navy has been a feature which has received the special attention of the Medical Corps. Probably nowhere except in the military service are the diagnosis and treatment of these diseases so carefully carried out and such careful and complete records made. Despite this fact the most hopeful aspect of the whole situation is that the Medical Corps has not grown complacent in regard to the venereal problems, but on the contrary has realized that while the measures for the control, and the diagnosis and treatment are excellent, they are still far from ideal and can be greatly improved. One evidence of this is shown in this short course in the venereal diseases arranged this year by the Naval Medical School, to follow directly after the completion of the regular course given the class under instruction there. This short course consists of 1 week's intensive instruction on the history, prevention, diagnosis, prognosis, and treatment of all the venereal diseases. In addition to the regular faculty of the school and the staff of the naval hospital, Washington, D. C., such distinguished civilian authorities in this field as Stokes, Pelouze, and Moore have accepted invitations to give part of this instruction. Medical officers on duty in and near Washington were invited to attend and many availed themselves of the invitation. In the preventive medicine section of this Bulletin will be found the detailed schedule of the course given.

BOOK NOTICES

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

The Editor, United States Naval Medical Bulletin,
Bureau of Medicine and Surgery, Navy Department,
Washington, D. C.
(For review.)

The Practice of Medicine. By *A. A. Stevens, A. M., M. D., professor of applied therapeutics at the University of Pennsylvania.* 1,150 pages, illustrated. Third edition. W. B. Saunders Co., Philadelphia. $8.

Many American physicians are familiar with Dr. Stevens' little manual of The Practice of Medicine and consider it as one of their best friends. This larger Practice has all the excellent qualities of the smaller book with a greater detail as to the subject. One of the principal advantages of this excellent book is the exact and definite manner in which diagnostic tests and therapeutic measures are given. Directions for the handling of cases are explicit. There are many fine tables of differential diagnosis and numerous tried and true prescriptions used by Dr. Stevens himself in a highly successful practice. Treatment receives a relatively greater amount of space in this book than in most works of this size on the subject. A pupil of Osler, Dr. Stevens' book supplements that master clinician's classical work in a remarkable way and is an American textbook on the practice of medicine of which we can well be proud.

Toward Mental Health. The Schizophrenic Problem. By *Charles M. Campbell, M. D., Harvard University Press, Cambridge, Mass.*

In this little book are collected the Adolph Gehrmann lectures on mental hygiene delivered at the University of Illinois College of Medicine in 1932. While mentioning the mental disorders definitely due to physical causes such as syphilis, alcoholism, encephalitis, pellagra, anemia, endocrine disturbances, vascular conditions and toxins, the principal subject matter is devoted to the study of that realm of personality disorder which, at least at present, is not explained on physical grounds, unless perhaps defects in the germ plasm from which the individual is constructed may be considered as a physical basis of mental disorder. Studies of the harmonizing of conflicts, and of failure or success in achieving independence and

convictions of personal value are illustrated by brief but well-outlined little case reports. These admirable thumbnail case reports such as Dr. Campbell uses should be more frequent in medical literature, not only in the mental field but in all branches of medicine.

This book is a valuable work for the general practitioner to read and for social workers, religious workers, administrators, lawyers, and all those called upon to deal with and solve problems involving the better understanding of those struggling to adjust themselves to life.

REPORT OF SEVENTH INTERNATIONAL CONGRESS OF MILITARY MEDICINE AND PHARMACY HELD AT MADRID, SPAIN, MAY–JUNE 1933. By *Capt. William Seaman Bainbridge, MC–F, United States Naval Reserve, member of the permanent committee, delegate from the United States.* The Collegiate Press, Geo. Banta Publishing Co., Menasha, Wis.

This report of what is one of the most interesting of these Congresses has a foreword by the Surgeon General of the Navy, Rear Admiral P. S. Rossiter. This Congress will be of importance as the one which brought to the attention of the world the recommendations of the Monoco Council of Military Medical Men and International Jurists in regard to certain features designed for the protection of wounded and nonbelligerents in time of war. These recommendations were four in number:

(1) The creation of medical towns and districts.
(2) Assistance by nonbelligerents.
(3) Protection of prisoners of war.
(4) Protection of civilian population.

DISEASES OF THE SKIN. By *Richard L. Sutton, M. D., and Richard L. Sutton, Jr., M. D., professor and assistant professor, respectively, of dermatology at the University of Kansas School of Medicine.* 1,433 pages, 1,310 illustrations, 11 colored plates. Ninth edition, 1935. C. V. Mosby Co., St. Louis. $12.50.

This is one of the best written and most handsomely illustrated manuals of dermatology in print. The book should be of particular interest to the Navy for the senior author was formerly a member of the Medical Corps. Furthermore, the skin lesions of gangosa, verruca peruana, oriental sore, leprosy frambesia, and other tropical skin lesions are given more extensive treatment than is commonly the case in American works on dermatology. Dr. Sutton and his son are well-known big game hunters who have published books on their travels in Africa and elsewhere. They have not lost the opportunity to observe and bring back material for illustrations of the tropical skin lesions they have seen and they form a valuable feature of this part of the book.

The publisher claims for the binding of this book, that it is water and insect resistant and will not become sticky on storage in a humid climate. Genuine gold leaf is used on the back stamp and the appearance is unusually attractive.

BRONCHOSCOPY, ESOPHAGOSCOPY, AND GASTROSCOPY. By *Chevalier Jackson, Professor of Bronchoscopy and Esophagoscopy, and Chevalier L. Jackson, Clinical Professor of Bronchoscopy and Esophagoscopy, Temple University.* 500 pages, 200 illustrations, many in color. W. B. Saunders Co., Philadelphia. Third edition. $10.

This is a complete revision of this important book. The section on gastroscopy is new and, with the increased use of this method, particularly valuable. Of course, the most important feature is the detailed description of the technic used by Chevalier Jackson. Foreign-body removal is covered in the most complete manner. The constantly widening use of bronchoscopy both in diagnosis and treatment of various pathological conditions is given much consideration. There is something of a parallel between the use of bronchoscopy and X-ray. Both, from limited uses, have extended over the whole field of medicine and surgery as essential accessory specialties.

A TEXTBOOK OF SURGERY. By *W. Wayne Babcock, M. D., Professor of Surgery, Temple University.* 1,251 pages, 1,032 illustrations, 8 plates in color. W. B. Saunders Co., Philadelphia. 1935. Second edition. $10.

This excellent work on surgery was one of the best sellers of medical books and now, with a second edition, has been extensively revised and rewritten. The outstanding characteristic is the definiteness of the instruction given. Vague and indefinite generalities have been rigidly suppressed. Everything is set down in the plainest manner possible. Another feature is the illustrations, which are mostly original and designed to show clearly the right and wrong methods of doing things, and illustrating operative technic, step by step.

PRACTICAL ENDOCRINOLOGY. SYMPTOMS AND TREATMENT. By *Max A. Goldzieher, M. D., Endocrinologist, Gouverneur Hospital.* 326 pages, 41 illustrations. D. Appleton-Century Co., New York and London. $5.

This is the very latest book on endocrinology from the medical press of this country and was considered by another leading American internist better than anything now in print in English on the subject. The author has aimed to present clearly the diagnostic features of each type of endocrine disorder and then to discuss the treatment giving specific advice as to the methods of administration and dosage. The book is designed as a practical clinical guide not only for the internist but for the pediatricion, gynecologist, and general practitioner. Theoretical discussion has been largely eliminated and a minimum of space devoted to anatomy, physiology, and embryology.

PHYSIOLOGY IN HEALTH AND DISEASE. By *Carl J. Wiggers, M. D., Professor of Physiology in the School of Medicine of Western Reserve University, Cleveland, Ohio.* 1,156 pages. Illustrated with 182 engravings. Lea & Febiger, Philadelphia, Pa.

This book combines a complete treatise on academic physiology with a capable presentation of the altered physiological processes in disease. The understanding of this altered physiology, with the resultant explanation of the symptomatology of the disease, is as essential to the clinician's understanding, diagnosis, and treatment, as is the study of tissue pathology. More and more, in fact, the physician is finding himself explaining the anatomical changes in the body in the light of altered function, rather than considering the symptomatology so much on the basis of anatomical change.

Too often in the past, text books and instructors have taught physiology as applied to the small laboratory animal, rather than the applied physiology of man, and medical students and physicians alike have found themselves at the bedside without an understanding of the mechanisms behind some of the most common symptoms of disease. Happily, Dr. Wiggers' book goes a long way toward correcting this defect.

The author's explanation of transitory or paroxysmal hypertensive states, and their relationship to essential hypertension, as being due to some extent, at least, to an overflow of stimuli from the emotional center to the closely associated vasomotor center, certainly agrees with the experience of many naval medical examiners, who have often been able, through the media of the annual physical, and promotion examinations, to trace the pathogenesi of this condition, and see these transitory states of hypertension become fixed, first as essential hypertension, and then finally as malignant hypertension, with a generalized arteriolosclerosis at autopsy.

The chapter on the physiology of the sex hormones is well written, and agrees with the modern concepts of their actions. It is to be hoped that future editions of the book will devote more discussion to the altered activity of these hormones in disease, or disturbed physiological states, as the importance of these hormones, as well as other hormones, in menstrual disturbances, sterility, abortion, dysmenorrhea, the so-called diseases of metabolism, and tumor formation in the uterus, breast, and prostate, is now being appreciated.

The arrangement of the material is convenient, the index is most satisfactory and complete, and the format of the book is excellent.

DIETETICS FOR THE CLINICIAN. By *Milton A. Bridges, M. D., F. A. C. P. Director of Medicine, Department of Correction Hospitals, New York.* 994 pages. Second edition. Lea & Febiger, Philadelphia. 1935. Price $10.

The second edition of this book contains 300 more pages than the first, a strong evidence of how complete the revision and how much new material has been added. The diet lists and menus are very

complete and practical. The section on infant feeding makes it a valuable work for both the pediatrician and general practitioner. Vitamines are given a most comprehensive survey, and is yet concise, practical, and up-to-the-minute as to the latest knowledge regarding these important food factors. An interesting feature is a section on beverages with reliable tables regarding those containing alcohol. An extensive bibliography and a good index conclude this excellent book.

PERIODIC FERTILITY AND STERILITY IN WOMAN. By *Herman Knaus, Professor of Gynecology and Obstetrics, University of Prague. Authorized English translation by D. H. and K. Kitchen.* William Mandrich, Vienna. Price $6.50

A Japanese and an Austrian are responsible for the statement of the facts regarding the periodic fertility and sterility phases of the menstrual cycle now generally accepted. Both appeared to have arrived at their conclusions independently and relatively at the same time. Consequently the discovery is very generally referred to as the Ogino-Knaus law. A book by Ogino has been recently translated into English and we now have this work by Knaus. The two books each state this important biologic law. Its advantages are twofold, furnishing as it does a biologic method of contraception unattended by injuries which may result from chemical or mechanical methods, and furnishing also an advantageous method in the treatment of sterility, always one of the most difficult problems of gynecology.

SURGICAL APPLIED ANATOMY. By *Sir Frederick Treves. Revised by C. C. Choyce, Professor of Surgery, University of London.* Ninth edition, 720 pages, 174 engravings including 66 in color. 1934. Lea & Febiger, Philadelphia. Price $4

This is an anatomical classic written 50 years ago by one of the world's foremost surgeons. Most of our medical men have studied it and this last edition has been revised and brought up to date by Professor Choyce in a most thorough manner.

SYSTEM OF DIET WRITING. By *William S. Collens, M. D., Chief of Diabetic Clinic, Israel Zion Hospital.* Form Publishing Co., 200 Hudson Street, New York City. Price $5

This is a diet notebook, particularly useful to a general practitioner or a busy internist.

PHYSICAL DIAGNOSIS. By *Warren P. Elmer, M. D., Associate Professor of Clinical Medicine, Washington University School of Medicine, and W. D. Rose, M. D., Late Associate Professor of Medicine in the University of Arkansas.* Seventh edition, 1935. C. V. Mosby Co., St. Louis. Price $8.50

This is a new edition of a well-known and tried work on physical diagnosis. The general arrangement of the book has not been altered but the section on electrocardiography has been completely rewritten and new material on aortic disease and silicosis and new diagnostic methods have been added.

THE DIVISION OF PREVENTIVE MEDICINE

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

VENEREAL DISEASES, UNITED STATES NAVY, 1900–33

By S. S. Cook, Lieutenant Commander, Medical Corps, United States Navy

Venereal diseases constitute the largest problem in preventive medicine with which the Navy is confronted. These diseases from whatever angle they are considered surpass in magnitude and significance all other communicable diseases. The mass of reports, circular letters, recommendations, and orders in the files of the Bureau

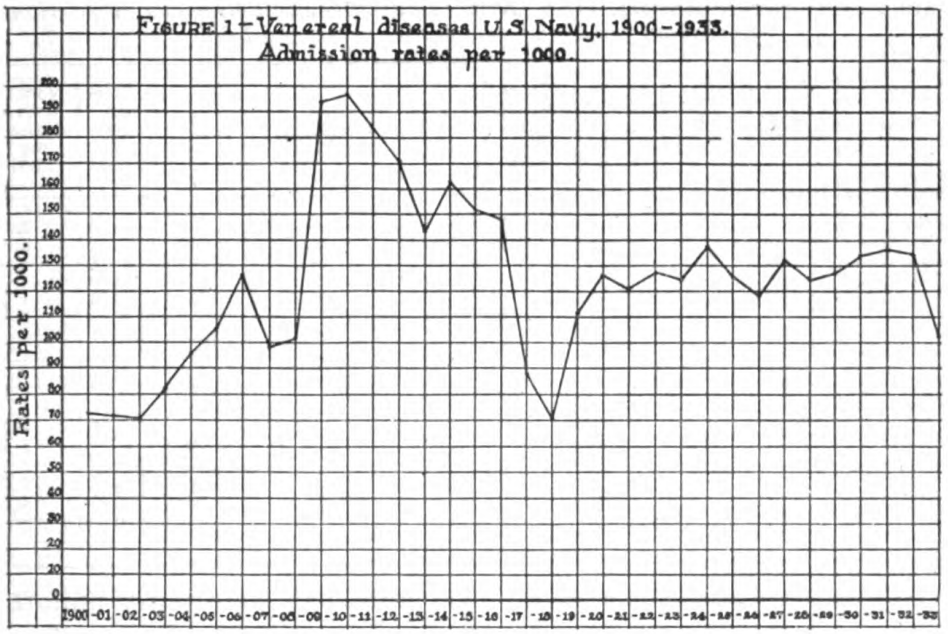

FIGURE 1—Venereal diseases U.S. Navy, 1900–1933. Admission rates per 1000.

of Medicine and Surgery indicates to some extent the enormous amount of time and thought that has been devoted to this subject.

It is the purpose of this study to review the prevalence of syphilis, gonorrhea, and chancroid in the Navy during the 34-year period 1900–33. During these years there were reported 420,025 original admissions for the three diseases. Of this number, 68,704 were for syphilis, 244,206 for gonorrhea, and 90,275 for chancroid. These figures are for primary admissions and do not include complications.

In table I are shown mean strengths, admissions, admission rates per 1,000, and noneffective ratios per 1,000 for venereal diseases in the 34-year period 1900–33. It may be seen that there was a marked increase in the admission rates in 1909 over that of 1908. This

increase is attributed to a change in policy in 1909. Prior to that time men were not admitted to the sick list because of venereal disease unless incapacitated for duty. It was decided in 1909 to admit for statistical record all cases regardless of the degree of disability. As a result the rate was increased from 101.83 per 1,000 in 1908 to 193.52 per 1,000 in 1909. Another significant event transpired in 1909, namely the promulgation of an order by the Secretary of the Navy which officially recognized and advised the administration of prophylaxis to all men who exposed themselves to infection. In the succeeding 5 years the admission rate gradually declined. There was a marked decrease in reported incidence in the World War years of 1917 and 1918. It is of interest to note that incidence has been consistently lower since 1918 than it had been for several years prior to 1918. The rates are depicted in figure 1.

TABLE I.—*Venereal diseases, U. S. Navy, 1900–33—mean strengths, admissions, admission rates, and noneffective ratios per 1,000*

Year	Average strength	Admissions	Admission rate per 1,000	Noneffective ratios per 1,000	Year	Average strength	Admissions	Admission rate per 1,000	Noneffective ratios per 1,000
1900	23,756	1,723	72.53	6.12	1918	503,792	35,364	70.20	2.50
1901	26,873	1,942	72.27	5.63	1919	298,774	33,350	111.62	5.12
1902	31,240	2,205	70.58	5.31	1920	140,773	17,763	126.18	4.66
1903	37,248	3,070	82.42	5.87	1921	148,861	17,871	120.05	4.60
1904	40,555	3,905	96.29	5.70	1922	122,126	15,554	127.36	4.96
1905	41,313	4,353	105.37	5.50	1923	116,565	14,376	123.33	5.06
1906	42,529	5,364	126.13	5.94	1924	119,280	16,409	137.57	4.94
1907	46,336	4,499	97.10	4.50	1925	115,391	14,604	126.56	4.47
1908	52,913	5,388	101.83	4.17	1926	113,756	13,516	118.82	4.44
1909	57,172	11,064	193.52	6.68	1927	115,316	15,253	132.27	4.72
1910	58,340	11,469	196.59	6.48	1928	116,047	14,403	124.11	4.63
1911	61,399	11,271	183.57	7.29	1929	117,388	14,968	127.51	4.76
1912	61,897	10,580	170.93	6.54	1930	117,453	15,728	133.91	5.15
1913	65,926	9,434	143.10	5.88	1931	112,767	15,425	136.79	5.29
1914	67,141	10,932	162.82	5.84	1932	110,717	14,794	133.62	4.74
1915	68,075	10,318	151.57	6.08	1933	108,183	11,083	102.45	4.52
1916	69,294	10,261	148.08	6.56					
1917	245,580	21,786	88.71	2.58	Total	----------	420,025	----------	----------

Noneffective ratios except for 1917 and 1918 were fairly uniform throughout the 34-year period. The ratio has usually been 4, 5, or 6. In other words, the average number of men on the sick list with venereal disease per 1,000 strength was 4, 5, or 6 in each year except 1917 and 1918 when the ratio was 2 and 1911 when it was 7. The noneffective ratios are shown in figure 2.

In table II are shown admissions and admission rates per 1,000 for syphilis in the period 1900–33. In the 34 years there were 68,704 admissions, or an average of approximately 2,000 new cases each year. The lowest admission rate, 10 per 1,000, was experienced in 1917 and the highest, 27 per 1,000, in 1911 and 1932. The rates have usually ranged between 19 and 26.

TABLE II.—*Syphilis, U. S. Navy, 1900–33—Admissions and admission rates per 1,000*

Year	Admissions	Admission rate per 1,000	Year	Admissions	Admission rate per 1,000
1900	465	19.57	1918	5,960	11.83
1901	546	20.32	1919	4,916	16.45
1902	606	19.40	1920	2,470	17.54
1903	816	21.90	1921	2,656	17.84
1904	880	21.69	1922	2,446	20.03
1905	981	23.73	1923	2,170	18.62
1906	1,147	26.96	1924	2,355	19.74
1907	881	19.01	1925	2,261	19.60
1908	1,001	18.91	1926	2,505	22.02
1909	1,476	25.81	1927	2,833	24.57
1910	1,315	22.54	1928	2,633	22.69
1911	1,665	27.11	1929	2,540	21.64
1912	1,424	23.00	1930	2,940	25.03
1913	1,447	21.94	1931	2,871	25.46
1914	1,332	19.83	1932	3,062	27.66
1915	1,454	21.35	1933	2,639	24.39
1916	1,542	22.25			
1917	2,469	10.05	Total	68,704	

In table III are shown admissions and admission rates per 1,000 for gonorrhea in the 34-year period 1900–33. In these years 244,206

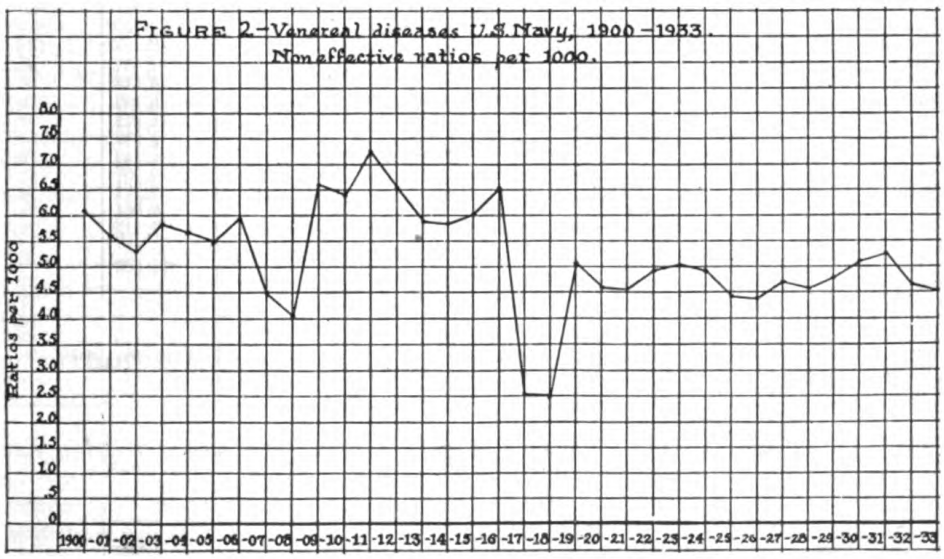

FIGURE 2.—Venereal diseases U.S. Navy, 1900–1933. Noneffective ratios per 1000.

admissions were reported. The relatively small numbers in the first 8 years of the period are doubtless due to the practice in those years of only admitting men to the sick list who were incapacitated for the performance of duty. The marked increase in 1909 is attributed to the inauguration of the policy of admitting for record all cases. Excluding 1917 and 1918, there were no striking changes in admission rates from 1912 to 1932. There was a decrease from 81 in 1932 to 65 in 1933.

In table IV are shown admissions and admission rates per 1,000 for chancroid in the 34-year period 1900–33. In these years there were 90,275 admissions reported. This number does not include chan-

croidal complications. The reason for the relatively small number of admissions in the period 1900–08 is the same as previously stated for gonorrhea. There was considerable irregularity in incidence but at no time has a sustained decrease been experienced.

It has been shown in the preceding paragraphs that the prevalence of venereal diseases was relatively constant in the 34-year period under consideration. Except for a low reported incidence in 1917 and 1918 no marked and sustained increase or decrease has been experienced. Incidence was not appreciably less in the latter half of the period as compared to the earlier half. It is of interest to note that the ratio between admissions for syphilis and for gonorrhea changed greatly in the 20-year period 1914–33. As shown in table V, the ratio in 1914 was 1 to 4.67. There was a progressive decrease to a ratio of 1 to 2.70 in 1933. The reasons for this change are not apparent.

TABLE III.—*Gonorrhea, U. S. Navy, 1900–33—Admissions and admission rates per 1,000*

Year	Admissions	Admission rate per 1,000	Year	Admissions	Admission rate per 1,000
1900	525	22.10	1918	21,404	42.49
1901	617	22.96	1919	20,410	68.32
1902	771	24.68	1920	11,140	79.13
1903	1,032	27.70	1921	11,621	78.06
1904	1,512	37.28	1922	9,987	81.77
1905	2,085	50.46	1923	9,142	78.43
1906	2,640	62.07	1924	10,132	84.94
1907	2,274	49.07	1925	9,114	78.98
1908	3,015	56.98	1926	8,084	71.06
1909	5,861	102.51	1927	8,227	71.34
1910	6,062	103.90	1928	8,353	71.98
1911	5,658	92.15	1929	8,776	74.76
1912	5,403	87.29	1930	8,659	73.72
1913	5,320	80.69	1931	8,761	77.69
1914	5,703	84.94	1932	8,987	81.17
1915	5,985	87.91	1933	7,116	65.78
1916	5,731	82.70			
1917	14,099	57.41	Total	244,206	

TABLE IV.—*Chancroid, U. S. Navy, 1900–33—Admissions and admission rates per 1,000*

Year	Admissions	Admission rate per 1,000	Year	Admissions	Admission rate per 1,000
1900	214	9.01	1918	7,996	15.87
1901	217	8.08	1919	8,019	26.84
1902	284	9.09	1920	4,153	29.50
1903	396	10.63	1921	3,594	24.14
1904	542	13.36	1922	3,121	25.55
1905	538	13.00	1923	3,064	26.29
1906	733	17.23	1924	3,922	32.88
1907	554	11.95	1925	3,229	27.98
1908	665	12.56	1926	2,927	25.73
1909	1,573	27.51	1927	4,193	36.36
1910	1,968	33.73	1928	3,417	29.44
1911	1,929	31.41	1929	3,652	31.11
1912	2,169	35.04	1930	4,113	35.02
1913	1,855	28.13	1931	3,785	33.56
1914	2,908	43.31	1932	2,742	24.77
1915	2,200	32.31	1933	1,326	12.26
1916	3,057	44.12			
1917	5,220	21.26	Total	90,275	

TABLE V.—*Ratio of syphilis to gonorrhea, U. S. Navy, 1914–33*

Year	Syphilis to gonorrhea	Year	Syphilis to gonorrhea	Year	Syphilis to gonorrhea
1914	1 to 4.67	1921	1 to 4.38	1928	1 to 3.17
1915	1 to 4.43	1922	1 to 4.08	1929	1 to 3.46
1916	1 to 3.94	1923	1 to 4.21	1930	1 to 2.95
1917	1 to 5.81	1924	1 to 4.30	1931	1 to 3.05
1918	1 to 3.59	1925	1 to 4.03	1932	1 to 2.94
1919	1 to 4.15	1926	1 to 3.23	1933	1 to 2.70
1920	1 to 4.51	1927	1 to 2.90		

The prevalence of venereal diseases in the 34-year period has been shown and it is now of interest to mention briefly the measures which were adopted during these years in an effort to reduce the incidence of these diseases. The first official step of which there is record was taken in 1909 when on January 9 the Secretary of the Navy issued an order to the Service which required promulgation of information regarding venereal disease to men of the Service and directed that facilities for prophylaxis be made available.

On June 15, 1914, General Order 100 was issued, the first two paragraphs of which are:

(1) The following extract from "An act making appropriations for the support of the Army for the fiscal year ending June 30, 1915, and for other purposes", approved April 27, 1914, is published for the information and guidance of all concerned:

"*Provided*, That hereafter no officer or enlisted man in active service who shall be absent from duty on account of disease resulting from his own intemperate use of drugs or alcoholic liquors or other misconduct shall receive pay for the period of such absence, the time so absent and the cause thereof to be ascertained under such procedure and regulations as may be prescribed by the Secretary of War."

This law, under section 1612, Revised Statutes, applies to like cases arising in the Marine Corps.

(2) Absence from duty of any officer or enlisted man in the United States Marine Corps in active service on account of disease resulting from his own intemperate use of drugs or alcoholic liquors or other misconduct is within the purview of the statute quoted above; and any officer or enlisted man who, on or after April 27, 1914, has been absent or may hereafter be absent from duty from any such cause or causes is not entitled to pay, as distinguished from allowances for the period of such absence. But where the disease, within the purview of the statute quoted above, was contracted prior to April 27, 1914, as the result of which the officer or enlisted man is absent from duty after that date, no deduction of pay will be made.

The provisions of this order were held applicable to all persons in the Navy in General Order 231 of August 31, 1916. The portions of this order referring to venereal disease are quoted herewith:

Hereafter no officer or enlisted man in the Navy or Marine Corps in active service who shall be absent from duty on account of sickness or disease resulting from his own intemperate use of drugs or alcholic liquors, or other misconduct,

shall receive pay for the period of such absence, the time so absent and the cause thereof to be ascertained under such procedure and regulations as may be prescribed by the Secretary of the Navy: *Provided*, That an enlistment shall not be regarded as complete until the enlisted man shall have made good any time in excess of 1 day lost on account of sickness or disease resulting from his own intemperate use of drugs or alcoholic liquors, or other misconduct.

On October 18, 1917, General Order 332 was issued in which section 18 of the Selective Service Act was quoted. This order deals with prostitution in extra cantonment zones and prohibits the setting up of houses of ill fame, brothels, or bawdy houses within 5 miles of any place under naval jurisdiction. The provisions of this order were amplified and made more specific in General Order 359 of January 17, 1918. This order was further modified by General Order 411 of August 3, 1918, and the prohibited zone was increased to 10 miles.

The subject of General Order 451 of February 5, 1919, was forfeiture of pay. General Orders 100 and 231 were amended by this order as follows:

* * * any officer or enlisted man who, on or after July 1, 1918, has been absent or may hereafter be absent from duty on account of such injury is not entitled to pay, as distinguished from allowances for the period of such absence. But no deduction of pay will be made in the case of an officer or enlisted man absent from duty on account of injury coming within the purview of the above-mentioned statute, where such injury was incurred prior to July 1, 1918.

Measures for the prevention and control of venereal disease were set forth in General Order 530 of May 12, 1920. This order provided for instruction as to the nature and dangers of venereal disease, the necessity for prophylaxis after exposure, and prohibited the issuance of prophylactic packages to liberty parties. This order was reissued as General Order 29 on January 5, 1921. General Order 29 was amended on September 16, 1921, by General Order 69, the principal changes being (1) failure to take prophylaxis did not constitute disobedience of orders; (2) the issuance of prophylactic packets to liberty parties was no longer prohibited.

The order which is in force at present is General Order 69, amended, which was issued on April 13, 1926. This order comprises the recommendations of a board appointed in December 1924, to study the venereal disease problems of the Navy. Upon recommendation of the Surgeon General and by direction of the Secretary of the Navy this board was convened by order of the Chief of the Bureau of Navigation. The officers composing the board represented the Bureau of Navigation, the office of the Major General Commandant, United States Marine Corps, office of the Judge Advocate General of the Navy, and the Bureau of Medicine and Surgery. Their report was published in the United States Naval Medical Bulletin, December 1925.

In the preceding paragraphs data have been presented with respect to the prevalence of venereal disease in the period 1900–1933 and also a narrative of administrative preventive measures.

It is now of interest to refer to statistical data which are available for a part of the period regarding the treatment of syphilis. The magnitude of this phase of the problem is very great, the extent of which may be more easily appreciated when one recalls that there are about 2,500 new cases of syphilis each year and about 12,000 syphilitics in the Navy.

In table VI are shown the number of doses of arsphenamine, neoarsphenamine, and other arsenicals given in the 9-year period 1925–33. More than 840,000 doses were given, of which number about seven-eighths were neoarsphenamine.

TABLE VI.—*Arsenicals, U. S. Navy, 1925–33*

Year	Arsphenamine	Neoarsphenamine	Other arsenicals	Total	Year	Arsphenamine	Neoarsphenamine	Other arsenicals	Total
1925	5,282	41,789	1,804	48,875	1931	1,353	95,444	7,116	103,913
1926	6,501	55,637	2,243	64,381	1932	174	128,540	10,002	138,716
1927	5,749	68,740	2,443	76,932	1933	89	138,490	8,050	146,629
1928	5,103	71,753	5,704	82,560					
1929	5,018	76,652	3,825	85,495	Total	30,662	762,691	47,026	840,379
1930	1,393	85,646	5,839	92,878					

In table VII are shown by years the number of reactions due to arsenicals in the 9-year period 1925–33 and also the ratio of reactions to doses. There were 614 reactions reported, giving a ratio of one reaction to 1,369 doses.

TABLE VII.—*Arsenical reactions, U. S. Navy, 1925–33, ratio of reactions to doses*

Year	Doses	Reactions	Ratio of reactions to doses	Year	Doses	Reactions	Ratio of reactions to doses
1925	48,875	82	596	1931	103,913	62	1,676
1926	64,381	42	1,533	1932	138,716	72	1,927
1927	76,932	45	1,710	1933	146,629	95	1,543
1928	82,560	67	1,232				
1929	85,495	66	1,295	Total	840,379	614	1,369
1930	92,878	83	1,119				

In table VIII are shown the types of reactions with relative frequency for the 5-year priod 1929–33. It may be seen from this table that about one-half the reactions were classified as disturbances of the vasomotor system and about one-third as arsenical dermatitis. It is of interest to note that damage to the liver and kidneys was relatively uncommon. Although not shown here it is a matter of record that reactions to the hematopoietic system are reported more frequently than was the case a few years ago.

TABLE VIII.—*Arsenical reactions, U. S. Navy, 1929–33, types and proportions*

Type	Number of reactions	Percent of total reactions
Vasomotor phenomena	190	50.26
Arsenical dermatitis	118	31.23
Reactions of minor importance	17	4.50
Liver damage	12	3.18
Blood dyscrasias	11	2.91
Table reactions	11	2.91
Jarisch-Herxheimer	7	1.85
Acute renal damage	6	1.59
Hemorrhagic encephalitis	3	.79
Polyneuritis	1	.26
Border-line, hemorrhagic encephalitis	1	.26
Liver damage (doubtful reaction)	1	.26
Total	378	100.00

In table IX it is shown that there were 51 deaths due to administration of arsenicals in the 15-year period 1919–33.

TABLE IX.—*Arsenical deaths, U. S. Navy, 1919–33*

Year	Deaths	Year	Deaths	Year	Deaths
1919	3	1925	2	1931	0
1920	2	1926	4	1932	4
1921	4	1927	5	1933	7
1922	4	1928	6		
1923	1	1929	3	Total	51
1924	3	1930	3		

SUMMARY

The statistical data which have been presented serve to indicate the prevalence of venereal diseases in the Navy in the 34-year period 1900–33. Additional data have been presented to show the number of doses of arsenicals given during a part of the period and also the reactions resulting therefrom.

There are so many factors which influence the incidence of venereal diseases that attempts to interpret or explain variations which have occurred are open to question. It would serve no useful purpose to recite the possible reasons for such variations in incidence as they are all theoretical.

SPECIAL COURSE ON VENEREAL DISEASE, WEEK OF APRIL 1–6, 1935

An advanced special course on venereal disease was given at the Naval Medical School during the week of April 1–6, 1935, the detailed program of which follows:

Monday, April 1

9:30–10 a. m. Introductory address, Surg. Gen. P. S. Rossiter, U. S. Navy.

10–11 a. m. History of Syphilis, Comdr. L. H. Roddis, (MC), U. S. Navy.

11 a. m.–12 m. Syphilis in the Navy, Lt. Comdr. S. S. Cook (MC), U. S. Navy.

1–3 p. m. Public Health Aspects of Syphilis, Passed Asst. Surg. R. A. Vonderlehr, U. S. Public Health Service.

3–4 p. m. Social Means of Control of Venereal Disease, Dr. Valeria H. Parker, director, community relations, the American Social Hygiene Association.

Tuesday, April 2

9–10 a. m. Personal Hygiene, Capt. H. W. Smith (MC), U. S. Navy.

10 a. m.–12 m. Laboratory Diagnosis, Lt. Comdr. F. M. Rohow (MC), U. S. Navy.

1–3 p. m. Gonorrhoea, Dr. Percy S. Pelouze, assistant professor of urology, University of Pennsylvania School of Medicine.

a. A Consideration of the Basic Factors in Gonococcic Infection.

b. The Clinical Course and Complications.

3–4 p. m. Pathology, Lt. Comdr. G. A. Alden (MC), U. S. Navy.

Wednesday, April 3

9–10 a. m. Arsenicals, Medical Director Carl Voegtlin, U. S. Public Health Service; pharmacologist director, National Institute of Health.

10–11 a. m. General Management, Lt. Comdr. C. L. Andrus (MC), U. S. Navy.

11 a. m.–12 m. Syphilis of the Spinal Cord, Dr. Walter Freeman, neurologist, St. Elizabeths Hospital; professor of neurology, George Washington University Medical School.

1–3 p. m. Biology of Syphilis, immunological principles, therapy, and criteria of cure, Dr. John H. Stokes, professor of dermatology and syphilology, University of Pennsylvania School of Medicine.

3–4. p. m. Cardio-Vascular Syphilis, Comdr. W. W. Hargrave (MC), U. S. Navy.

Thursday, April 4

9–10 a. m. Heavy Metals, Dr. Stanford M. Rosenthal, senior pharmacologist, U. S. Public Health Service, National Institute of Health.

10 a. m.–12 m. Early Syphilis, Dr. Joseph E. Moore, associate in medicine, Johns Hopkins University; physician-in-charge, syphilis division of the medical clinic; and assistant visiting physician, Johns Hopkins University.

1–3 p. m. Gonorrhoea, Dr. Percy S. Pelouze.

a. Therapeutic Considerations.

b. The Application of Treatment of Large Goups of Patients.

3–4 p. m. Treatment, Syphilis of Central Nervous System, Dr. Theodore C. Fong, St. Elizabeths Hospital.

Friday, April 5

9–10 a. m. Reactions, Lt. Comdr. C. L. Andrus (MC), U. S. Navy.

10–11 a. m. X-ray Diagnosis, Lt. Comdr. R. W. Hayworth (MC), U. S. Navy.

11 a. m.–12 m. Biological Relationship between Yaws and Syphilis, Comdr. Paul W. Wilson (MC), U. S. Navy.

1–2 p. m. Latency, Dr. Thomas W. Murrell, associate professor, dermatology and syphilology, Medical College of Virginia.

2–3 p. m. Syphilis of Central Nervous System, Lt. Comdr. A. A. Marsteller, (MC), U. S. Navy.

Saturday, April 6

9–11 a. m. Administrative and Legal Aspects, Capt. M. A. Stuart (MC), U. S. Navy.

11 a. m.–12 m. The "Sixth Venereal Disease", Surgeon Ralph D. Lillie, U. S. Public Health Service. Division of Venereal Diseases, United States Public Health Service.

HEALTH OF THE NAVY

Statistical returns for the last quarter of 1934 gave a general admission rate of 621.63 for all causes—the highest rate for all causes recorded for this quarter since 1929.

The admission rate for disease was 555 per 1,000 per annum. The rate for the corresponding months of 1933 was 402 per 1,000 per annum and the median rate for the preceding 5 years, 502.

The admission rate for accidental injuries (62) was lower than the rate for the corresponding quarter of 1933 but was slightly higher than 57, the expected rate for the corresponding quarter of the preceding 5 years. Except when influenced by unusual accidents this rate varies little from year to year.

Poisonings increased from 4.28 per 1,000 per annum for the third quarter to 5.43 per 1,000 for October, November, and December, due to an outbreak of food poisoning on board the U. S. S. *Lexington.* The 5-year median rate for the fourth quarter is 1.27 per 1,000. To quote from a report received from the U. S. S. *Lexington:* "A moderately severe outbreak of food poisoning, affecting a total of 123 men, occurred aboard the U. S. S. *Lexington,* October 3, 1934. The common symptoms were nausea, vomiting, and diarrhea; a few evidencing severe abdominal cramps, profuse perspiration, pallor, and cold, clammy skin. That it resulted from some article of food originating aboard ship, the particular article of food and contaminating organism being undetermined." One hundred and twenty cases of food poisoning were admitted to the sick list.

During the quarter, 1,301 cases of the common infections of the respiratory type were reported from shore stations in the United States, 79 percent of which were charged to catarrhal fever. The stations recording the greatest number of admissions are shown in the following tabulation:

Station	October	November	December
Naval Academy, Annapolis, Md. (midshipmen)	25	13	22
Naval Academy, Annapolis, Md. (other than midshipmen)	9	12	7
Marine Barracks, Quantico, Va	41	47	94
Naval Training Station, Norfolk, Va	70	161	294
Marine Corps Base, San Diego, Calif	9	11	17
Naval Training Station, San Diego, Calif	67	70	59

The Naval Training Station, Norfolk, Va., reported that the cases of acute catarrhal fever were of short duration and that the increase in admissions for this disease was due to the prevailing inclement weather.

Shore stations outside the continental limits of the United States reported 279 cases of these diseases for the quarter, as follows: Hawaii, 27; Panama Canal Zone, 99; Philippine Islands, 25; Guam, 11; Guantanamo Bay, Cuba, 9; China, 90; Samoa, 18.

Sixty-one cases of German measles were reported from the Naval Training Station, Norfolk, Va., and 87 cases from the Naval Training Station, San Diego, Calif., for the quarter. The latter station reported that a universally mild type of German measles was admitted from the recruit companies. In many instances the transitory rash was the sole diagnostic sign exhibited, the recruit being entirely free from subjective symptoms.

The Naval Training Station, San Diego, Calif., reported one case of chickenpox in October.

The receiving ship, Cavite, P. I., reported 1 case of paratyphoid fever in November and the receiving ship, San Francisco, Calif., 1 case of typhoid fever in December. No questionnaire has been received in the Bureau for either case, consequently information regarding prophylaxis, etc., is not available.

Deaths from cerebrospinal fever during the year 1934 occurred as follows:

Ship or station	Date of death	Length of service
G. R. H.—Naval Training Station, Norfolk, Va	June 9	1 month.
J. H. L.—On leave, U. S. S. *Humphreys*	July 31	7 months.
F. C. S.—Naval Training Station, Norfolk, Va	Aug. 13	1½ months.
F. P. P.—Naval Training Station, Norfolk, Va	Aug. 14	3 months.
F. G. F.—U. S. S. *Texas*	Oct. 7	5 months.
G. H.—U. S. S. *Relief*	Oct. 11	4 years.
A. F. O.—Naval Training Station, Norfolk, Va	Oct. 15	1 month.
B. B. S.—U. S. S. *Idaho*	Nov. 4	9 months.

In addition to the fatal cases listed above for the fourth quarter there were 5 nonfatal cases, as follows: 1 at the Naval Training Station, Norfolk, Va., in October; 1 at the Hospital Corps School, Portsmouth, Va., in November; 1 on board the U. S. S. *Humphreys* in December; and 2 on board the U. S. S. *Lexington*, 1 in November and 1 in December.

The admission rate, all causes, forces afloat, was 586 per 1,000 per annum, a 45 percent increase when compared with the fourth quarter of 1933. The median rate for the corresponding quarter of the preceding 5 years is 524.

There were 2,740 cases of acute respiratory diseases reported by all ships of the Navy during October, November, and December, 1934, indicating a 49 percent increase from the number of cases notified for the preceding quarter and an incidence much higher than expectancy for this season of the year.

The ships notifying 40 or more cases of acute respiratory infections distributed over the quarter are as follows:

Ship	October	November	December	Ship	October	November	December
U. S. S. Argonne	10	35	9	U. S. S. Mississippi	59	19	5
U. S. S. Arizona	8	35	4	U. S. S. New Mexico	7	58	10
U. S. S. Augusta	50	6	6	U. S. S. New Orleans	7	41	20
U. S. S. California	84	11	10	U. S. S. Ranger	5	9	37
U. S. S. Colorado	13	34	3	U. S. S. Texas	5	95	9
U. S. S. Detroit	4	37	2	U. S. S. West Virginia	14	36	9
U. S. S. Idaho	31	17	14	U. S. S. Whitney	5	46	5
U. S. S. Langley	8	52	4				
U. S. S. Lexington	24	109	36	Total	334	789	188
U. S. S. Marblehead	0	149	5				

A moderately severe case of typhoid fever was readmitted to the U. S. S. *Relief* from the U. S. S. *Houston* on October 17, 1934. A complete course of prophylaxis had been administered in August, 1931, but no information is available as to type of vaccine used. One case of paratyphoid fever was admitted on board the U. S. S. *Sacramento* in October.

Four cases of chickenpox were reported in December, one each on the U. S. S. *Idaho*, U. S. S. *Langley*, U. S. S. *Ranger*, and U. S. S. *West Virginia*.

TABLE 1.—*Summary of morbidity in the U. S. Navy for the quarter ended Dec. 31, 1934*

Average strength	Forces afloat, 71,878		Forces ashore, 38,612		Entire Navy 110,490	
	Admissions	Rate per 1,000	Admissions	Rate per 1,000	Admissions	Rate per 1,000
All causes	10,538	586.44	6,633	687.14	17,171	621.63
Diseases only	9,431	524.83	5,887	609.86	15,318	554.55
Injuries	961	53.48	742	76.87	1,703	61.65
Poisonings	146	8.12	4	.41	150	5.43
Communicable diseases transmissible by oral and nasal discharges (class VIII):						
(A)	383	21.31	336	34.81	719	26.03
(B)	2,873	159.88	1,856	192.27	4,729	171.20
Venereal diseases	2,500	139.12	653	67.65	3,153	114.15

TABLE 2.—*Deaths reported, entire Navy, during the quarter ended Dec. 31, 1934*

		Navy			Marine Corps		Nurse corps	Total
		Officers	Midshipmen	Men	Officers	Men		
Average strength		9,537	1,703	81,606	1,176	16,135	333	110,490
Cause—Disease								
Primary	Secondary or contributory							
Abscess, liver	None			1				1
Angina pectoris	do				1			1
Arteriosclerosis, coronary artery.	Thrombosis, coronary artery.			1				1
Carcinoma:								
Back	Pneumonia, broncho-	1						1
Colon	None	1						1
Liver	do			1				1
Tongue and lymph-glands of neck.	do			1				1
Cerebrospinal fever	do			4				4
Cirrhosis, liver	Hemorrhage, stomach	1						1
Hemorrhage, cerebral	Pneumonia, broncho-	1						1
Do	Pneumonia, lobar			1				1
Hodgkin's disease	None	1						1
Influenza	Nephritis, acute			1				1
Myocarditis, chronic	Exhaustion from over-exertion.					1		1
Nephritis, acute	None	1						1
Do	Status lymphaticus			1				1
Nephritis, chronic	None	1						1
Obstruction, intestinal, from spastic or paralytic causes.	Ulcer, duodenum	1						1
Pancreatitis, acute	Myocarditis, chronic					1		1
Pneumonia, lobar	None			2		1		3
Septicemia	do			1				1
Thrombosis, coronary artery.	Aortitis			1				1
Do	Arteriosclerosis, general	1						1
Tuberculosis, pulmonary, chronic.	None			1				1
Do	Hemorrhage, pulmonary			1				1
Do	Tuberculosis, larynx			1				1
Tuberculosis, meningeal	None			1				1
Total for disease		9	0	19	1	3	0	32
Injuries and poisonings								
Caisson disease	None			1				1
Drowning	do			6				6
Do	Injuries, multiple, extreme.					1		1
Dislocation, vertebra, cervical.	None					1		1
Exhaustion from over-exertion.	Dilatation, cardiac, acute.	1						1
Fracture, compound, skull	None			3		1		4
Fracture, simple, skull	Hemorrhage, traumatic subdural.			1				1
Do	Intracranial injury					1		1
Fracture, simple, vertebra, cervical.	None	1				1		2
Injuries, multiple, extreme	do	1		9		3		13
Intracranial injury	do	1				1		2
Do	Pneumonia, lobar			1		1		2
Strangulation, neck	None			2				2
Wound, gunshot, abdomen.	Hemorrhage, abdomen					1		1
Wound, gunshot, head	None	3				1		4
Wound, incised, neck	Dementia praecox	1						1
Total for injuries and poisoning.		8	0	23	0	12	0	43
Grand total		17	0	42	1	15	0	75
Annual death rate per 1,000:								
All causes		7.13		2.06	3.40	3.72		2.72
Disease only		3.77		.93	3.40	.74		1.16
Drowning				.29				.22
Poisonings								
Other injuries		3.36		.83		2.97		1.34

ADMISSIONS FOR INJURIES AND POISONINGS, FOURTH QUARTER, 1934

The following table, indicating the frequency of occurrence of accidental injuries and poisonings in the Navy during the fourth quarter 1934, is based upon all form F cards covering admission in those months which have reached the Bureau:

	Admissions, October, November, and December 1934	Admission rate per 100,000, per annum	Admission rate per 100,000, year 1933
INJURIES			
Connected with work or drill	607	2,198	2,237
Occurring within command but not associated with work	673	2,436	1,692
Incurred on leave or liberty or while absent without leave	423	1,531	1,757
All injuries	1,703	6,165	5,686
POISONINGS			
Industrial poisoning	4	14	26
Occurring within command but not connected with work	142	514	191
Associated with leave, liberty, or absence without leave	4	14	19
Poisonings, all forms	150	543	236
Total injuries and poisonings	1,853	6,708	5,922

Percentage relationships

	Occurring within command				Occurring outside command (leave, liberty, or A. W. O. L.)	
	Connected with the performance of work, drill, etc.		Not connected with work or prescribed duty			
	October, November, and December 1934	Year 1933	October, November, and December 1934	Year 1933	October, November, and December 1934	Year 1933
Percent of all injuries	35.6	39.3	39.5	29.8	24.8	30.9
Percent of all poisonings	2.7	10.9	94.6	80.9	2.7	8.2
Percent of total admissions, injury, and poisoning titles	33.0	38.2	44.0	31.8	23.0	30.0

Poisoning by a narcotic drug or by ethel alcohol is recorded under the title "Drug addiction" or "Alcoholism", as the case may be. Such cases are not included in the above figures.

STATISTICS RELATIVE TO MENTAL AND PHYSICAL QUALIFICATIONS OF RECRUITS

The following statistics were taken from monthly sanitary reports submitted by naval training stations:

October, November, and December 1934	U. S. Naval Training Station	
	Norfolk, Va.	San Diego, Calif.
Recruits received during the period	2,010	2,002
Recruits appearing before board of medical survey	7	0
Recruits recommended for discharge from the service	7	0
Recruits discharged by reason of medical survey	5	0
Recruits held over pending further observation	0	0
Recruits transferred to the hospital for treatment, operation, or further observation for conditions existing prior to enlistment	14	49

The following table was prepared from reports of medical surveys in which disabilities or disease causing the surveys were noted as existing prior to enlistment. With certain diseases, survey followed enlistment so rapidly that it would seem that many might have been eliminated in the recruiting office.

Cause of survey	Number of surveys	Cause of survey	Number of surveys
Abscess, periapical	1	Effort syndrome	1
Abscess, acquired, teeth	2	Enuresis	7
Arterial hypertension	1	Epilepsy	1
Arthritis, acute, sacro-iliac	1	Flat foot	9
Arthritis, chronic, right wrist	1	Fracture, simple, multiple, right astragulus and scaphoid, old	1
Cardiac disorder, functional	1	Gonococcus infection, epididymis	1
Caries, teeth	1	Hypothyroidism	1
Cicatrix, skin	2	Nephritis, chronic	1
Conjunctivitis, follicular	1	Otitis, media, chronic	5
Constitutional psychopathic inferiority, without psychosis	5	Pes cavus	2
Constitutional psychopathic state, emotional instability	2	Spasm, habit	1
Deafness, unilateral	1	Sprain, left knee (recurrent)	1
Deformity, acquired, right hand	1	Syphilis	2
Dementia praecox	1	Union of fracture, faulty	1
Diabetes mellitus	1	Valvular heart disease, combined lesions, aortic and mitral	1
Dislocation, articular cartilage, right knee (football)	1	Valvular heart disease, mitral insufficiency	1

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COMMANDER LOUIS H. RODDIS
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NAVY DEPARTMENT,
Washington, March 20, 1907.

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

TRUMAN H. NEWBERRY,
Acting Secretary.

Owing to the exhaustion of certain numbers of the BULLETIN and the frequent demands from libraries, etc., for copies to complete their files, the return of any of the following issues will be greatly appreciated:

Volume IX, no. 1, January 1915.
Volume X, no. 2, April 1916.
Volume XI, no. 3, July 1917.
Volume XII, no. 1, January 1918.
Volume XII, no. 3, July 1918.

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PREFACE

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

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

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

In order that each service contributor may receive due credit for his efforts in preparing matter for the BULLETIN of distinct originality and special merit, the Surgeon General of the Navy will send a letter of 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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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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U. S. NAVAL MEDICAL BULLETIN

VOL. XXXIII OCTOBER 1935 No. 4

SPECIAL ARTICLES

VITAMINS AND THE EVOLUTION OF THE NAVY RATION

By W. L. MANN, Captain, Medical Corps, United States Navy

Without vitamins in our food, we die.—During the present generation medical science has discovered that a diet composed of the correct proportions and proper quantities of fat, proteins, carbohydrate, and mineral salts, yet lacking in vitamins, causes various diseases and even death.

In fact, an adequate ration, balanced in every respect, yet lacking in vitamins will, not infrequently, cause death to ensue more promptly than a starvation diet composed of scarcely any food, yet containing a concentrated supply of vitamins.

The quantity of vitamins needed in the day's ration to preserve health is almost infinitesimal. Vitamins exist in minute proportions and the daily supply is so small that it can be placed under one's thumb-nail.

The daily requirement of one of the vitamins has been placed at one six-thousandths of a milligram (0.00000257 grain), an amount that can be held easily by the point of a small pin.

The study of ship-board diseases, particularly in relation to the evolution of the naval ration, played a prominent role in the elucidation of facts leading to the discovery of vitamins. It is most interesting to review some of the naval customs and practices in the issue of rations with high vitamin content. In this résumé one can almost sense that naval surgeons of the days of sailing ships were on the verge of the discovery of the existence of vitamins and of the recognition of the fact that these substances were essential for the maintenance of health of the sea-going personnel.

VITAMIN-RATIONS OF THE ANCIENT NAVIES

From the earliest times, the cruising radii of the sailing vessels, a type of ship that does not require fuel for propulsion, were limited

almost entirely by the health of the crews. Health conditions were in turn largely dependent upon the relative quantity of vitamins in the diet, thus preventing such ship-board diseases as scurvy, beriberi, etc.

Even the success of ancient Phoenicians in making long voyages may be attributed to the practice of feeding a ration of high vitamin content to their sailors. As a general procedure, the ships of this maritime power did not make long uninterrupted cruises, but frequently put into port and remained long enough enroute for the crews to disembark, and then plant and harvest crops of fresh foodstuffs. In their trip around Africa and return, they resorted to this practice which enabled them to feed the required amount of vitamins and accordingly preserve the health of the personnel. Whether the Phoenician authorities recognized the necessity of fresh food in the seaman's diet, or whether the interruption in the voyages were demanded primarily for other reasons, such as ship repairing, it is impossible to state.

As we know today, bread made from flour has less vitamin content than bread made of whole wheat. The *panis nautious* (sea-bread), used in the ancient Roman navy, was perhaps a bread of high vitamin content used to prevent dietetic diseases aboard ship, since we have records that the military leaders ashore issued whole wheat and prohibited the use of flour, i. e., wheat deprived of much of the vitamin. In Caesar's expeditions into Gaul, wheat, and not flour, was fed to his troops.

The Roman forces carried their canteens filled with vinegar, which was diluted with water and drunk, thus serving the combined purpose of partial sterilization and supplying additional vitamins.

There are more than three types of vitamins, but for the purpose of this discussion vitamins A, B, and C, only, will be considered.

Name	Source	Disorder caused by the lack of—
Vitamin A.....	Green vegetables, liver, butter, etc.	Eye disorders; lowers resistance to various infections.
Vitamin B.....	Husks of grain, fresh vegetables, yeast, etc.	Beriberi; swollen limbs, and body, paralysis of legs, etc.
Vitamin C.....	Lemons, oranges, grain, etc..	Scurvy; pallor, swollen and bleeding gums, shortness of breath, etc.

VITAMIN A IN THE NAVY RATION

Lack of vitamin A causes a disorder among sailors which was successfully treated centuries ago by supplying the missing vitamin. Fresh vegetables contain an abundance of vitamins, and "green trade" (an obsolete term for these articles) were used by

a naval surgeon in 1696 to cure and prevent dietetic disorders on his ship.

Ulceration of the eyes (xerophthalmia) and night blindness (nyctalopia) were prone to occur among the crews of sailing vessels, especially when becalmed in tropical waters. Such ocular disturbances are due to a lack of vitamin A, and these conditions were treated by the Chinese from the earliest times with extracts of liver, a gland particularly rich in this substance. The livers of codfish are used by modern physicians as a primary source of supply for this vitamin.

When grains and seed are allowed to sprout (germinate), the process markedly increases the vitamin content. This has been discovered recently, but our predecessors aboard ship, over 100 years ago, observed that the curative powers of certain food stuffs were considerably enhanced by germination. In 1807, a medical officer of the United States Navy used a ration of "peas soaked in water and allowed to vegetate" for the treatment of certain dietetic deficiency diseases aboard.

VITAMIN B IN THE NAVY RATION

Ships beriberi, caused by the deprivation of vitamin B, was a common disease in the days of sailing ships, and since it was so often associated with scurvy, it is highly probable that scurvy and beriberi were not always sharply differentiated by the observer of earlier periods. Hence in reading of the epidemics of scurvy afloat we can assume that some undiagnosed cases of beriberi were present.

However, the sea-going personnel were one of the first to successfully combat beriberi by a change in diet. Admiral Takaki, of the Japanese Navy, although not of medical training yet a practical observer, was able to reduce the incidence of this disease among his seamen from 32 percent to a negligible rate, and the results of the change in the ration of the Japanese Navy were, in a measure, responsible for the subsequent conception of vitamins.

VITAMIN C IN THE NAVY RATION

The lack of vitamin C in the sailors' ration caused scurvy to be the dreaded scourge of the days of the sailing ships, "the calamity of sailors."

When Vasco de Gama found passage to the East Indies by the way of the Cape of Good Hope, in 1497, "one hundred of his one hundred sixty men died of scurvy."

On Commodore Anson's trip around the world (1740–44) 19 out of every 20 men failed to return to England, the majority dying of

scurvy. One of his vessels, the *Gloucester*, only a few months out, buried 292 men who died from scurvy, and had only 82 surviving sailors, many of whom were sick and died before the completion of the voyage.

The British Channel Fleet, in a voyage of 10 weeks, had 2,400 cases of this malady.

When the Spanish and French Fleets arrived at the West Indies, they landed 1,000 sick on arrival, and during their 40-day stay, buried over 1,000 others.

One of the last expeditions of the British Navy in which scurvy occurred was in 1824, when the *Larne* lost one-half her crew, and the *Sophie*, one-quarter.

The death rate in the early American Navy was also high. On the frigate *Macedonian*, from April 2 to September 5, 1827, 101 men of 376 died, including Dr. Cadle, the ship's surgeon.

The forces afloat were likewise the first to use vitamin C to cure dietetic diseases. About 400 years ago the Frenchman, Jacques Cartier, came sailing up the St. Lawrence River with his ship full of sick sailors. Twenty-six of them had already died of scurvy, and those remaining were saved by drinking a tea brewed from pine needles—the first real discovery to indicate the existence of vitamin C—as someone has stated.

As early as 1564, an officer of the Dutch fleet, B. Ronsseus, discovered that the use of fruit juices would prevent and cure scurvy. This finding was further proved in the British Navy by Sir Richard Hawkins (1593), Commodore Lancaster (1600), Felix Plater (1608), and Surgeon Woodall (in his Surgeon's Mate, published in 1636).

Attention is invited to the fact that centuries elapsed from the dates when vitamin C was first used to cure scurvy until the last epidemics in the modern navies. This unusual delay in utilizing these findings was not due to any lack of recommendations made by the naval medical corps. The eminent naval surgeon, James Lind, in 1753, recommended the compulsory use of lime juice in the British Navy, and it then required over 40 years more, or about two centuries after the original discovery, for the Admiralty to place in effect this recommendation and eradicate scurvy from the British forces.

The British Admiralty, in 1796, was finally persuaded, by the insistency of such naval medical officers as Lind, Trotter, and Blane, to supplement the navy ration with 1 ounce of lemon juice per man. However, a classical experiment was first conducted to test the efficacy of the proposed change in the bluejacket's ration.

With this object in view, the *Suffolk*, a flagship of 74 guns, made a voyage of 19 weeks to the East Indies, with orders not to touch

at any port. During this trip, lemon juice was issued to each man daily in order to test its antiscorbutic properties. Upon arrival at Madras, not a single case of scurvy was on the sick list, a most phenomenal result for those days. The crew was able to exist nearly 5 months away from a fresh food supply and arrived in excellent health, a condition rendered possible by the simple procedure of increasing the quantity of vitamin C in the daily dietary.

The loblolly boy, the naval hospital corps man of sailing ship days, owes his vernacular designation of "loblolly" to a form of gruel made from maize and used in dietetic therapy of naval patients suffering from avitaminoses. In Colloquia Maritima, published in 1688, a dietary of "husked hominy and loblolly" was recommended as a curative for sailors suffering from "Calentures, scarbotes (i. e., scurvy) and the like contagious diseases." Note the term "contagious", which may appear, in the light of our newer knowledge of nutrition, an erroneous observation. However, based upon personal experience in treating disorders due to faulty diet, I feel prompted to suggest that this old sea-going physician was partly correct in his contention that an infectious element may play a role in causing symptoms of food deficiency diseases to become manifested.

In some instances at least, an infectious disorder, such as influenza and dysentery, acting upon a crew suffering from latent and unrecognizable symptoms of scurvy and beriberi, may cause such a sudden and wholesale aggravation of the symptoms of the latter diseases that the outbreak would resemble an epidemic.

It has been my experience to witness just such epidemical outbreaks of food deficiency diseases apparently precipitated by some casual disease-producing organism lowering the constitutional resistance.

SUNLIGHT AND VITAMINS

Admiral Nelson appreciated the military importance of health and, upon the advice of the ship's surgeon, to maintain his personal health he walked 6 to 7 hours daily on the deck exposed to the direct or indirect irradiation of the sun's rays. There are other allusions in the records of Naval Medicine that tempts one to suggest that the value of the solar rays in the food deficiency diseases was almost anticipated by the earlier generations, or rather they were inclined to attribute to the concomitant conditions of fresh air, the benefits that were actually produced by sunlight, a fallacious deduction which one can detect in some of the claims of modern "fresh air" propagandists.

In this connection it may be mentioned that fresh air *per se* assumes less importance than that once attributed to it. For example,

it has been reported that the sea-going personnel of certain fishing trawlers habitually batten down the sleeping compartments at night and in the morning the air is so vitiated and the oxygen content is so lowered that a candle will not burn in this air. Yet, these fishermen live to ripe, healthy, old age, in spite of sleeping in this extreme lack of fresh air where the oxygen content has been lowered from 20.94 percent to less than 16 percent. Sunlight is more important to the maintenance of the proper equilibrium of the human mechanism than some of the refinements of modern ventilation, a statement not intended to depreciate the value of the latter.

The action of sunlight—ultra-violet or actinic rays—bears a very direct relation to the process of vitamin formation, and in fact, when certain food constituents are exposed to the short waves of the spectrum, vitamins are produced.

For example, when the ultra-violet rays are directed on certain chemical substances (sterols), the vitamin activity is so increased that the radiated substance possesses 100,000 times the vitamin value as the same weight of cod liver oil.

I have studied, in Haiti, hundreds of cases of a dietetic deficiency disease which were subsequently proved to have been developed by the deprivation of the sun's rays. Not a single case of this disease developed in inmates of this series of institutions who had access to sunlight. yet hundreds of fatal cases occurred in inmates subsisting on the same diet but were denied the benefits of the actinic rays of the sun.

It is rather interesting to note that this clinical observation was made synchronously (1921) with the laboratory demonstration of the effect of ultra-violet rays on the development of vitamins.

CONCLUSION

In conclusion, it may be stated that the modern conception of the value of vitamins is based largely upon the original discoveries of our predecessors in naval medicine in treating sailors suffering from affections due to faulty diet.

If the naval medical officers of today could be as successful in the eradication of the respiratory diseases, such as coughs, colds, pneumonia, flu, etc., as the earlier naval medical officers proved eventually to be in the eradication of dietetic disorders, we would leave a cherished heritage to posterity. Unfortunately, the respiratory group of diseases have, so far, yielded very little to the medical efforts to limit their spread or to decrease their severity.

Modern naval medicine was as helpless in combating the great pandemic of influenza in 1917, as the earlier medical officers were in coping with some of their epidemics of dietic diseases on sailing

ships before the necessity of adding fruit juices and fresh vegetables to the bluejacket's ration was discovered.

Recently, a change has been made in the ration of the United States Navy in order to maintain the naval dietary abreast of the progress of modern dietetics and to include foodstuffs of high vitamin content. This fact indicates that modern naval officials are inclined more readily to approve scientific recommendations for improvements.

BENIGN LYMPHOCYTIC CHORIOMENINGITIS (ACUTE ASEPTIC MENINGITIS)—A NEW DISEASE ENTITY[1,2]

By PAUL F. DICKENS, Lieutenant Commander, Medical Corps, United States Navy, and CHARLES ARMSTRONG, Surgeon, United States Public Health Service

In 1926, Wallgreen (1) in Germany described a self-limited benign meningitis under the title Acute "Aseptic" Meningitis and since that time many confirmatory articles have appeared in the European literature. After the publication in 1929 by Viets and Watts (2) of three cases in this country, interest in the syndrome became more widespread and cases were reported by Gager (3) 1930, Dickens (4) 1932, Bloedorn (5) 1932, and again by Viets and Watts (2a) 1934.

In August of 1934 Armstrong (6) called attention to a virus which he had recovered and which differed from any with which he was then familiar. It was encountered in the course of virus transmission work on monkeys, and it is uncertain whether the infection originated independently in the animals used or was inoculated with material from a human source. Monkeys seem to be usually susceptible as are mice and guinea pigs, the infection producing in monkeys as in man a uniform symptom-complex. On the fourth to the eighth day after inoculation with the virus the temperature rises 104°–105° F., continuing at this elevation for 3 to 10 days. Defervescence is by lysis. The blood leucocyte count ranges from 10,000 to 19,000 per cubic millimeter. The cerebrospinal fluid is clear or at most slightly hazy, is under slight increase in pressure, and contains from 150 to 3,000 cells per cubic millimeter, these being almost entirely lymphocytes. (The average normal cerebrospinal fluid cell count in a series of control monkeys was 19 lymphocytes). The chemistry of the cerebrospinal fluid does not deviate from normal range. In the series of sick monkeys the sugar averaged 61, sodium chloride 891, and the urea nitrogen 17.6 mgm percent. (In the series of 10 control monkeys the average content per 100 cubic centi-

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[2] All virus experiments at National Institute of Health, other laboratory and clinical work done at the U. S. Naval Medical School and Gallinger Memorial Hospital, Washington, D. C.

meters of cerebrospinal fluid was sugar 56, sodium chloride 812, and urea nitrogen 16.8 mgm percent). The sick animal characteristically sits quietly with head drooping and eyes closed, but is easily aroused: and if disturbed sufficiently to make it move the motions are slow and hesitating, as if the muscles were stiff. Armstrong (16) stated that the human disease most nearly resembling this disease in monkeys is, perhaps, the so-called "lymphocytic or asceptic meningitis" described by Wallgreen, Viets and Watts, Dickens, Bloedorn, and others, and demonstrated protective antibodies in the serum of a recovered case (7). Traub (8–9) recently recovered a virus from white mice which appeared to resemble closely the virus isolated by Armstrong. Soon thereafter (May 2, 1935), Rivers and Scott (10) reported the isolation of a similar virus from two cases of meningitis, and stated that the serum from these cases protected animals from this virus. An exchange of protective sera was made with Traub in order that a serological comparison, and study of the two viruses could be made. At the same time Traub tested two strains of his virus against the National Institute of Health's immune monkey serum.

In a previous communication, we (11) published in June 1935 our protocol supporting the view of the identity of these three viruses and at the same time reached the conclusion, seemingly supported by animal experimentation, that these three viruses were immunologically identical. We also demonstrated that the serum from animals and man recovered from the disease protects animals from either the virus of Armstrong, Traub, or Rivers.

Summarizing the results of these tests, it is seen that serum from guinea pigs rendered immune to Traub's virus protected animals inoculated with Armstrong's virus, and that serum from monkeys rendered immune to Armstrong's virus protected animals inoculated with Traub's virus. The results of these two independent tests indicate that the Armstrong and Traub viruses are identical (serologically).

Rivers and Scott (10) have also isolated a virus from two human cases of meningitis which appears to be immunologically identical with the Armstrong virus. Mice were obtained from Rivers which had been rendered immune to his virus, together with mice from the same stock for normal controls, and Armstrong conducted tests in which these mice were inoculated with his virus. The results indicate the serological identity of the viruses.

Further confirmatory work was done by Rivers, using his virus against Armstrong's immune serum, Traub's immune serum, and Rivers' immune serum in tests on guinea pigs, inoculating the serum-virus mixtures subcutaneously. The results indicate the immunologi-

cal identity of the Armstrong virus, the Traub virus, and the Rivers' virus, and appear to prove that this virus is a definite specific entity.

It has often proved difficult to establish an etiologic diagnosis in the case of patients showing signs and symptoms of cerebrospinal involvement, especially when the cellular response in the cerebrospinal fluid is predominantly lymphocytic in character; and occasionally cases are met in which heretofore it has not been possible to detect any living etiologic agent. For that reason, the term acute aseptic meningitis has been proposed as most nearly descriptive. The purpose of this paper is to show that some if not all such cases represent a disease entity due to a filtrable virus.

The clinical picture of the disease is that of an infection of the upper respiratory tract, followed by meningeal symptoms which are ushered in by sudden onset with headache, nausea or vomiting, rise in temperature to 100° to 103° F., stiff neck, and usually a positive Kernig sign. There is no evidence of nerve involvement and other than noted above the neurological examination is negative. The disease runs a benign course for about 10 days to 2 weeks. The temperature declines by lysis, and recovery is complete without residuals of any kind. Four patients who have been followed for more than 3 years remain entirely well.

The cerebrospinal fluid is under slight increase in pressure and is clear or at the most slightly hazy. The cellular response is almost entirely lymphocytic—rarely do we find as many as 10 percent polymorphonuclear leucocytes in the fluid. The number of cells may range anywhere from 50 to 2,000, according to the severity of the attack. The chemistry of the cerebrospinal fluid is important in that the sugar, chlorides, and urea content will be found within normal range. The Kahn or Wassermann is negative and the colloidal gold curve will be in the meningitic zone and of low color change. No organism or clot can be demonstrated. Drainage of a few cubic centimeters of cerebrospinal fluid will usually relieve the headache and nausea, and quiet the patient. The white blood cell count may show a slight increase up to 9,000 or 11,000 with a fairly normal differential percentage. The fact that the ceresbrospinal fluid shows no tendency to clot and that the sugar and especially the chlorides remain within normal limits are most important diagnostic points definitely against tuberculous meningitis, with which the disease is at first often confused. The fact that no muscle weakness, or definite neurological signs are found helps to rule out encephalitis (of all types) and acute anterior poliomyelitis.

Case 1.—White woman; age 19; unmarried. First seen May 13, 1931, complaining of severe headache, more marked over the frontal region, nausea, vomiting, and pain in the epigastrium. She stated that for several days

previous to the onset of the acute symptoms she had had a cold, and that she had not felt well for about 2 weeks.

Examination: Temperature 100; pulse 92, and respiration 20. There was some slight tenderness over the frontal sinuses, which, however, transilluminated equally and well. The chest was clear to auscultation and percussion. The heart showed an occasional extra-systole. The abdomen was negative except for slight tenderness over the epigastric region.

Laboratory examination: Urine showed a slight trace of albumin; white blood count 6,000 differential: polymorphonuclear leucocytes 58 percent with 5 percent band forms, lymphocytes 36 percent and monocytes 1 percent. A provisional diagnosis of influenza was made.

Course: The following day the temperature rose to 102. She complained of severe headache. Examination revealed a well-marked rigidity of the neck, and a suggestive Kernig's sign. A spinal puncture was done with relief of the headache; 15 cc of clear fluid was obtained under no apparent increase in pressure. The cell count was 590, with 80 percent lymphocytes, and 20 percent polymorphonuclear leucocytes. Smears were negative for organisms. The Wassermann and colloidal gold tests on the spinal fluid were negative. Urea N. 10, sugar 60, and chlorides 712 mg per 100 cc of spinal fluid. On the third day the temperature was 99.4, but the rigidity of the neck was decidedly more marked, and there was retraction of the head. Nausea and vomiting continued. A second spinal tap was done and 30 cc of fluid obtained. The pressure was 18 mm Hg; cell count 3,200 with 96 percent lymphocytes and 4 percent polymorphonuclear leucocytes. Smears and cultures were negative for organisms. Sugar 60 mg, urea N. 12, chlorides 710 mg per 100 cc of spinal fluid. The impression at this time was "tuberculous meningitis." On the fourth day the headache decreased in severity; there was no vomiting, and little nausea. The rigidity of the neck and retraction of the head continued, and Kernig's sign was positive. Spinal puncture was repeated; 4 cc of fluid under 4 mm Hg pressure was removed. Cell count 2,900, with 86 percent lymphocytes, and 14 percent polymorphonuclear leucocytes. On the sixth day the temperature remained normal, and the patient showed improvement. From this time on there was steady improvement. The white cell count of the blood during the illness varied from 6,600 to 8,700; the differential count showing an average of 61 percent polymorphonuclear leucocytes, and 32 percent lymphocytes. On the thirteenth day of the illness the spinal fluid showed 38 cells of which 93 percent were lymphocytes, and 7 percent polymorphonuclear leucocytes. Sugar 75 mg, urea N. 15, and chlorides 730 mg per 100 cc of spinal fluid.

The patient made an uneventful recovery, and in 6 weeks was apparently well. At no time was there any evidence of cranial nerve involvement, or any other significant localizing neurological findings. She has been under observation since then, and has been free from symptoms.

On April 25, 1935, or 3 years and 11 months after the illness, blood serum was obtained from this patient and her serum protected mice against the virus of Armstrong.

Case 2.—White male, age 28 (reported by courtesy of Dr. Walter A. Bloedorn). The patient was first seen on April 2, 1934, at which time he complained of headache, nausea and vomiting, stiff muscles, and fever. He stated that 3 days before he was suddenly taken ill with severe headache, coryza, and fever.

Examination: The patient was a well-developed, somewhat obese male; he did not look toxic, or gravely ill. The only significant findings were: Stiffness of the neck, a positive Kernig's sign, and a temperature of 101° F.

Laboratory examination: Red blood cells 4,800,000; white blood cells 10,200; differential: Polymorphonuclear leucocytes 66 percent (divided as follows: Segmented 50 percent, bands 16 percent), lymphocytes 30 percent, monocytes 4 percent. Spinal fluid: cell count 1,260, almost exclusively lymphocytes (8 red blood cells and 2 polymorphonuclear leucocytes were seen); globulin positive; chlorides, estimated as sodium chloride, 690 mgm per 100 cc; sugar 60 mgm per 100 cc. Kahn and Wassermann were negative; colloidal gold curve 0011221100. Culture negative after 48 and 72 hours, and on the seventh day. Animal inoculation was negative for tuberculosis. Due to the sudden onset, absence of tuberculosis elsewhere in the body, and absence of paralysis and muscle weakness, together with the relief of the main symptoms and lowering of the body temperature by spinal puncture. Bloedorn made a tentative diagnosis of aseptic meningitis which was confirmed by laboratory findings. The illness lasted 1 week, was of a mild nature, and recovery was complete without residual manifestations. On April 8, 1935, or 1 year after the illness, blood serum was obtained from this patient, and his serum protected mice and monkeys from the virus of Armstrong.

Case 3.—White male; age 33. First seen October 28, 1931, complaining of a severe headache, more marked at the occiput, nausea, and general soreness of the muscles. He stated that 2 weeks previously he had had a severe cold which had cleared within a week, but which was followed by herpes labialis.

Examination: Temperature 102, pulse 88; respiration 20. Residuals of herpes noted about nose and lips. There was no rash or erythema. The throat was moderately inflamed; tonsils had been removed. There was some stiffness and tenderness of the neck. The posterior cervical lymph glands and inguinal glands were palpable. Lungs, heart, and abdomen normal; blood pressure 130/80.

Neurological: Bilateral Kernig and hyper-active knee kicks; ophthalmoscopic examination showed some blurring of the disk margins.

Laboratory: Urine negative; red blood count 4,850,000; hemoglobin 85 percent; white blood cell count 8,000; differential: Polmorphonuclear leucocytes 60 percent; lymphocytes 35 percent; monocytes 5 percent. Blood Kahn negative. The cerebrospinal fluid was under increased pressure. The cell count was 1,255 the first day of disease, gradually reduced to 120 the fourteenth day of the disease. The cells were all lymphocytes. The sugar, chlorides, and urea remained normal.

Smears and cultures from the fluid were negative for organisms. Animals inoculated and killed 5 weeks later showed no evidence of tuberculosis. There was no pellicle formation. Colloidal gold curve: 0011211000. X-ray of the head and chest negative for tumor, abscess, or tuberculosis.

Course: The spinal taps relieved the headaches, and upon two occasions the patient asked for the spinal tap to ease the pain. The treatment was essentially symptomatic, and nursing. The temperature the first 8 days ranged from 99.5 F. in the morning to 102° F. in the afternoon. On the eleventh day of the illness the temperature fell to normal and remained there. Recovery was without incident, and 6 weeks later the patient was apparently well. A check-up 2 months later showed the patient to be in good health. On April 2, 1935, or 3½ years after the illness, blood serum obtained from this patient protected mice from the National Institute of Health's strain of virus (Armstrong).

Case 4.—White woman, J. R., nurse, age 20. First seen March 15, 1935, at which time she complained of a cold, severe headache, nausea, and vomiting, disturbances in vision, and pain in the sinuses. She stated that she had an acute attack of sinusitis in January 1935.

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Examination: Temperature 100.8° F.; pulse 90; respiration 20. There was some blurring of the optic disks, and there was a positive Brudzinski sign together with a positive Kernig sign, otherwise the examination was essentially negative.

Laboratory: Urine, negative; red blood count, 4,500,000; hemoglobin, 85 percent; white blood cell count, 8,000. Differential: polymorphonucdear leucocytes, 69 percent; lymphocytes, 21 percent; monocytes, 10 percent. Blood Kahn, negative. Spinal fluid: cell count, 209, exclusively lymphocytes; no organisms noted in the smear; the pressure showed no significant increase, and the fluid was practically clear.

Course: Throughout the illness the main symptoms were headache, nausea, and vomiting. The temperature maintained a level of 100.8° F. for 3 days, dropped to normal for 1 day, and fluctuated between 99° and 100° F. for 3 more days before dropping to normal and remaining there. Spinal taps gave the patient relief early in the illness, but caused some reaction in the form of headache later on in the course of the disease. On the fifth day the blood examination was as follows: Red blood cell count, 4,500,000; hemoglobin, 85 percent; white blood cell count, 9,500. Differential: Polymorphonuclear leucocytes, 44 percent; band forms, 5 percent; eosinophiles, 3 percent; lymphocytes, 41 percent; monocytes, 7 percent. Blood chemistry: Urea 12, sugar 91, and chlorides 675 mg percent. Spinal fluid on the third day of the illness: Cell count 409, exclusively lymphocytes; sugar 60, and chlorides 775 mg percent; no organisms could be found by smear, and cultures of the fluid were negative. Kahn and Wassermann negative, and the colloidal gold curve was 000322221. On the tenth day of the illness the spinal fluid cell count was 22, all lymphocytes; there was no pellicle, or clot formaton in the fluid. On the twenty-first day the chloride content of the spinal fluid was 775 mg percent, and the colloidal gold curve was 0000000000. The blood counts were essentially normal. The treatment other than the spinal taps was essentially symptomatic, and nursing. The patient made a slow and uneventful recovery, and 1 month later was feeling well.

On March 24, 1935, or on the tenth day of her illness, blood serum obtained from this patient did not protect animals inoculated with the virus of Armstrong; however, on May 15, 1935, or 2 months after the onset of the illness, her blood serum did protect the animals inoculated with the virus of Armstrong.

It will be noted that in the human, as in the experimental animals, the blood serum does not have protective power in preventing the disease until after the second week of the illness. This case is important in that the serum was not protective early in the disease, but became definitely protective after the illness, probably indicating definite immunity.

It is believed that these are important observations in that they seem to prove that we are dealing with a new disease entity caused by a virus, that was independently isolated by Armstrong and Lillie, and Traub and Rivers, and that the serum of patients recovered from this disease protects animals against this virus.

SUMMARY

(1) A symptom-complex of headache, fever, meningeal irritation, cerebrospinal fluid under increased pressure, with an increase in cells

(with a lymphocytic response dominant) above 50, coupled with a normal chloride, sugar, and urea content in the cerebrospinal fluid, and a negative spinal fluid Wassermann, is a clinical entity, which has previously been designated in man as *acute aseptic meningitis.*[3]

(2) The virus of Armstrong produces a symptom-complex in monkeys similar to the above.

(3) The blood serum of patients recovered from the disease protects animals from the virus of Armstrong. (National Institute of Health strain.)

(4) This disease occurs sporadically in man, and has been transferred experimentally to animals.

(5) Traub has isolated a virus from white mice and Rivers and Scott have isolated a virus from human patients which are serologically identical with the National Institute of Health strain of the Armstrong virus.

(6) Cases reported in this paper, and by Dickens (4) and Armstrong (6) cover a large geographical area, having their origin in California, Maryland, District of Columbia, Illinois, Ohio, and Virginia.

CONCLUSIONS

(1) The symptom-complex is a disease entity.

(2) This condition by priority should be designated "acute aseptic meningitis" (7, 8), but in view of the recent advance in the knowledge of its etiology, this designation is a misnomer, and we suggest the designation "acute lymphocytic choriomeningitis" as a more accurate designation.

(3) The etiological agent is a filtrable virus first described by Armstrong (6).

(4) The blood serum of patients recovered from acute aseptic meningitis protects animals from the virus. This may be used to confirm the diagnosis.

(5) Monkeys, mice, and guinea pigs are susceptible to the virus and it is conceivable that a reservoir for its disease may exist in animals.

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[3] Since the ailment here considered is caused by a virus the "aseptic" is a misnomer and we consequently prefer to denote the condition by the term (6–11) as benign lymphoocytic choriomeningitis.

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CAISSON DISEASE AND ITS RELATION TO TISSUE SATURATION WITH NITROGEN[1] [2]

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Since the sixteenth century men have been working under increased air pressure either in caissons or in diving suits. Men so exposed have been subject to a malady peculiar to pressure workers called caisson disease or "bends."

The cause of caisson disease was shown by Paul Bert (1878) to be due to the fact that excess gas, chiefly nitrogen, which goes into solution in the blood and tissues during exposure to compressed air, is liberated in the form of bubbles on too rapid decompression and produces local or general blockage of the circulation. All subsequent investigations by Heller, Mager and von Schrötter (1900), Hill and

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McLeod (1903) (1906), Hill and Greenwood (1906), and Boycott, Damant, and Haldane (1908) have shown this to be true.

As pointed out by Haldane (1922), the blood in the lungs is exposed to a greatly increased partial pressure of nitrogen and oxygen, and thus takes up an extra amount of nitrogen and oxygen proportional to the increased pressure. The extra oxygen disappears at once when the blood reaches the tissues but the extra nitrogen does not disappear, and gradually saturates the whole of the tissues until they are charged with nitrogen at the partial pressure existing in the air breathed. When the external atmosphere is reduced to normal, the internal partial pressure of nitrogen is, of course, far above the atmospheric pressure. The blood and tissues are therefore supersaturated with nitrogen and bubbles begin to form. These bubbles consist principally of nitrogen, but of course take up a little oxygen and carbon dioxide from the surrounding blood and tissue liquids. The bubbles are formed not merely in the blood but also in the surrounding tissues.

The symptoms of caisson disease vary according to the location of the bubbles. The multiplicity of possible symptoms preclude their enumeration but a general classification includes the following: Skin involvement—pruritis, rash, and mottling; phenomena in the trunk of the body—girdle pain, chest pressure, etc., myalgia, arthralgia, monoplegia, paraplegia, vertigo, aphasia, and asphyxia. These conditions may be very mild in nature or so severe as to lead to death within a few minutes. Caisson disease may develop immediately after reaching the surface or may be delayed for hours or even days, although usually it appears within 6 hours.

Early recompression is the treatment for all cases of caisson disease. If symptoms recur following the first recompression further treatment by air pressure is indicated. The breathing of oxygen has been recommended as beneficial, and sometimes symptomatic treatment is necessary. The milder symptoms may clear up within a few hours without treatment or paralysis may remain throughout life despite all treatment.

CAISSON DISEASE CASES

All the cases of caisson disease to be presented in this paper occurred during the course of 2,143 experimental dives which were made over a period of 3 years at the experimental diving unit, Navy Yard, Washington, D. C. These dives were made in the interest of submarine escape, to determine how long a group of subjects could remain at a given depth and come to the surface without stops for decompression and not develop caisson disease. The actual decompression time varied from 1.1 minutes at 100 feet to 2.2 minutes at

200 feet, i. e., the time necessary to reduce the air pressure in the diving tank, which is comparable to the time required for an actual ascent from a sunken submarine at these depths. The depths at which experiments were conducted were 100, 150, 167, 185, and 200 feet.

At a depth of 100 feet, the first case of caisson disease occurred after an exposure of 34 minutes although the exposure was carried to 52½ minutes before terminating the 100-foot experiments. At 150 feet, the first case of caisson disease occurred following the 18½-minute exposure while the terminating exposure was 28 minutes. At 167 feet, the first case of caisson disease was noted following a 17½-minute exposure and the series was terminated following an exposure of 22½ minutes. The first case of caisson disease in the 185-foot series occurred following an exposure of 12½ minutes and the series was terminated following the 13½-minute exposure. In the 200-foot series, the first case of caisson disease occurred following the 13-minute exposure while the terminating exposure was 14 minutes.

The men were watched carefully for the first indications of trouble and thus the caisson disease encountered during the course of these experiments was early diagnosed and given the proper treatment.

All of the cases of caisson disease are presented in table 1 together with the facts concerning the depth and time of exposure, the decompression, the type and onset of symptoms, and the recompression treatment.

Some of the cases seemed to us to be worthy of special mention either because of their unusual character or their severity and they are presented below, individually, and in greater detail than in table 1.

Case no. 11.—Depth, 100 feet; exposure, 48 minutes; decompression time, 1.5 minutes. Ten minutes after reaching the surface from an experimental dive, the subject experienced a sudden sharp pain in the right lumbar region which immediately radiated to the epigastrium causing the subject to double over. (This is the only subject having the symptoms which led to naming the disease "bends.") Mild respiratory embarrassment was also experienced. The subject was placed in the recompression chamber and pressure raised to 60 pounds with complete relief, and then was decompressed for 300 minutes. Four hours after completion of recompression symptoms returned in the left leg and increased in severity sufficient to necessitate a second recompression 9 hours after completion of the first, following which he was completely relieved.

Case no. 14.—Depth, 150 feet, exposure, 18½ minutes; decompression time, 1.7 minutes. The subject experienced stiffness and itching of the back of neck 5 minutes after reaching the surface. Examination disclosed a red mottled rash over the entire area. He was immediately placed in the recompression chamber and the pressure raised to 80 pounds, the rash disappearing at 20 pounds. Decompression occupied 60 minutes.

Shortly after completing the recompression, the subject noticed itching of the right shoulder and periodically recurring muscular spasms in the right knee. These symptoms persisted, and it was necessary after 8½ hours, because of weakness and loss of function of the right leg to give a second recompression. The pressure was raised to 100 pounds and the subject was decompressed over a period of 164 minutes with symptomatic relief. Neurological examination on the following day, however, revealed some residual involvement of the right motor tract of the spinal cord which cleared during a short hospitalization.

Case no. 30.—Depth, 150 feet; exposure, 27 minutes; decompression time, 2 minutes. About one-half hour after completing experimental dive, the subject felt a sharp severe pain in the anterior chest and a dull headache in the frontal-parietal region. He was recompressed to 80 pounds and decompressed over a period of 310 minutes with gradual loss of all symptoms except a marked tiredness.

Twenty-four hours later, he felt very tired and stiff, and 72 hours later continued to have stiffness of legs and a number of ecchymotic areas appeared over the lower legs. He was transferred to United States Naval Hospital, Washington, D. C., where the neurological examination showed the following findings: "Pupils equal, limited in their reaction to light, some hippus present. Left pupil is almost pear shaped, right somewhat irregular but less so than the left. Both react to accommodation. Upon rapidly repeating several times the more difficult test phrases, there is a strong suggestion of slurring speech. All the tension reflexes are markedly diminished except the supinator longus. The triceps jerks are not elicited. All tendon reflexes are equal on both sides. The abdominal and cremasteric reflexes are totally absent. The plantar reflex is almost absent. No Babinsky, the deep pressure sense to pain over the tendo-Achilles is practically absent. The pressure-pain sense over the testicles is practically absent. There is more than normal swaying in the Rhomberg posture, the patient at one time almost falling. The usual coordination tests are fairly performed by the upper extremities; there is more than normal difficulty, however, in performing these by the lower extremities. There is a subjective sense of numbness and weakness in both legs from the knees down and the patient states that he has a feeling that his knees are going to "give away" while walking. The gait is a sort of steppage-gait, but is not actually ataxic. Examination, otherwise, negative.

Spinal puncture: Fluid clear. Laboratory report: Kahn and Wasserman negative. Globulin negative. Cell count 3. Sugar 71. Chlorides 710. Colloidal gold curve negative.

Following the spinal puncture there was a remarkable recovery with almost complete loss of all neurological symptoms, and he was discharged from the hospital after a 3-day stay.

Case no. 35.—Depth, 150 feet; exposure, 28 minutes; decompression time, 2 minutes. While at stool about 25 minutes after a dive, the subject felt numbness of buttocks and sacral region. On rising there was an intense burning sensation and posteriorly down the thighs. As this burning sensation increased in intensity, he was recompressed to 75 pounds with complete relief. But during the 294 minutes decompression he had a definite chill and began a dry nonproductive cough. On emerging from the recompression chamber, the chilliness and cough persisted and his temperature was 102.4° F., pulse 108, and respiration 56. He was put to bed and given symptomatic treatment, and 2 hours later he was resting more comfortably in spite of his cough and temperature of 101.8° F., a pulse of 112, and respiration of 43.

He was transferred to the hospital that evening for examination and treatment.

Examinations upon admission were as follows: X-ray examination of the chest, anterior-posterior views with patient lying in stretcher, shows irregular congestion at the base of the right upper lobe, the right mid lobe and also the left lower lobe. Conclusion: These findings are not typical of pneumonic consolidation but resemble more that of an irritation.

Blood sedimentation index: 11 mm in 60 minutes. R. B. C.: 4,980,000; W. B. C.: 27,100; Hgb.: 90 percent; Diff.: Neutrophiles 2 percent, band form neutrophiles 36.5 percent, segmented neutrophiles 53 percent, lymphocytes 5 percent, and monocytes 3.5 percent. Urine: albumin positive (Neg. in 1–10 dilution) otherwise normal.

Diagnosis of "bronchitis, acute, condition considered to have been an arritative bronchitis" was made, and after 9 days in the hosptial condition had cleared sufficiently for discharge.

Two divers (cases nos. 33 and 34) who were exposed with case no. 35 also experienced the mildest type of symptoms diagnosible as definite caisson disease. Case no. 33 had itching of the upper anterior part of the chest. Case no. 34 had a definite rash over the anterior surface of the chest. No recompression was required for either of these cases as no further symptoms developed

Case no. 43.—Depth, 185 feet; exposure, 13½ minutes; decompression time, 2.1 minutes. About 35 minutes after a dive this subject noted a loss in visual acuity. Images were seen as distorted and not on a normal plane. A few minutes later he noticed that vision was confined to an area of about 2 square feet immediately ahead, unless the neck was bent and the head held at a different angle. It appeared to the subject as if he was looking through a tube. No eye pain but slight stiffness of the posterior neck muscles was noted. Recompression to 82 pounds and decompression over a period of 84 minutes gave complete relief of eye symptoms but a headache developed which persisted for 24 hours.

Case no. 45.—Depth, 200 feet; exposure, 13 minutes; decompression time, 2.2 minutes. A dull pain in right hip joint was experienced about 15 minutes after reaching the surface. He was recompressed to 27 pounds with relief at 20 pounds, and decompressed over a period of 80 minutes. The point of interest in this case is that during his ascent from a dive, the subject held a weight on his right thigh which apparently impeded the circulation. This is an example of a mechanical constriction preventing the elimination of the extra nitrogen in the blood and tissues, and thus producing caisson disease. This man continued the series making longer dives at this depth without trouble.

Case no. 46.—Depth, 200 feet; exposure, 14 minutes; decompression time, 2.2 minutes. About 3 minutes after reaching surface, this subject felt a sense of restriction in his chest which rapidly increased in severity so that he was unable to breathe, and collapsed. He was taken into the recompression chamber within a few seconds but before pressure could be raised cyanosis had developed. Partial return of respiratory function was noted at 7 pounds with complete return at 18 pounds. At this time sharp pain in the occipital region developed associated with "lightning streaks before the eyes." Pressure was carried to 45 pounds and he was decompressed in 38 minutes. Headache and ocular symptoms were still present on reaching atmospheric pressure and as these symptoms continued and he also developed difficulty in urinating, he was recompressed a second time 5 hours and 13 minutes after start of first recom-

pression. This recompression was carried to 22 pounds and decompression lasted 38 minutes. He was relieved and allowed to go home where he developed numbness and tingling of both feet and legs, and some difficulty in walking.

He was again recompressed, 3 hours after the second recompression, this time to 89 pounds and decompressed in 147 minutes. The subject was relieved again, but soon developed a pain in left elbow joint which made a fourth recompression necessary. This recompression was carried to 111 pounds where he remained for 10 minutes, and was then decompressed over a period of 230 minutes. This did not relieve the subject but further recompression was deemed inadvisable and the patient was transferred to the United States Naval Hospital, Washington, D. C., for observation and symptomatic treatment. Residual symptoms, i. e., partial paralysis and tingling sensations of the lower extremities, severe constipation, and difficult urination, lasted for several months. The subject was rendered impotent by this attack of caisson disease and has remained so for over 18 months. It is apparent from the outcome of this case that the original recompressions were not carried to a great enough depth. In the treatment of caisson disease in certain cases it is essential that the patient be subjected to greater pressure than that at which he was diving originally, and then be decompressed over a prolonged period of time, in order to avoid such unfortunate sequelae as occurred in this case.

Table 2 shows the total number of dives for each individual subject at all depths, and the total number of attacks of caisson disease for each individual. An individual predilection to caisson disease is shown by cases B. A. M. and J. C. C. with 6 attacks of caisson disease each out of 139 and 136 dives, respectively, while W. H. S. made 236 dives with only 3 attacks, and G. B., S. A. K., J. H. M., and A. L. L. made 154, 140, 135, and 111 dives, respectively, with only 1 attack of caisson disease each. We are also of the opinion, after a careful history on each case, that alcoholic consumption the evening before a dive is a predisposing factor in the occurrence of caisson disease. Although sufficient proof is not available it also appears that the site of an old injury is a common place for the occurrence of caisson disease.

DISCUSSION

Many reports of caisson disease are to be found in the literature, but none of them have been analyzed in relation to the relative saturation of the theoretical tissues of the subjects.

It is known that the blood supply to different parts of the body varies greatly and it is also known that the capacity of different parts of the body for dissolving and storing nitrogen differs. Thus the white matter of the central nervous system has a small blood supply but a high capacity for storing nitrogen; while, on the other hand, the gray matter has an enormous blood supply and no extra storing capacity. Muscular blood supply varies according to the work of the muscle. Fat stores five times as much nitrogen as water yet its blood supply is small.

Thus we can easily see that the time taken for different parts of the body to saturate with nitrogen will vary greatly. On the basis of these and other facts Boycott, Damant, and Haldane (1908) proposed the use of 5, 10, 20, 40, and 75 minute theoretical tissues in the calculation of the necessary decompression following a given exposure. They demonstrated that when the excess of atmospheric pressure did not exceed about 1¼ atmospheres, there were no symptoms of caisson disease no matter how long the exposure or how rapid the decompression. They reasoned that since the volume of nitrogen liberated would be the same when the total pressure was halved whether that pressure be high or low, it would be as safe to diminish the pressure rapidly from 4 atmospheres to 2, or from 6 to 3, as they knew it was from 2 to 1. These premises were proven to be true experimentally, and a safe ratio for decompression was figured to be 2 to 1. As long as none of the theoretical tissues were allowed at any time to exceed this ratio during the decompression no caisson disease was to be expected, and the present divers' decompression tables are based on this ratio.

The method of calculating the depth or saturation of any of the theoretical tissues during decompression or after reaching the surface is too involved and too lengthy to be presented in a paper of this nature. Reference should be made to the preceding paper by Hawkins, Shilling, and Hansen (1935) for a complete discussion. The saturation (depth in feet absolute) of the theoretical tissues upon reaching the surface following the experimental exposure has been calculated by the Haldane method for each case of caisson disease reported in this paper, and is presented together with the saturation ratio and other data in table 1. For example, when case no. 1 reached the surface (atmospheric pressure) his 5-minute theoretical tissues were saturated as if left at a depth of 124 feet (absolute), the 10-minute tissues were left at 120 feet, the 20-minute tissues were left at 101 feet, the 40-minute tissues were left at 78 feet, and the 75-minute tissues were left with a saturation equivalent to a depth of 60 feet (absolute). The saturation of 124 feet leaves the 5-minute tissues with a ratio of 3.8 to 1 (124–33), similarly, the 10-, 20-, 40-, and 75-minute tissues are left at the ratios of 3.6, 3.1, 2.4, and 1.8 to 1, respectively.

Although it was thought dangerous to exceed a ratio of 2.3 to 1 in any of the tissues, it is surprising to note that not only in case no. 1 but in every case of caisson disease occurring during these experiments as shown in table 1, the 5-, 10-, and 20-minute tissues exceed this ratio. From these high saturation ratios, it might be concluded that the cases of caisson disease presented were caused by bubbles occurring in the 5- or 10-minute tissues; but when one calculates the position of the tissues for the subjects in runs prior

to the first run in which caisson disease occurred at each depth, it is seen that the 5- and 10-minute tissues are of no significance in the production of caisson disease for they are left with a ratio of 4 to 1 or 5 to 1 in every case, and yet no symptoms of caisson disease are encountered.

The 20-minute tissues are the first to have a significant relation in the production of caisson disease for when these tissues are left saturated at 100 feet absolute (3 to 1) or deeper, "bends" begin to appear. Whether the 20-minute tissues could be left with a higher saturation without trouble resulting is difficult to demonstrate, for when they are left at 100 feet absolute, the 40-minute tissues have reached a saturation of 74–76 feet or a ratio of 2.3 to 1. Thus it is impossible to determine whether the resulting attack of "bends" was produced in the 20- or the 40-minute tissues, but by the same reasoning which we used in calculating the 5- and 10-minute tissues we can increase the safe ratio of the 20-minute tissues to 3 to 1 instead of 2 to 1.

It is thus seen that the incidence of caisson disease is very definitely related to the saturation of the theoretical tissues. As pointed out by Hawkins, Shilling, and Hansen (1935), the relationship between this tissue saturation and the calculation of decompression tables is also important for on the basis of these facts they have shown that it is possible to calculate decompression tables without the necessity of considering the 5- and 10-minute tissues, and using a ratio of 2.8 to 1 for the 20-minute tissues. Thus from the "lung" experiments it is shown that the time of decompression can be cut on dives of short duration since the 40- and the 75-minute tissues do not become deeply saturated.

SUMMARY

Forty-six cases of caisson disease occurring during 2,143 experimental dives have been presented. Thirteen cases occurred following exposures of from 34 to 53½ minutes at a depth of 100 feet (gage). Twenty-two cases occurred following exposures of from 18½ to 28 minutes at a depth of 150 feet (gage). Six cases occurred following exposures of from 17½ to 22½ minutes at 167 feet (gage). Three cases occurred following exposures of 12½ to 13½ minutes at 185 feet (gage). Two eases occurred following 13- and 14-minute exposures, respectively, at 200 feet (gage).

It has been shown that the nitrogen saturation of the theoretical tissues is definitely related to the incidence of caisson disease.

TABLE No. 1

Case no.	Series and run nos.	Exposure, depth in feet gage	Exposure time, minutes	Decompression time, minutes	Depth of theoretical tissues in feet absolute					Tissue saturation ratio					Symptoms of caisson disease	Time of onset	Recompression, pounds, gage	Decompression, minutes	Outcome of case
					Minute tissues					Minute tissues									
					5	10	20	40	75	5	10	20	40	75					
1	11–43	100	34	1.5	124	120	101	78	60	3.8–1	3.6–1	3.1–1	2.4–1	1.8–1	Tingling and numbness, lower extremities; slight abdominal distress.	32	60	290	No recurrence.
2	7– 7	100	37½	1.1	126	123	105	81	62	3.8–1	3.7–1	3.2–1	2.5–1	1.9–1	Pain and rash left upper arm and shoulder.	420	27	147	Do.
3	11–48	100	39	1.5	124	122	106	79	63	3.8–1	3.7–1	3.2–1	2.4–1	1.9–1	Pain, right deltoid region	382	75	292	Do.
4	11–48B	100	39	1.5	124	122	106	79	63	3.8–1	3.7–1	3.2–1	2.4–1	1.9–1	Pain, right shoulder and right upper chest.	Imm.	None	None	Complete relief in few hours.
5	7– 9A	100	39½	1.1	126	124	107	83	64	3.8–1	3.7–1	3.2–1	2.5–1	1.9–1	Slight pain, left shoulder	Imm.	58	64	No recurrence.
6	7– 9A	100	39½	1.1	126	124	107	83	64	3.8–1	3.7–1	3.2–1	2.5–1	1.9–1	Pain and tingling, left knee and hip.	5	36	63	Do.
7	9– 7	100	40½	1.1	126	124	107	84	64	3.8–1	3.8–1	3.2–1	2.5–1	1.9–1	Tingling, left hip and knee joints.	10	58	102	Do.
8	9– 9	100	42½	1.1	126	125	109	85	66	3.8–1	3.8–1	3.3–1	2.6–1	2.0–1	Tingling, dorsal surface right hand and arm.	30	58	103	Do.
9	11–52A	100	43	1.5	124	124	109	86	66	3.8–1	3.8–1	3.3–1	2.6–1	2.0–1	Numbness, tingling, and tenseness, right buttock and thigh.	110	60	286	Complete relief.
10	9–12	100	45½	1.1	126	126	111	89	67	3.8–1	3.8–1	3.4–1	2.7–1	2.0–1	Rash and itching, region of left deltoid muscle.	90	None	None	Partial relief three hours.
11	11–55	100	48	1.5	124	124	112	89	68	3.8–1	3.8–1	3.4–1	2.7–1	2.1–1	Sudden sharp pain, right lumbar and epigastric region, subject, doubled over.	10	60	300	Recurrence, see write-up.
12	9–20	100	53½	1.1	126	127	116	93	72	3.8–1	3.8–1	3.5–1	2.8–1	2.2–1	Tingling feet and legs, pain lower right chest.	45	58	100	No recurrence.
13	9–24	100	53½	1.1	126	127	116	93	72	3.8–1	3.8–1	3.5–1	2.8–1	2.2–1	Itching of left upper arm	155	58	102	Do.
14	10–1A	150	18½	1.7	158	157	104	75	57	4.8–1	4.2–1	3.2–1	2.3–1	1.7–1	Rash, itching and stiffness back of neck.	5	80	60	Recurrence, see write-up.
15	3–53	150	19½	1.7	159	140	107	76	59	4.8–1	4.2–1	3.2–1	2.3–1	1.8–1	Pain in abdomen. Nausea, sweating, and weakness.	23	33	23½	No recurrence.
16	10– 5	150	20	1.7	160	142	108	78	59	4.8–1	4.3–1	3.3–1	2.4–1	1.8–1	Pain in chest, dizziness and nausea.	Imm.	82	232	Do.
17	3–54	150	20½	1.7	160	143	109	79	60	4.8–1	4.3–1	3.3–1	2.4–1	1.8–1	Pain in abdomen. Sweating and pruritis.	5	44½	41	Do.
18	8– 8A	150	20½	1.7	160	143	109	79	60	4.8–1	4.3–1	3.3–1	2.4–1	1.8–1	Soreness left hip and upper leg.	15	45	39	Do.
19	8– 9A	150	21½	1.7	161	144	112	81	61	4.9–1	4.4–1	3.4–1	2.5–1	1.8–1	Rash and soreness right hypochondriac region.	25	80	120	Do.
20	10–12	150	23½	1.7	162	148	114	83	63	4.9–1	4.5–1	3.5–1	2.5–1	1.9–1	Pain and rash, right wrist	30	80	118	Residual swelling
21	10–14	150	24½	1.7	163	152	118	86	64	4.9–1	4.6–1	3.6–1	2.6–1	1.9–1	Tingling and pain, forehead	5	80	236	No recurrence.

22	10–14	150	24½	1.7	163	152	118	86	64	4.9–1	4.6–1	3.6–1	2.6–1	1.9–1	Pain, left elbow and shoulder	35	80	224	Do.
23	3–71 A	150	25½	1.7	164	152	120	88	66	5.0–1	4.6–1	3.6–1	2.7–1	2.0–1	Pain, muscles of left thigh	20	22	31	Do.
24	3–71 A	150	25½	1.7	164	152	120	88	66	5.0–1	4.6–1	3.6–1	2.7–1	2.0–1	Pain and rash, left upper arm	20	45	14	Do.
25	3–72	150	26½	1.7	164	153	122	89	67	5.0–1	4.6–1	3.7–1	2.7–1	2.0–1	Pain, tingling, and cyanosis of both hands.	30	None	None	Cleared in 24 hours.
26	3–72 A	150	26½	1.7	164	153	122	89	67	5.0–1	4.6–1	3.7–1	2.7–1	2.0–1	Slight pain, neck and right shoulder.	10	None	None	Cleared in 1 hour.
27	16–8	150	26½	2.0	162	152	122	89	67	4.9–1	4.6–1	3.7–1	2.7–1	2.0–1	Pain in chest and tingling sensation in arms.	45	80	294	Residual soreness of chest.
28	15–34	150	26½	2.0	162	152	122	89	67	4.9–1	4.6–1	3.7–1	2.7–1	2.0–1	Dull ache and cyanosis, left breast.	120	None	None	Disappeared in 48 hours.
29	15–35	150	27	2.0	163	153	123	90	67	4.9–1	4.6–1	3.7–1	2.7–1	2.0–1	Soreness, right chest. Pain left orbital region.	Imm.	None	None	Disappeared in 10 hours.
30	16–9 A	150	27	2.0	163	153	123	90	67	4.9–1	4.6–1	3.7–1	2.7–1	2.0–1	Sharp severe pain, chest. Mottling chest and abdomen.	30	80	310	Residual paralysis; see write-up.
31	3–73	150	27½	1.7	165	156	124	91	68	5.0–1	4.7–1	3.8–1	2.8–1	2.1–1	Severe pain and slight rash, left shoulder.	3	{ 44½	40½	Recurred.
32	3–73 A	150	27½	1.7	165	156	124	91	68	5.0–1	4.7–1	3.8–1	2.8–1	2.1–1	Swelling of right wrist and hand. Pain in right wrist.	15	15½ 22	78½ 31	No recurrence. Do.
33	15–37	150	28	2.0	163	155	125	92	68	4.9–1	4.7–1	3.8–1	2.8–1	2.1–1	Itching, upper right chest	30	None	None	Disappeared in 90 minutes.
34	15–37	150	28	2.0	163	155	125	92	68	4.9–1	4.7–1	3.8–1	2.8–1	2.1–1	Rash over anterior chest	60	None	None	Disappeared in 40 minutes.
35	15–37	150	28	2.0	163	155	125	92	68	4.9–1	4.7–1	3.8–1	2.8–1	2.1–1	Intense burning, sacral region	25	75	294	Recurrence; see write-up.
36	4–13	167	17½	1.9	169	146	110	79	59	5.1–1	4.4–1	3.3–1	2.4–1	1.8–1	Dyspnea, substernal pain, and sweating.	12	21	39½	No recurrence.
37	4–16	167	20½	1.9	173	155	118	85	63	5.2–1	4.7–1	3.6–1	2.6–1	1.9–1	Dull pain, right hip and right shoulder.	330	30	60	Do.
38	4–16 A	167	20½	1.9	173	155	118	85	63	5.2–1	4.7–1	3.6–1	2.6–1	1.9–1	Rash and sense of constriction, left lower chest.	15	27	21	Do.
39	4–17 A	167	21½	1.9	174	156	121	85	64	5.3–1	4.7–1	3.7–1	2.6–1	1.9–1	Rash, anterior neck. Difficulty in swallowing.	20	27	20	Do.
40	4–19	167	22½	1.9	175	159	124	88	65	5.3–1	4.8–1	3.8–1	2.7–1	2.0–1	Slight pain, left shoulder	270	76	64	Do.
41	4–19 A	167	22½	1.9	175	159	124	88	65	5.3–1	4.8–1	3.8–1	2.7–1	2.0–1	Sharp pain and rash, left shoulder.	405	27	39	Do.
42	5–8	185	12½	2.1	170	138	100	71	54	5.2–1	4.2–1	3.0–1	2.2–1	1.6–1	Sharp pain and rash, left shoulder and arm. Pain, right lumbar region. Weakness and tingling right leg.	25 30	82	66	Do.
43	5–9	185	13½	2.1	174	142	104	73	56	5.3–1	4.3–1	3.2–1	2.2–1	1.7–1	Visual acuity distorted. Skin cold and clammy. No pain.	35	82	84	No recurrence; see write-up.
44	5–9	185	13½	2.1	174	142	104	73	56	5.3–1	4.3–1	3.2–1	2.2–1	1.7–1	Pain, right upper arm and clavicle.	5	82	84	No recurrence.
45	6–21	200	13	2.2	182	148	107	75	57	5.5–1	4.5–1	3.2–1	2.3–1	1.7–1	Dull pain, right hip joint	15	27	80	No recurrence; see write-up.
46	6–23	200	14	2.2	185	154	112	78	58	5.6–1	4.7–1	3.4–1	2.4–1	1.8–1	Extremely severe case. Varied multiple symptoms.	3	(1)	(1)	(1)

[1] Varied and numerous; see write-up.
Depth (absolute) = depth (gage) + 33 feet.

TABLE 2

Subject	Dives	"Bends"	Subject	Dives	"Bends"
S. A. K.	140	1	G. —. B.	154	1
P. J. B.	147	2	E. N. W.	19	1
B. A. M.	139	6	H. F. S.	114	4
C. S. M.	90	2	L. —. Z.	120	0
C. A. R.	94	3	A. L. L.	111	1
O. L. C.	52	1	J. E. G.	97	3
J. C. C.	135	6	J. —. M.	92	2
H. M. Y.	20	1	R. A. H.	25	0
J. H. M.	135	1	R. W. C.	26	3
K. B. M.	34	1	L. B. H.	1	1
W. H. S.	236	3			
T. E. N.	162	3	Total	2,143	46

NOTE.—L. B. H. made his dive at the end of a series in which caisson disease had already occurred in other subjects.

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NAVAL HOSPITAL ADMINISTRATION

By G. F. COTTLE, Captain, Medical Corps, United States Navy

An American College of Hospital Administrators has been formed this year. Its objectives are stated to be:

(*a*) To elevate the standard of hospital administration.

(*b*) To establish a standard of competency for hospital administrators.

(*c*) To develop and promote standards of education and training for hospital administrators.

(*d*) To educate hospital trustees and the public to understand that the practice of hospitalization calls for special training and experience.

(*e*) To provide a method for conferring fellowship in hospital administration on those who have done or are doing noteworthy service in the field of hospital administration.

For several years the American Hospital Association has held meetings, issued bulletins, conducted round table discussions and supervised a "refresher" course of two weeks called the Hospital

Institute at the University of Chicago in problems of hospitalization. Its institutional and individual membership is very large and is rapidly growing. The meetings are most instructive and well attended.

A further step in the post-graduate training of hospital administrators has been taken at the University of Chicago by the establishment of a course lasting through the academic year, open to physicians, doctors of public health, and college graduates, with business and executive training who are desirous of fitting themselves for the hospital field.

The transition of the naval medical officer from the clinical to the administrative field is a gradual one. The administrative part of his life begins in the function of ward officer and officer of the day in naval hospitals, becomes expanded in the function of senior medical officer, and reaches a most definite form in the assignment as executive officer of a naval hospital. The all-absorbing interest of the clinical phase of the naval medical officer's life often overshadows the administrative phase. Naval medical officers are as a rule, like most physicians, individualistic. Their administrative ability is most readily exhibited in the close personal relationship of the sick bay, the dispensary and the smaller naval hospital. When subjected to the more complicated and administrative demands of a large naval hospital the individualism of their past experience is given a severe test. In some it is expanded rapidly by the method of trial and error under the more mature administrative guidance of the commanding officer, into an attitude which increases the efficiency of the staff, the contentment of the personnel and the value of the hospital to the patient.

The American College of Hospital Administrators is patterned after the American College of Surgeons. It is possible that a conscious effort on the part of both junior and senior naval medical officers to continuously seek to develop their own executive efficiency by attuning themselves to the objective of such organization as the American Hospital Association and a college of hospital administrators may improve the administrative ability in the same manner that the efforts of our surgeons to attain recognition by the American College of Surgeons has tended to raise the standard of surgical practice in the Navy.

It may be that the time has come when to the postgraduate courses in clinical specialties, to the educational opportunity afforded by assignment to the Naval War College and to the Army Industrial School, there will be added by the Bureau an opportunity to attend a refresher course in hospital administration or even an academic post-graduate course in hospital administration.

Recognition of the ability of naval hospital administrators by the American College of Hospital Administrators may not be neces-

sary as an index of efficiency but naval medical officers who fit themselves to attain the objective of that organization by study and practice will improve their personal ability in the field of naval medical administration.

The naval medical officer who in his earlier years becomes one capable of effectively meeting the medical and surgical emergencies that appear in his practice aboard ship, who later perfects himself in a chosen specialty of medical or surgical practice, has not rounded out his career until he has proven himself capable of taking his place as an able administrator in the hospital field. In the building of this firm foundation he will acquire that wide experience and knowledge which make him of value to the naval medical service when at the outbreak of war he finds himself called upon to supervise and direct activities of physicians suddenly dislocated from their civilian environment and expected to function in the military environment.

Only when the administrative ability of the naval medical officer keeps pace at all times with his clinical ability will he in reality deserve to be considered not only able in his chosen professional field but have added to that the right to be considered an able naval officer.

MENINGOCOCCAL SEPTICEMIA WITH REPORT OF CASE SHOWING ORGANISMS IN THE DIRECT BLOOD SMEAR

By JOEL T. BOONE, Commander, and W. W. HALL, Lieutenant Commander, Medical Corps, United States Navy

"No other infection so quickly slays" says Herrick.

That statement in itself justifies the reporting of a very fulminating case of meningococcal septicemia admitted to the U. S. S. *Relief* from a battleship on March 9, 1935, with death occurring 2½ hours later. The patient was first seen by one of the medical officers of the ship 2½ hours before his transfer to the *Relief*. In other words as far as could be determined he was sick approximately only 5 hours prior to death.

Meningococcal infection has been called by many names among which are cerebrospinal meningitis, epidemic meningitis, meningococcus meningitis, Meningococcus sepsis, spotted fever, petechial fever, brain fever, etc. The term cerebrospinal fever is now generally accepted as indicating a disease entity, without any necessary anatomic localization, while at the same time giving prominence to the usual site of attack. The disease is listed as cerebrospinal fever in the 1918 American edition of the International Classification of Causes of Death and is so designated in the Navy Nomenclature of Diseases and Injuries.

The term meningococcal septicemia has a special significance, although it is generally recognized that the disease may occur without an accompanying meningitis but with sufficient other findings to definitely establish its identity. Boyd says:

In every case of meningococcal meningitis there is probably an element of septicemia, but the term meningococcal septicemia is usually reserved for those cases in which there is a blood infection without a corresponding infection of the meninges. It is an extra-meningeal meningococcal infection. Meningitis may sometimes develop after the septicemia has been in progress for a number of weeks; this is called meningitis tarda. The course of meningococcal septicemia varies enormously. The fulminating cases may be incredibly rapid and in these the infection may be so heavy that large numbers of meningococci can be seen in the blood smears.

The question raised by Mink (2) whose observations as to an extrameningeal meningococcal infection are not only interesting but find receptive reaction on the part of the contributors of this article. To quote Mink:

The textbooks of 1916 recognized no meningococcus infection except that characterized by inflammation of the meninges. * * * At that time there appears to have been no conception of the possibility of a mild infection involving other tissues and not involving the meninges. The experiences during the war demonstrated a pure septicemic form, without meningeal involvement. It seems worth while inquiring if this conception of milder forms of infection may not be extended. Is there not in existence a still milder, more localized form of meningococcus infection involving the walls of the nasal and pharyngeal cavities and the accessory sinuses? Is it not possible that the so-called "carrier" who increases in numbers so rapidly under unfavorable climatic conditions represents not a mere carrier condition but rather an individual with a local pathological condition who is suffering from the mildest local form of meningococcus infection?

While the case herewith reported is an example of an extrameningeal meningococcal infection, it is in contradistinction to the mild or possibly transient type of infection above referred to, a very severe, very fulminating type.

Patient J. J. L. admitted to U. S. S. *Relief* 10:30 a. m., April 9, 1935, as stretcher case from one of the battleships with diagnosis undetermined (meningitis) with history stating patient had been first seen by one of the medical officers of his ship at 7:30 a. m. that date when he was complaining of sore throat, headache, malaise and some nausea, temperature at that time was 99.2° F.; 2 hours later it had risen to 105° F. with aggravation of symptoms complicated by vomiting. As the battleship was sailing that morning and that hospitalization be not delayed, patient was transferred without spinal puncture, although meningitis was suspected with the aggravation of earlier symptoms. Upon admission to the *Relief*, patient was conscious, well oriented and not particularly disturbed although realizing that he was very ill. It was noted that there was some confusion as to history of onset, his statement not corresponding with the recorded history. He first stated that he had been sick 4 days aboard his ship; later stating that he reported to sick bay the previous day; then not being sure when he first felt ill. Symptoms recorded aboard

Relief were: Sore throat, headache, photophobia, nausea, numbness and coldness of feet, feverishness. Physical examination revealed the following: A very acutely ill youth—examiner considered him critical from beginning—18 years of age (had enlisted Feb 17, 1934), fairly well developed for age and general build, 68½ inches tall, weighing 125 pounds; mental state rational, well oriented, normal insight, some realization of his serious condition, concerned for the welfare of his parents who had suffered financial adversity, courteous, respectful and cooperative during examination. E. E. N. & T.: Eyes injected, lacrimation, dilated pupils which react to light and accommodation. Ears negative. Nose congested and inflamed. Throat very red; some slight hypertrophy of tonsils which are spongy; pharynx very red with stringy exudate. Mouth: tongue clean, breath not offensive, sordes on teeth. Neck negative. Thorax well developed, symmetrical masculature good. Lungs negative. Heart negative except expansive pulsations; rate 160; pulse not well sustained and poor quality. Abdomen negative. Genito-urinary negative. Extremities negative. Skin-cyanosis pronounced of entire face and body except lower portion of lower extremities. Mottling of all skin surfaces with a hemorrhagic purpura with large coalescence of some of the lesions, same enlarging visibly and strikingly during examination. Both eye lids deep arcola. Reflexes; no stiffness of neck; no Babinski; no Kernig; exaggerated knee jerks.

Spinal puncture was immediately performed. Spinal fluid was not under increased pressure; same was clear; 30 cc fluid withdrawn and sent to laboratory; and 30 cc antimeningococcic serum administered replacing the spinal fluid withdrawn.

Blood was taken for counting and smear preparation made. Hemoglobin 80 percent; W. B. C. 6,000; differential count—myeloblasts 1 percent, myelocytes 5 percent, juveniles 6 percent, band forms 6 percent, segmented 11 percent, lymphocytes 71 percent, several normoblasts; blood film loaded with Gram negative diplococci (Illustrations are photomicrographs of these blood films.)

Spinal fluid report: Fluid clear; cell count 3; globulin (Pandy) negative; colloidal gold 0–0–0–0–0–0–0–0–0–0.

Patient was failing rapidly with loss of consciousness, extreme cyanosis, extension and coalescence of purpura, and appearance of large ecchymotic blotches on lip, cheeks, chest, abdomen, back, and extremities, occurring within the first hour of admission. At the conclusion of intravenous administration of 90 cc antimeningococcus serum, there was large quantity of greenish fluid vomited with a second vomiting attack in a few minutes of coffee-ground vomitus and patient died.

Summary of the case: Patient admitted at 10:10 a. m. critically ill, cyanotic and extensive hemorrhagic purpura distributed over face, body and extremities; spinal puncture resulted in removal of 30 cc clear fluid not under pressure, with cell count of 3 and without organisms; W. B. C. 6,000 with a very marked shift to the left—normoblasts present; Gram negative diplococci present in blood smear; blood culture—pure culture of Gram negative diplococci; neurological signs were essentially negative; no response to specific serum therapy administered intraspinally and intravenously; cyanosis and purpura rapidly increased; consciousness lost, with life terminated at 12:35 p. m.—2 hours and 25 minutes after admission to the hospital ship.

AUTOPSY FINDINGS (GROSS)

The body is that of a white male of medium frame, well developed, well nourished, light brown hair, blue eyes. Rigor mortis is slightly developed, the entire

skin is cyanotic and peppered with purpura, both small and large confluent spots.

Brain.—The vessels of the meninges and cortex are congested. There appears to be some excess of clear subarachnoid fluid. The brain substance is congested and moderately edematous. No exudate is present in the ventricles nor in the subarachnoid spaces of the brain, medulla, or cord, as far down as examined. The ependyma appears normal, the choroid congested.

Cest.—There is no excess of fluid in the pleural cavities. The parietal pleura is spotted with small petechiae. The lungs are acutely congested. The heart is normal in size and position. The myocardium is soft and semiopaque in appearance. The endocardium contains some small and two rather large (3 x 4 mm) petechiae. The valves are normal.

Thymus.—The thymus is large, weighing approximately 35 grams. The parenchyma appears hyperplastic and is not surrounded and infiltrated by fat and fibrous tissue as is usual in the age involution to be expected.

Abdomen.—The peritoneal cavity contains no excess fluid. Both perietal and visceral peritoneum are studded with many petechiae of various sizes. There is acute congestion and cloudy swelling of all solid viscera. *Adrenals* are swollen and appear hemorrhagic. No other important pathological change noted.

MICROSCOPIC FINDINGS

Heart.—The muscle fibers are separated by edema in some places and in the endocardial margin there are hemorrhages which separate the endocardium from the muscle and also separate various bundles of muscle from the main mass of myocardium.

Thymus.—The thymus does not present the picture of involution to be expected in this aged individual. Lymphocytes of the cortex are abundant. The usual adipose tissue replacement is not seen. Hassels corpuscles are numerous in the medulla.

Spleen.—The spleen is acutely congested and shows areas of hemorrhage. The malpighian corpuscles show active hyperplasia.

Adrenal.—The adrenal is acutely congested, the cells of the cortex are swollen and separated one from the other. The medulla is hemorrhagic throughout its entire extent.

Kidney.—The kidney shows extreme acute congestion with edema and granular degenerative change of the epithelium of the tubules.

Liver.—The liver shows acute congestion and granular degenerative change of the polygonal cells.

Brain.—The brain tissue appears normal other than acute congestion of the vessels. In the subarachnoid space there is no evidence of inflammatory reaction.

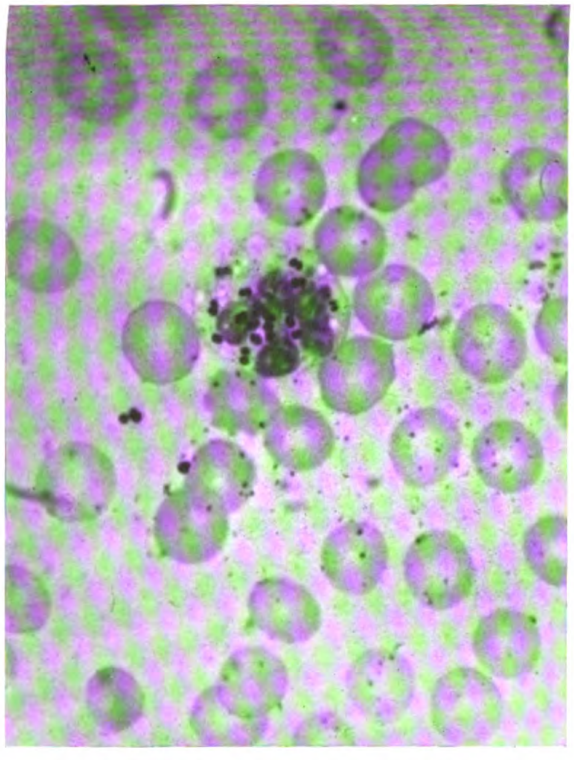
It will be particularly noted that the spinal fluid, obtained at puncture antemortem, was entirely negative and that corresponding to this there was no pathological change noted in the gross or microscopical study of the brain, cord, choroid nor ependyma of the ventricles other than congestion and a few wandering cells in the subarachnoid spaces, not of the polymorphonuclear variety. The infection definitely had not localized in the central nervous system. Another important and interesting observation was that the thymus was persistent.

Leake (3) says with reference to the thymus "an interesting observation has been made by Symmers (1918) that patients with status lymphaticus are prone to be attacked by a fatal form of cerebrospinal fever; 60 percent of the necropsies on cerebrospinal fever cases at Bellevue Hospital revealed status lymphaticus, while the incidence of the condition in necropsies on cases other than cerebrospinal fever was 8 percent. A similar predisposition was noted in Westenhoeffer's Silesian series of 29 necropsies."

At present there is considerable difference of opinion as to the importance of even existence of such a state as thymicolymphaticus. Various authorities (Marine, Warthin, and others) describe the condition as a constitutional defect associated with lowered resistance and characterized by general hyperplasia of lymphoid tissue as well as thymus with hypoplasia of cardiovascular system adrenals and gonads. The latter portion of the description does not apply to our case as heart gonads and adrenals appeared normal in size.

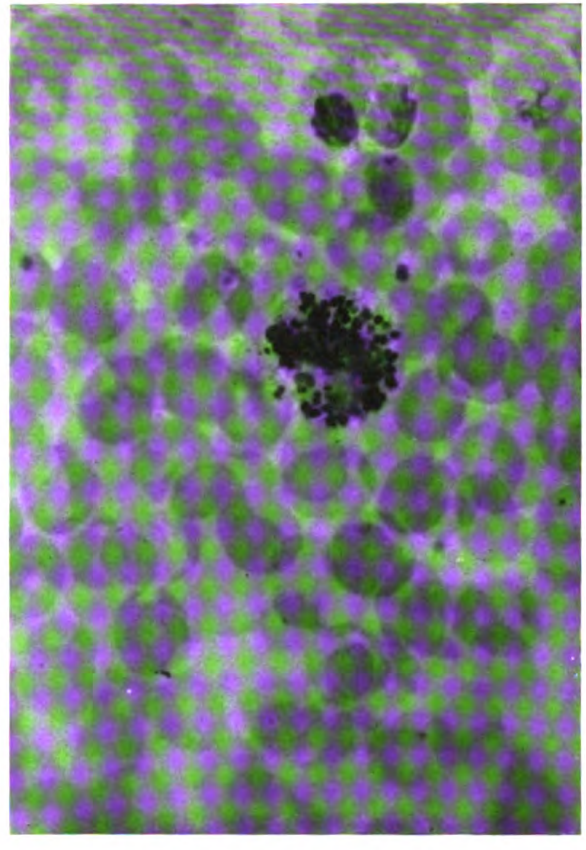
It is generally accepted that all cases of cerebrospinal fever first harbor the organisms in the naso-pharynx, that is, they are for a variable length of time a "carrier" of the organism. The something occurs which lowers the resistance of the tissues and the organisms spread. The route by which the meningococci pass from the nasopharynx to the meninges has been a debated point. If they pass along the lymphatics in the ethmoid and thus reach the subarachnoid space they leave no trace either of organisms nor sign of inflammation in the ethmoid. Furthermore there is really no communication between the lymphatics of the sinuses and the cerebrospinal system. Leake (3) says "The subarachnoid space with the ventricles and the perivascular and perineural spaces of the brain and cord form a closed sac, and lymph spaces are not found. Cerebrospinal fluid takes the place of lymph within that part of the central nervous system which is enclosed by the dura mater." It, therefore, seems most likely that the organisms first invade the blood stream. Next a metastatic focus is set up in the choroid plexus. They then implant themselves on the ependymal lining of the ventricles which is an epithelial surface, as is, the nasopharyngeal lining from which they have come. Only at last as the infected ventricular fluid reaches the subarachnoid space do they reach their natural pathogenic habitat, the meninges. The blood stream phase then is only transitory, strictly speaking a bactermia. Only rarely do the meningococci multiply in the blood stream to cause what may be considered a true septicemia.

In the case here reported the organisms were profuse in the blood stream and were multiplying therein rapidly, causing a virulent septicemia. The blood smears, stained by Wright's differential blood stain, showed many diplococci of characteristic morphology both



MENINGOCOCCAL SEPTICEMIA.

FIGURE 1.—Photomicrograph of blood film (× 1,250) from a case of meningococcal septicemia, stained with Wright's differential blood stain. Diplococci are seen within a polymorphonuclear leukocyte and some less distinctly extracellularly. Other smears stained by Gram showed the organisms to be Gram negative. Organisms cultured from the blood and identified as meningococci.



MENINGOCOCCAL SEPTICEMIA.

FIGURE 2.—(× 1,250) Photomicrograph of blood film from a case of meningococcal septicemia showing a polymorphonuclear leukocyte filled with diplococci.

intracellular, appearing in the polymorphonuclear leukocytes, and extracellular. Other smears stained by the Gram method showed the diplococci to be Gram negative. Blood culture taken at the same time was found positive; the organisms were subcultured and identified as meningococci.

SUMMARY

1. A fulminating case of meningococcal septicemia of five and one-half hours duration from time first seen by a medical officer until death, is reported. Organisms were found in the direct blood smears both intra and extracellularly (photomicrographs).

2. Ventricles and meninges had not been invaded by the organisms.

3. A persistent thymus, hemorrhagic adrenals, with congestion and cloudy swelling of all solid viscera were found at autopsy.

4. Meningococcus infections have an extremely wide range of expression from the very mild unobserved, apparently innocuous nasopharyngitis, without constitutional symptoms, to the virulent fulminating types with or without meningeal involvement as exampled with the case presented.

5. The present concept of the court of a meningococcus infection is nasopharyngitis-bacteremia (septicemia) -choroiditis-ependymitis-meningitis. The infection may remain stationary at any one of these stages or develop distant metastatic foci other than meningitis.

6. Scrutiny of the blood smears in cases of meningococcus infection is, as here demonstrated, of practical value as well as of theoretical interest for, though rare, a certain number of such fulminating cases as the one presented occur.

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THE SCHILLING COUNT IN ACUTE SURGICAL CONDITIONS[1]

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A complete blood picture is a routine procedure in the study of medical and surgical cases. The interpretation of the reports depends on the understanding of the hematopoietic system, by the internist or surgeon reviewing them.

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The blood is a delicate organ, if we may refer to it as such; it is readily affected by internal and external stimuli, but in spite of its sensitivity it maintains a normal level unless the disturbing influences become too great.

White and differential counts very often give no information, either because the blood is able to maintain a normal level despite the disturbing influences, or because of failure to interpret properly the counts. When the internist or surgeon lacks confidence in the white and differential counts as aids in diagnosis and prognosis the reason lies only too often in his lack of familiarity with the physiology and pathology of the white cells and his consequent inability to interpret the counts correctly.

Throughout adult life, under normal conditions, the red cells, white cells and platelets are formed in the bone marrow of the ribs, sternum, vertebrae, and upper ends of the humerus and femur. The lymphocytes are formed by the lymphatic system. The origin of the monocytes is a disputed question but they are in all probability derived from the reticulo-endothelial system.

The polymorphonuclear granular leucocytes develop from the reticular cells of the bone marrow and in their maturation pass through the intermediate states of myeloblast, premyelocyte, myelocytes, juvenile, band (metamyelocyte) to the mature polymorphonuclear leucocyte. The myeloblast has a round nucleus which contains nucleoli and a fine reticular chromatin network. The cytoplasm is nongranular. As the cells mature, nucleoli disappear, the round nucleus becomes indented and this indentation continues until at first we have two lobes separated by a nuclear band of chromatin material. Later in the development as many as 5 or 6 lobes may be present, all connected by a filament of chromatin material. As the nucleus goes through the various stages changes in the cytoplasm take place. The first granules to appear stain purplish red, and vary in size (azur granules). Then granules of 3 types appear which are present and distinct in all further states of maturation. They are the neutrophilic granules which are small, of equal size, and uniformly scattered throughout the cell. The eosinophilic granules are large and take an eosin stain. Cells whose granules are besophilic are few in number; these granules vary in size and stain a purplish blue.

The polymorphonuclear leucocytes pass through the intermediate stages in the bone marrow and are delivered to the blood stream in the band and mature forms. The leucocytes become more mature in the blood stream by nuclear segmentation. The questions of duration of life and mode of destruction of the white blood cells have not been settled. Leucocytes are delivered into the blood stream in two

tides, one in the forenoon which reaches its height in the afternoon, with the ebb in the evening; the other starts in the evening, attains its height about midnight, with the ebb in the early morning. These tides were thought formerly to be due to food and were referred to as digestive leucocytosis. But they are now known to be uninfluenced by food, exercise, or sleep. Blood counts should be made at the same hour daily so that the count is made at the same tide level. Shaw has shown that the total number of leucocytes may vary as much as 50 percent in 24 hours, but that the percentages of different cells vary only slightly. Normally there are 4 to 7 percent of immature leucocytes present in the blood and this percent is not affected by the tide of the so-called "digestive leucocytosis."

The total white blood count in normal adult males is from 5,000 to 10,000. In children the count runs much higher—from 8,000 to 18,000.

Leucocytosis is an increase in the total number of white blood cells per cubic millimeter. It is uncommon to have an increase of all the varieties of leucocytes at the same time. Usually only one type of cell is increased, the rest remain normal or reduced in number. If the total number of a particular cell is greater than normally present, we speak of it as absolute increase, if less than that normally present it is called a relative increase. The normal total number present is found by multiplying the normal percentage of any cell type by the normal total count.

The polymorphonuclear neutrophilic granular leucocytes (segmenters) in health form about 55 to 75 percent of all the leucocytes. Arneth classified the cells according to their nuclear lobes. It is accepted that band forms or metamyelocytes are less mature than lobed polymorphonuclear cells. Arneth used this variation for his Arneth Index. He classified the cells into 5 groups.

Group I. Cells with simple round or indented nucleus, 5 percent. This group included all immature forms.

Group II. Two lobed cells, 35 percent. The lobes are separated by a definite chromatin filament.

Group III. Three lobed cells, 42 percent.

Group IV. Four lobed cells, 17 percent.

Group V. Five lobed cells, 2 percent.

Arneth further increased his classification by subdividing each of the classes. Class I was divided into myelocytes (M cells), metamyelocytes with slightly indented nuclei (W cells) and metamyelocytes with deeply indented nuclei (T cells). The other groups with definite nuclear lobes were classified as to the shape of the lobes, whether they were bend (S) or knob-like (K).

There is no question but that the Arneth count is of great value. But it is time consuming and not practical for the routine count. In the acute infectious stages, where great numbers of the leucocytes show toxic changes, as vacuolation of the cytoplasm, loss of granules, appearance of toxin granules and nuclear changes (pyknosis) the Arneth count becomes difficult to do and unreliable. Cooke modified the Arneth count, making it simpler and more accurate. He referred to it as the polynuclear count. Cooke classed all cells as immature cells in which there was a nuclear band separating the lobes. If the lobes were separated by a chromatin filament they were said to be mature.

Cooke's classification contained 5 groups:

Class I. Immature, 12 percent.

Class II. Two lobed, 25 percent.

Class III. Three lobed, 44 percent.

Class IV. Four lobed, 15 percent.

Class V. Five lobed, 4 percent.

Cooke attributed the same significance to the various classes as Arneth did.

Both Arneth and Cooke took into consideration only the percentage of the total polymorphonuclear neutrophilic differential count, but neither the Arneth or Cooke counts became very popular because of the difficulty in classifying the cells and interpreting the counts, and because of the time-consuming feature.

Cooke also developed an "Index" based on his polynuclear count. He multiplied the percentage in class I by one, class II by two, etc. The total thus obtained was divided by the number of cells counted. The result was called the weighted mean, which normally was about 2.7. Lower figures indicated immature cells, while a high figure indicated maturity of cells.

In 1912 Schilling further simplified the classification of the neutrophilic cells into the following groups:

1. Myelocytes, cells with round nucleus, the primary granulocyte normally present in the bone marrow.

2. Young metamyelocytes or juveniles, cells having slight indentation of the nucleus.

3. Band or older metamyelocytes, in which there is deep indentation of the nucleus but no true lobulations.

4. Mature polymorphonuclear cells with definite nuclear segmentation, lobes being separated by a chromatin filament.

In doing a differential count each of the above types is classed separately along with the other forms of white blood cells so that in the completed differential the varieties of neutrophiles are expressed as a percentage of the total number of leucocytes.

In the Arneth and Cooke counts only the percentage of the various neutrophilic cells were indicated.

TABLE I.—*Normal Schilling hemogram*

	Average normal	Normal limits
Total leucocytes	8,000	6,000–10,000
Neutrophilic granular cells; band or deeply indented metamyelocytes	4	3–5
Mature polymorphonuclear segmenters	63	51–67
Eosinophiles	2	2–4
Basophiles	1	0–1
Monocytes	6	4–8
Lymphocytes	23	21–35

Schilling also discusses a degenerative and regenerative shift which he interprets as indicating the state of bone marrow function. Schilling does not count the nuclear lobes, although he does group the neutrophiles according to their maturity. This method is much simpler than either the Arneth or Cooke counts. It is impossible for toxic changes to interfere with the count, so it remains practicable for routine work.

There is great need for a differential count which takes into consideration the immaturity of cells. The ordinary differential count gives us normal figures in patients who are suffering from infective processes, and it is in these cases that the shift is most marked and of greatest value in diagnosis and prognosis.

The Schilling count is employed routinely at this hospital. It is not more time consuming than the ordinary differential, and the information obtained in most cases is a great aid in diagnosis and prognosis. It is not possible for the average hospital corpsman to differentiate the various cells but with a little instruction he is able to make a reliable count. The chief requisite is a good stained blood smear. The usual laboratory technician is in such a hurry that he makes his smears too thick; does not understand his Wright's stain; uses tap water instead of a buffer solution as a diluent, and seeks to make his count on a part of the slide where the cells are the thickest. In making a reliable Schilling count the accepted technic of blood staining and counting must be adhered to. adhered to.

In the Schilling count or any count where the immaturity of cells is taken into consideration we speak of a "shift to left" and "shift to right." By shift to the left is meant an increase over normal of the number of immature cells present in the blood and by shift to the right an increase in the more mature forms.

There are several general types of leucocytosis. An increase in the neutrophilic leucocytes is spoken of as a neutrophilia. Increase

in basophilic granular cells as a basophilia or mast cell leucocytosis, and increase in eosinophilic granular leucocytes as an eosinophilia, etc. It is only within the province of this paper to discuss neutrophilia, the remainder of the cells will only be discussed as they enter the picture of a eutrophilia or neutropenia.

Neutrophilic leucocytosis occurs in acute infection. There are several important exceptions, some of which are typhoid, malaria, uncomplicated tuberculosis, mumps, smallpox, influenza, German measles and agranulocytic angina, etc. In pneumonia, scarlet fever, and all types of acute meningitis, and osteomyelitis there is a marked and characteristic increase in neutrophiles. An absence of neutrophilia in a case where it is definitely known to be characteristic is evidence of severe infection or a hematopoietic defect and the prognosis is grave. The blood should be examined repeatedly during the course of the acute infective diseases because it may be the first indication of a complication developing.

It is not unusual to find the blood picture 24 hours ahead of the clinical symptoms. It may not be needed in diagnosis but may be a great aid in prognosis or may give information of a developing complication. The blood examination must be correlated with all the features of the case as it is only a part of the general study. A single blood count gives us in most cases little information. Repeated counts should be made to determine the progress of the shift. A progressive shift to the left is always evidence of an intense demand on the bone marrow.

Neutrophilia is most important in cases where there is accumulation of pus, as in appendicitis, abscesses, otitis media, empyema, peritonitis, etc. In these conditions a blood count gives valuable information as to whether the process is progressing, stationary, or subsiding.

Many conditions which are not infective may give a neutrophilia, as toxemic conditions, uremia, shock, traumatic and surgical, diabetic coma, coronary occlusion, intestinal obstruction, eclampsia, and malignant tumors. In malignancy it is usually found that the bone marrow is involved.

Leucocytosis may result from severe muscular activity, pregnancy, pain, cold bathing, hemorrhages and drugs.

It is not known how the number of leucocytes in the circulation is regulated. In the bone marrow there is a large stock of mature leucocytes which are ready to be poured forth upon demand. When these cells are called forth there is an increase in the white blood count plus a "shift to the left." As the demand continues the bone marrow pours forth blood cells which are less mature, and if the demand becomes exceedingly great over a period of time even blast

cells may appear and the picture change from that of a neutrophilia to that of a neutropenia.

Just what stimulus is necessary to call forth the white blood cells is not known. Buchner and others believe that bacterial proteins have chemotactic properties. Piney believes that it is possible that concentration of cells in one place, away from the circulation as a whole, can itself be a stimulus to leucocytic production.

When the infection is mild the bone marrow can produce mature cells to keep up with chemotaxis and there is no marked increase in immature cells in the blood stream. When the infection becomes severe the bone marrow cannot keep pace with the demand so that large numbers of immature cells appear in the blood, and finally when all the bands and juveniles have been called forth the myelocytes and even the blast cells appear in the blood with a resultant neutropenia.

In neutrophilia there is usually a decrease in the other forms of cells. This is particularly true of the eosinophiles and lymphocytes. If the infection is long-continued a secondary anemia develops because the neutrophiles and red blood cells develop from the reticuloendothelial cells of the bone marrow, and the toxic action upon it for production of neutrophiles affects the red-cell production. This anemia usually progresses to a certain point and remains stationary, occasionally it may become severe.

In 1905 Sondern concluded from a large number of counts that "the increase in the relative number of polymorphonuclear cells is an indication of the severity of toxic absorption and the degree of leucocytosis is an evidence of the body resistance toward infection." In 1919 Walker verified the work of Sondern, Gibson, and Wilson, and presented an Index of Resistance in which 10,000 leucocytes and 20 percent lymphocytes were regarded as normal, for every 1 percent decrease in lymphocytes the leucocytes must rise 1,000. For example 26,000 leucocytes with 13 percent lymphocytes would give an index of + 9; indicating good body resistance and 10,000 leucocytes with 14 percent lymphocytes would give an index of − 6, indicating poor body resistance. This index works very satisfactorily in acute infective conditions and should be used more.

Slight variations in the blood picture as a slight leucocytosis and moderate shift to the left are difficult to interpret. Repeated counts should be made in such cases. A high leucocytosis and marked shift to the left denotes a severe infection, particularly if the juveniles exceed the band forms. With a low total count and marked shift to the left, the bands and juveniles exceeding the mature polys, the prognosis is exceedingly grave. In these cases the eosinophiles are greatly reduced or absent, the lymphocytes likewise greatly reduced

In recovering from an infection the total number of leucocytes decrease; there is a fall in the neutrophiles; the lymphocytes increase and the eosinophiles reappear.

When once a neutrophilia has been found it should be followed up by repeated total white and Schilling counts until they return to normal. In acute infective cases as appendicitis, abscesses, etc., hourly counts should be made. After the acuteness subsides daily counts and biweekly counts should be made until the blood picture returns to normal. By so doing, complications may be discovered before they are clinically in evidence. Lymphocytosis following a neutrophilia with the eosinophilia appearing and the total count becoming less, is indicative of recovery or of convalescence.

In many acute cases the total white and Schilling count is not needed for diagnosis. But there is valuable information to be gained from it. It gives us information as to the infection and how the body is combatting it. The information as to prognosis is invaluable.

Neutropenia may occur in any acute infection. It is characteristic of certain infections, particularly those due to bacilli, e. g. typhoid. In ordinary coccal infections if the hematopoietic system becomes damaged by toxins the rate of production of cells falls behind the rate of destruction and a neutropenia results. In these cases there is marked shift to the left and most of the neutrophiles are immature, which indicates a poor prognosis.

Many surgeons have little confidence in the total white and differential counts as an aid in the diagnosis of acute surgical conditions, particularly appendicitis. It is surprising how often the subject of appendicitis is discussed by noted surgeons without mentioning the blood picture.

A study of appendicitis cases, from a hematological standpoint has been undertaken at this hospital to determine the extent of diagnostic aid that could be thus obtained. A total white and Schilling count was made shortly before operation. After removal of the appendix it was sectioned and classed into 4 groups from a histological study. To date 100 cases have been studied. The 4 histological groups are:

1. Chronic appendicitis—appendices showing no evidence of acute inflammation.

2. Acute catarrhal appendicitis—appendices in which the inflammation was confined to mucous membrane only.

3. Acute suppurative appendicitis—appendices in which all the coats were congested, adematous and infiltrated with polymorphonuclear leucocytes.

4. Gangrenous appendicitis—appendices in which gangrenous areas were present, also those cases in which perforations and peritonitis were present.

In group 1, diagnosed histologically as chronic appendices there were 38 cases. These patients at time of operation gave histories of having had attacks of pain in the right lower abdominal quadrant, but at time of operation did not present acute symptoms. The total white counts varied between 4,850 and 14,800, average count 8,072. The immature cells varied between 0 and 15 percent. Average immature cells 5 percent.

In group 2, diagnosed histologically as acute catarrhal appendicitis, there were 17 cases. The total white count varied between 5,000 and 19,000, average total count 10,940. The immature forms varied between 11 and 25 percent. Average immature cells 16 percent.

In group 3, diagnosed histologically as acute suppurative appendicitis, there were 26 cases. The total count varied between 7,500 and 21,800, average total count 13,855. The immature forms varied between 17 and 30 percent. Average immature cells 24 percent.

In group 4, diagnosed histologically as gangrenous appendicitis, there were 19 cases. The total count varied between 12,000 and 25,550, average count 16,747. The immature cells varied between 32 and 63 percent. Average immature cells 40 percent.

Too much reliance should not be placed on the total white count, as it denotes only the resistance of the patient. This was brought to light in the study of these cases. In 7 of the 19 cases of gangrenous appendicitis the count did not exceed 12,000. The ordinary differential count is misleading in that oftentimes the polymorphonuclears do not exceed 65 percent, which is considered normal. One of the cases reported had a total white blood count of 12,300, Schilling count—juveniles 2, bands 20, segmented 40, lymphocytes 30, monocytes 6, eosinophiles 2. Here the neutrophilic percentage according to the ordinary differential was 62 percent, which is considered normal—but the Schilling count showed 22 of the 62 polys to be immature, which denoted a rather severe infection. Operation was performed in this case although the clinical signs and symptoms were not typical and an acute suppurative appendix removed. The degree of shift denotes the severity of infection. From a study of the immaturity of cells in this series of 100 cases one arrives at the conclusion that an acute appendix will be found at operation whenever the immature percentage is in the neighborhood of 20. With an immature count around 30 percent a gangrenous appendix will be found. With a normal immature count the diagnosis of acute appendicitis can definitely be excluded. A high immature count does not exclude pyelitis, cystitis, or other infectious condi-

tions. The cases must be judged from a clinical standpoint as well as from the blood pictures.

Occasionally, in the case of an appendix which is greatly distended, injected, and covered with a plastic exudate, the Schilling count shows only a slight shift to the left but with a marked leucocytosis. We must distinguish between two varieties of acute appendicular trouble, namely, acute appendicitis and acute appendicular obstruction. In acute inflammation of the appendix the essential feature is the inflammation of the wall of the appendix. In acute appendicular obstruction the primary lesion is the obstruction and the inflammation secondary. Primary obstruction is distinguished by the great severity of pain, suddenness of onset, normal temperature and pulse during the early stages, and slight shift to the left as evidenced by Schilling count. Acute obstruction of the appendix is just as serious a condition as acute inflammation and calls for immediate operation. It is an acute appendicular obstruction that the Schilling count may be misleading. In the early stages there is only a slight shift but there is a marked leucocytosis. A typiscal count in acute appendiceal obstruction is: White blood count 18,750, juveniles 2, bands 8, segmented 79, lymphocytes 8, eosinophiles 1, monocytes 1.

TABLE II.—*Typical counts in appendicitis*

	Total W. B. C.	E	Mo	Ba	My	J	B	S	L
Chronic appendicitis	8,000	2	3	------	------	------	5	65	25
Acute catarrhal	11,450	1	10	------	------	------	16	52	21
Acute suppurative appendix	15,500	------	1	------	------	4	19	64	12
Gangrenous appendicitis with or without perforation and peritonitis	18,000	------	------	------	2	10	38	45	5

W. B. C.—Total leucocyte count.
E —Eosinophiles.
Mo —Monocytes.
Ba —Basophiles.
My —Myelocytes.
J —Juveniles.
B —Bands.
S —Segmented—mature polys.
L —Lymphocytes.

Presented herewith are some unusual cases in which the Schilling count aided in the diagnosis and was a factor in deciding upon operation.

Case 101.—J. Z., Bmkrlc, weight 212 lbs., age 31, C. C. pain in abdomen. Pain began 18 hours before admission, bowels moved a few hours before admission and pain shifted low on right side, pain radiated to right testicle and thigh. No nausea or vomiting, T. 102, P. 100, R. 22. Had several chills. Examination showed an obese male. The abdomen was not distended, rigidity was difficult to elicit due to deep layers of fat on abdomen. There was pain on pressure

over lower portion of right rectus. The inguinal canal was extremely tender on examination. Urine analysis negative. Flat K. U. B. and X-ray chest negative. W. B. C. 15,300, J 2, B 34, S 41, L 18, E 1, Mo 4. As no cause could be found for the marked shift to left except possibly appendicitis, operation was immediately performed, and an acute suppurative appendix removed.

This case presented several peculiarities. Examination was unsatisfactory, due to his deep layers of adipose tissue covering the abdomen. Symptoms suggested appendicitis, ureteral colic, pyelitis, catarrhal fever. Temperature 102 and chills are unusual in the early stages of appendicitis. The Schilling count with 36 percent immature cells aided materially in deciding on operation.

Recently a gangrenous appendix was removed from a Filipino who had chills and a fever. Temperature 104 at time of operation. Total white count 8,800, B 41, S 54, L 5. The low total count, chills and fever denoted lowered resistance, while the marked shift gave the information as to the severity of infection.

Case 102.—M. S., A. S., weight 120, age 21. Symptoms began 24 hours before admission. Attack began with pain in upper abdomen. Vomited 10 times during the day and had 4 watery bowel movements. T. 99.2, P. 64, R. 22. Examination was negative except for slight rigidity and pain on deep pressure over McBurneys point. Urine analysis negative. W. B. C. 19,700, J 8, B 22, S 64, L 8. Due to the marked shift, operation was immediately performed and a gangrenous appendix removed.

In this case the Schilling count was of invaluable aid in diagnosis, due to the condition simulating gastro-enteritis. Without it operation might have been delayed. A few hours delay would have meant rupture of the appendix.

Case 103.—F. S., C. C. C. patient. Had been constipated for a week and during that time had had some abdominal discomfort. Night before admission, following a bowel evacuation, he developed a stomach-ache generalized over abdomen. He went to sleep at about 10 o'clock and was awakened at midnight with severe pain in abdomen. Pain shifted an hour later to right side. No nausea or vomiting. Patient in severe abdominal distress. T. P. R. normal, knees drawn up, abdomen of board-like rigidity. At 9 o'clock operation was decided upon and an acute suppurative appendix removed.

TABLE III.—*Showing value of serial hemogram, case 103*

Time	W. B. C.	J	B	S	L	E	Mo
0300	12,000	----	8	55	25	8	4
0700	16,000	1	11	65	16	7	----
0900	19,900	1	34	47	17	----	1

This case shows the value of serial hemograms as recommended by Baum. In 6 hours the immature cells rose from 8 to 35, and the total count from 12,000 to 19,900.

Case 104.—W. E. B., Pvt., U. S. M. C., admitted with typical signs and symptoms of appendicitis. (See table IV.)

TABLE IV.—*Case of acute appendicitis with a lung complication*

Date	W. B. C.	J	B	S	L	E	Mo
Oct. 23, 1934	18,400	6	12	73	9		
Oct. 25, 1934	23,450	2	46	46	4	1	1
Oct. 26, 1934	17,300	3	23	63	9		
Oct. 29, 1934	8,700	3	15	46	28	4	4
Oct. 30, 1934	8,600	0	9	52	31	4	4

Operation October 23, 1934, an acute appendix being removed. Two days later patient developed atelectasis of left lung, from which he recovered. The hemogram returned to normal in 5 days, which was in keeping with the clinical aspects of the case.

Repeated counts following operation give a good index as to prognosis. If the immature cells return to normal, a subsidence of infection has taken place. If the immature cells increase or remain stationary a continuation of the infective process is present.

TABLE V.—*Schilling Hemogram in ruptured gastro-duodenal ulcer*

No.		W. B. C.	My	J	B	S	L	E	Ba	Mo
105	Ruptured gastric ulcer	20,150	0	2	39	40	18	1	0	0
	First day postoperative	19,250	0	1	30	56	13	0	0	0
	Second day postoperative	10,300	0	6	22	47	25	0	0	0
	Third day postoperative	4,400	0	0	40	20	40	0	0	0
	Fourth day postoperative	5,900	1	10	36	18	33	0	0	2
	Fifth day postoperative	8,300	0	9	42	22	23	4	0	0
	Sixth day postoperative	9,200	0	4	44	28	20	3	0	1
	Seventh day postoperative	13,200	0	3	32	38	19	6	0	2
	Eighth day postoperative	16,700	0	2	31	52	14	1	0	0
	Tenth day postoperative	13,500	0	2	28	49	19	0	0	2
	Nineteenth day postoperative	9,600	0	2	13	45	37	1	0	2
106	Ruptured duodenal ulcer	18,150	0	1	11	51	35	0	0	2
	First day postoperative	14,500	0	2	24	69	5	0	0	0
	Third day postoperative	7,150	0	0	18	62	19	0	0	1
107	Ruptured duodenal ulcer	13,100	0	3	19	43	29	3	0	3
108	Ruptured gastric ulcer	21,100	0	2	24	50	24	0	0	0
109	Ruptured duodenal ulcer	16,600	0	1	17	77	5	0	0	0
	First day postoperative	17,350	0	5	35	45	10	0	1	4
	Second day postoperative	15,150	0	2	15	24	8	1	0	0
	Fifth day postoperative	9,000	0	0	23	60	13	2	0	2

W. B. C.—Total leucocyte count.
E —Eosinophiles.
Mo —Monocytes.
Ba —Basophiles.
My —Myelocytes.
J —Juveniles.
B —Bands.
S —Segmented—mature polys.
L —Lymphocytes.

TABLE VI.—*16 cases of acute surgical conditions*

No.		W. B. C.	My	J	B	S	L	E	Ba	Mo
110	Ruptured appendix, localized peritonitis	20, 100	0	2	25	65	8	0	0	0
	Fifteenth day postoperative	6, 950	0	0	4	66	29	1	0	0
111	Appendiceal abscess	12, 750	0	1	38	40	12	3	0	6
	Second day postoperative	14, 000	0	0	28	51	11	3	0	6
	Eighteenth day postoperative	7, 050	0	1	4	43	47	4	0	1
112	Appendiceal abscess	13, 400	0	4	22	66	5	0	0	3
	Second day postoperative	12, 700	0	1	11	47	34	0	0	7
	Twenty-first day postoperative	8, 100	0	0	5	65	28	2	0	0
113	Subdiaphragmatic abscess	12, 800	0	1	23	56	19	0	0	1
	Third day postoperative	10, 700	0	6	19	61	12	2	0	0
	Eighth day postoperative	4, 750	0	1	9	46	39	5	0	0
	Twentieth day postoperative	6, 250	0	0	3	64	31	0	2	0
114	Subdiaphragmatic abscess	31, 350	2	5	45	44	4	0	0	0
	Second day postoperative	20, 100	0	1	44	51	4	0	0	0
	Fourth day postoperative	12, 100	0	2	25	67	6	0	0	0
	Thirtieth day postoperative	8, 200	0	0	12	48	32	2	0	6
115	Periproctic abscess	12, 050	0	2	20	32	42	0	0	4
116	Osteomyelitis	9, 800	0	8	30	51	7	0	0	4
117	Empyema	18, 000	0	6	25	46	18	1	0	4
118	Cellulitis, hand	14, 400	0	2	28	48	22	0	0	0
119	Liver abscess	14, 750	2	8	58	25	4	1	0	2
120	Empyema	25, 500	0	6	41	43	8	0	0	2
	Third day postoperative	20, 500	0	0	28	37	30	1	0	4
	Thirtieth day postoperative	15, 000	0	2	12	63	23	0	0	0
	Fortieth day postoperative	15, 250	0	3	11	66	18	2	0	0
121	Empyema	13, 200	0	2	28	52	17	0	0	1
	Third day postoperative	12, 800	0	0	6	73	18	1	0	2
122	Infected pilonidal cyst	11, 550	0	2	16	54	27	1	0	0
123	Burns, second degree, chest, hands, and back	37, 900	0	3	31	55	9	0	0	2
124	Brain abscess	31, 500	0	3	13	82	2	0	0	0
125	Pyonephritis	5, 450	0	6	36	27	26	0	1	4

W. B. C.—Total leucocyte count.
E —Eosinophiles.
Mo —Monocytes.
Ba —Basophiles.
My —Myelocytes.
J —Juveniles.
B —Bands.
S —Segmented—mature polys.
L —Lymphocytes.

TABLE VII.—*Acute medical cases*

No.		W. B. C.	My	J	B	S	L	E	Ba	Mo
126	Catarrhal fever acute	12, 800	0	2	11	71	11	0	0	5
127	Gastric-enteritis acute	10, 100	0	3	17	62	15	3	0	0
128	----do	6, 500	0	2	26	44	22	5	0	1
129	----do	33, 800	0	5	16	71	7	1	------	------
	3 days later	8, 800	0	1	3	68	24	3	0	1
130	Influenza	5, 150	0	0	19	28	48	0	0	5
131	Typhoid fever	4, 300	0	1	24	42	33	0	0	0
132	Pneumonia	20, 400	0	12	33	42	9	2	1	1
133	----do	12, 500	0	2	28	55	12	0	0	3
	2 days later	13, 300	0	8	56	24	10	0	0	2
	7 days later	13, 050	4	12	48	18	13	0	0	5
	Death 14 days later	10, 550	10	14	50	16	9	0	0	1
134	Pneumonia	17, 200	0	2	30	66	2	0	0	0
	2 days later	12, 900	0	6	30	53	9	0	0	2
	3 days later	6, 050	0	8	9	55	28	0	0	0
	5 days later	4, 250	5	10	34	18	29	0	0	4
	Death 6 days later	17, 250	8	14	44	29	4	0	0	1

W. B. C.—Total leucocyte count.
E —Eosinophiles.
Mo —Monocytes.
Ba —Basophiles.
My —Myelocytes.
J —Juveniles.
B —Bands.
S —Segmented—mature polys.
L —Lymphocytes.

2605—35——4

In table V are represented the Schilling hemograms in five cases of rupture gastric and duodenal ulcers. The five cases of ruptured gastric and duodenal ulcers are interesting from a hemotological standpoint. In each case there was a definite leucocytosis. In each case the degree of shift was dependent on the time elapsed between the rupture and the taking of the hemogram. Case 105 had a definite peritonitis upon admission, the rupture having occurred about 5 hours before admission. Case 106 was admitted 1 hour following rupture and the shift was within normal limits although the total count was 18,150. The leucocytosis in these early cases is due to shock and not infection. In none of the cases was drainage instituted. In case 105 the blood picture rand hand in hand with the clinical features. On the third day postoperative the patient was moribund, the total leucocyte count was 4,400 with 40 percent immature cells. In the course of 5 days he received 50 cc of leucocytic extract and copious amounts of fluids intravenously, which no doubt saved his life. Another interesting feature is the reappearance of eosinophiles after being absent during the more acute stage of convalescence. Many observers have noted an eosinophilia of acute surgical infection and the reappearance of eosinophilia during recovery. This has been noted throughout the study of the acute surgical cases reported on in this paper.

The cases of appendiceal abscess and sub-diaphragmatic abscesses (table VI) made an uneventful recovery following drainage. The hemograms are self-explanatory. In surgical cases the total leucocytosis is not of prime importance but Moon and Kennedy have shown that increased concentration of the blood results from surgical and traumatic shock. The degree of concentration is proportionate to the severity of the shock. The concentration of blood is of value in estimating the degree of shock in clinical cases. In case 123 with extensive burns about hand, back, and chest, there was a marked leucocytosis and shift to left shortly following the accident. In skull fracture cases it is not unusual to find the white blood count around 20,000 with normal or slight shift. Recently in a case of hemothorax as a result of a chest injury, the white blood count was 21,300 with J 6, B 8, S 67, L 17.

In table VII are represented acute medical cases which at times must be differentiated from acute surgical conditions. This is particularly true of gastro-enteritis. In cases of gastro-enteritis there is usually a shift to the left. In these cases an eosinophilia is present. From a hematological standpoint the eosinophilia is the only diagnostic criterion by which these cases can be diagnosed from acute surgical conditions of the abdomen. In using eosinophilia as a differential point the conditions which ordinarily cause an eosinophilia must be ruled out.

SUMMARY

The conventional differential leucocyte count as an aid in acute surgical conditions is unsatisfactory, due to the fact that it does not take into consideration the immaturity of cells. In many cases of desperate illness the conventional differential gives a normal figure. It is in these cases that the immature percentage is the highest.

A surgeon has great need for the Schilling count in diagnosis and prognosis. The blood picture is usually several hours to a day ahead of the clinical signs.

The Schilling count can easily be made by a medical officer or hospital corpsman. The time required is only slightly more than the time required for an ordinary differential count. It should be a routine procedure in the study of a surgical case.

CONCLUSION

1. The immature cells mirror the functional state of the bone marrow.

2. Appendicitis can be excluded in a normal Schilling hemogram. The degree of inflammation is proportionate to the number of immature cells.

3. The Schilling hemogram runs hand in hand with the clinical course in surgical conditions.

4. Serial Schilling hemograms should be made in all cases where the diagnosis is in doubt.

5. The total white cell count is often misleading and little reliance should be placed on it.

6. The Schilling hemogram is a definite aid in diagnosis and prognosis.

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GRANULOCYTOPENIA

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The last decade has revealed a constantly growing interest in the white blood cells and the serious aspects of their numerical changes and stages of development. A tendency to leucopenia is a part of well-known diseases such as measles, mumps, malaria, influenza, types of lymphoblastomas, etc., but a disturbance of the neutrophils may present the grave disease of neutro or granulopenia.

The etiology is unknown. White cells and granulocytes show a marked reduction with a myeloid tissue that is usually aplastic. Schultz in 1922 described a rapidly fatal condition of prostration, oral sepsis, and fever accompanied by severe neutropenia. Roberts and Kracke early stressed the importance of analyzing data in terms of white cell level and symptomatology. In their first paper in 1930 the mechanism of complete granulopenia was divided into five stages and that concept still holds in their studies to the present time. First is the bone marrow onset whose myelocytic function fails and after about 4 days the second stage appears, namely, the lessening and disappearance of granulocytes in the blood stream. The third stage or clinical onset appears in 1 or 2 days following the neutropenia. Weakness, early fatigue, and severe prostration with fever are quickly followed by the fourth stage or sepsis, as a result of the loss of the defensive bacteria barrier. Necrosis, ulceration, and hemorrhage appear and the last stage is tersely stated—granulocytes or death.

In 1931 Roberts and Kracke reported in a study of 8,000 private clinic patients that one of every four presented a mild granulopenia. Fatigue, weakness, and exhaustion were present in one-half of the female patients between the ages of 46 and 61, accompanied by neutropenia with severity of symptoms proportional to the degree of granulopenia. Their studies reveal that life is incompatible after 7 days' absence of granulocytes in the blood stream. Schultz believes the blood picture is secondary to a septic process that produces an aplasia of myeloid cells but neutropenia has been observed preceding any signs of infection with autopsies revealing bone marrow not aplastic in myeloid elements. The term "maturation arrest" has been suggested by FitzHugh and Krumbhaar as basic in some cases or neutropenia is a sign and not a cause.

Allergy as an etiological factor was suggested by Pepper in 1928 who found allergic factors in the background of neutropenic patients. Early in 1934 Madison and Squier reported 14 cases of severe granulopenia following the administration of amidopyrine and barbituric derivatives. The cases were among routine hospital admissions for medical and surgical treatments who were given the above drugs in the sedative routine. After recovery from all symptoms and weeks later, during a normal blood picture, two cases were again given amidopyrine and barbituric drugs followed promptly by a return of the chills and granulopenia. Similar reports are rapidly appearing in the literature but in contrast to these reports are the hosts of patients whose blood picture is not disturbed with the same drug therapy. This etiological factor might account for the large percentage of cases reported in physicians, nurses, and their families

who frequently use sedative drugs. The toxic effects of benzine on the bone marrow was demonstrated by Weiskoten in 1930 and in the same year Talley and Griffith reported similar effects following the administration of arsenaphenamine. The depressing effects of benzine were further demonstrated by Kracke who produced a chronic granulopenia, oral sepsis, and death in rabbits following small doses, intramuscularly and intraperitonealy, over prolonged periods of time.

The neutropenic state may be acute or chronic. The acute cases may prove rapidly fatal or become chronic with the white cells remaining constantly below 4,000 or fluctuating between a relatively normal count and a severe leucopenia. Counts below 4,000 may be consistent with health and leucocytosis develop promptly with a pyogenic infection. Another group of patients with a relatively low white count do not possess a feeling of well being. They have decreased energy with increase of lymphocytes and absence of myelocytes. Infection further increases with the leucopenia and the rapid course of the Schultz syndrome ensues.

The early diagnosis depends upon careful daily blood counts with especial study of the leucocytes, their number, differential ratios and percentage of cell-stage development. Marked leucopenia with granulopenia is the outstanding sign with leucocytes falling to 1,000 or less, indicating an impairment of the myeloblastic tissues.

Treatment has been largely empirical due to the obscure etiology and varying pathology. Aside from the general supportive treatment of preserving life and strength, efforts to stimulate the myeloid tissues have received special attention such as the nucleotides, transfusions, X-rays and yellow bone marrow. It is believed the normal stimulus in calling the granulocytes to the blood stream are liberated products such as nucleic acid, guanine, and adenine from disintegrating granulocytes. The unbroken pentose nucleotide K–96 prepared by the Harvard Medical School was used by Jackson and associates in 1931 and remains standard therapy. The dosage is 0.7 g in 10 cc of distilled water intramuscularly, or 0.7 g intravenously in 100 cc of saline solution for 4 days or until definite improvement is noted. Fourteen of their 20 malignant cases recovered with the first signs of improvement evident in 7 days. K–96 is not proven a definite maturation factor for granulocytes and has not been effective where granulopoiesis is depressed and maturation has ceased. Similar to the nucleotide K–96, is an extract of white cells obtained directly from the white cells of normal animals. Subcutaneous injections of 5 cc every 4 hours produced a gradual increase of leucocytes in the primary case herewith reported, while K–96 was not well borne.

The value of transfusions varies with different clinics. Beck finds no evidence of granulocytic stimulation and believes the neutrophils contained in 500 cc of blood inadequate. Kracke believes transfusions are rational treatment in the milder cases. Doan considers it rational treatment as an aid to myelocytic delivery through disintegrating products of nucleic acid on the myeloid centers. X-rays in one-twentieth erythema doses over the long bones are considered a myeloid stimulant if a hard filter is used and exposure not prolonged, as cell destruction will follow the early stimulation. The use of yellow bone marrow in teaspoonful doses three times a day is being advocated as a rational therapeutic measure but specific results are not proven.

The following case is reported in some detail as it illustrates a recurrent granulocytopenia with two acute attacks followed by a chronic neutropenic state:

Case 1.—"R. C." Commander (M. C.), U. S. N.

February 3, 1931. Admitted on U. S. S. *West Virginia* with malaise, sore throat, and temperature 104. Small boil on right buttock opened and antiseptic dressing applied. Blood count revealed 800 W. B. C. with almost absence of polymorphonuclear cells. Transferred February 7, 1931, at sea to U. S. S. *Relief* with diagnosis undetermined (angina agranulocytica).

February 7, 1932. U. S. S. *Relief*.—Readmitted from U. S. S. *West Virginia*. Diagnosis undetermined (angina agranulocytica). Number 2122. Not due to own misconduct.

C. C.: High fever, sore throat, prostration.

F. H.: Unmarried. One sister living and well. Parents and grandparents lived to ripe old age.

P. H.: 22 years naval service; always in good health; appendectomy 5 years ago; uneventful recovery. He developed neuritis of a peripheral nature 6 months ago and in searching for a possible focus of infection, several teeth were found to be infected. In May 1930 the second molar, upper right, abscessed and was removed. The socket healed promptly. No general symptoms. In June the left upper second molar began to ache. The tooth had a gold inlay. This was removed with relief of aching. After a few days treatment an occlusive treatment was applied in the cavity left by the inlay. In 3 days the gum above the tooth swelled. The dentist at once removed the occlusive filling in the tooth, but before this was done arthritis of two metacarpophalangeal joints in the patient's right hand occurred. This arthritis subsided 3 days after removal of the filling. Two days after this (now early in July) the patient played golf and immediately afterward a generalized neuritis flared up, affecting peripheral nerves of arms and legs.

The offending molar was extracted. One cusp of the molar broke off in the extracting, necessitating considerable instrumentation to remove it. The tooth socket was curretted and several days later, on account of continued aching, again curretted. The socket now healed, but the alveolar process occasionally ached. Early in August the remaining molars in the upper jaw, one on each side, were extracted. The tooth sockets healed quickly.

Acting on the advice of two dentists on September 8, 1930, the remaining teeth in the upper jaw, 10 in all (6 of them nonvital), were extracted and an alveolectomy performed under local anesthesia.

About 2 weeks later patient noticed a sudden loss of appetite. Neuritic pains in hands and feet continued, at times troublesome. There were periods of 2 or 3 weeks when the neuritis was absent. Sodium amytal and capsules of pyramidin (small doses) were taken occasionally for relief from the neuritis.

During the next few months the patient lost about 10 pounds in weight. The upper jaw continued to cause trouble. During this time three dentures were made and used in succession. The absorption of bone in the alveolar process was apparently quite rapid.

In January 1931 the neuritis pains in arms and legs were at times severe and caused insomnia. About the middle of January 1931, a general physical examination was made of the patient on the U. S. S. *Relief* and nothing found. W. B. C. at this time was 5,500.

The salient points in the history are as follows: Malaise for several days, but up and about. Evening of February 4 (3 days before admission), he was seized with chill, high fever, general aches and pains, sore throat, and extreme prostration. A white blood count at this time was said to be 800 with predominance of large lymphocytes. The following 3 days his prostration, sore throat, and leucopenia increased.

Temperature 103.6° F., pulse 120, respiration 20. Anemic-looking adult in extreme state of prostration and mental depression. Mentality, however, is clear. Pallor marked and there is erysipeloid eruption extending from alae of nose out over malar bones. Lips and finger nails blue. Skin is hot and dry. Pupils equal, regular, and active. Breath is very foul, tongue heavily coated, mouth shows great quantity of thick, white, frothy saliva. Upper teeth absent. Lower teeth in good condition. Gums are tender to touch and fiery red, but no ulcers seen. Tonsils enlarged and inflamed; pillars deeply injected and edematous. There is a great deal of inflamed lymphoid tissue in the pharynx. Nasopharynx apparently edematous as evidenced by reduced aeration. Cervical glands are palpable and tender.

Chest.—Respiration slow and deep. Chest is resonant throughout. No abnormal breath sounds heard. Heart rapid but regular. No murmurs. Sounds are distant. A_2 barely audible. B. P. 110/68. Abdomen is distended but not rigid or tender. Liver and spleen not palpable. There is a small carbuncle on right buttock. Anus shows two necrotic fissures. Extremities show no petechia or ecchymosis. There is no generalized glandular enlargement. Reflexes sluggish. The white blood count is said to be below 1,000. Inspection of a blood smear by Wright's stain shows complete absence of polymorphonuclear leucocytes. A few myelocytes are seen. The haemoglobin is reduced.

Positive findings.—(1) Sore throat, mouth, and anus—this includes tongue, gums, tonsils, pillars, and pharynx; (2) fever and prostration; (3) leucopenia with absence of polymorphonuclear leucocytes; (4) anemia.

Impression.—(1) Agranulocytic angina. (2) Aplastic anemia. This is considered a case of immediate transfusion of blood.

February 7, 1931. Patient received on board *Relief* at 1 p. m. this date, his condition remains as noted above; he was typed for immediate transfusion and found to be type "0"; 300 cc of blood transfused immediately by citrate method; patient had light reaction and temperature up to 104° F., but he feels somewhat better; morphine gr ¼, atrophine gr 1/150 administered hypodermically.

The line of treatment to be followed will be: (1) Transfusion of blood every other day alternating with X-ray exposures to the long bones (one-twentieth skin doses). (2) Liver extract, 200 g every other day, alternating with Ventriculin, 200 g. (3) High protein diet supplemented with glucose to

elevate the caloric intake. (4) Absolute quiet and sedatives for rest and sleep. (5) Mouth and throat hygiene, i. e., cleansing with mild oxidising agents.

Blood.—W. B. C. 1,600; differential N. 6 percent, L 94 percent, R. B. C. 4,100,000 Hb 80 percent.

February 8, 1931. *Blood.*—W. B. C. 2,150; R. B. C. 4,220,000 Hb 80 percent. Red cells show some variation in size and shape. No pathological red cells noted. The white blood cells show a preponderance of myelocytic variety. No neutrophilic cells seen. A few large bilobed cells seen which resemble an endotheliocyte. Myelocytes and metamyelocytes constitute the greater portion of the white cells. Peroxidase stain: Granulocytes 62 percent, nongranulocytes 38 percent. Wright's differential: Myelocytes 35 percent, metamyelocytes 15 perecnt, lymphocytes 41 percent, juvenile polymorphonuclears 8 percent. Blood platelet count, 350,000, showing some improvement from the transfusion.

Smear from mouth secretion shows V. A. in great profusion; stomatitis well marked; tonsils enlarged, inflamed, and pillars edematous. General condition shows no improvement since yesterday, temperature 103° F., pulse 110, respiration 20, radiation over long bones.

Urine.—Amber, acid, sp. gr. 1.010, albumin positive (3 plus), sugar negative, acetone negative. Microscopical: Many fine and coarsely granular casts, occasional leucocyte and few squamous epithelium.

February 8, 1931. Five hundred cubic centimeter blood transfusion today; general condition improved; temperature 100° to 101° F., pulse 98 to 100. Patient eats with difficulty on account of stomatitis and sore throat. No ulcers seen, lower colon cleansed with salt solution enema.

Urine.—Amber, acid, sp. gr. 1.014, albumin positive (3 plus), sugar negative. Microscopic: Few finely granular casts, occasional leucocyte.

R. B. C. 4,480,000. There is little if any variation in the size and shape of the red blood cells. W. B. C. 2,700. Peroxidase: 62 percent granulocytes, 38 percent nongranulocytes. Differential: Four hundred cells counted. 29 percent lymphocytes, 4 percent myelocytes, 19 percent metamyelocytes, 47 per cent polymorphonuclear leucocytes, divided as follows: Immature nonsegmented 17 percent, segmented 29 percent, banded form 1 percent, eosinophiles 1 percent. The granules are definitely neutrophilic.

February 10, 1931. Long bones radiated today; general condition seems better; he complains of extreme weakness; sleeps poorly. Temperature 99–100 F., pulse 95–100.

Blood.—R. B. C. 4,260,000; W. B. C. 2,100, Hb 80 percent. Differential: (Wright's stain), lymphocytes 31 percent, large mononuclears 4 percent, myelocytes 3 percent, metamyelocytes 18 percent polymorphonuclear leucocytes 44 percent, differentiated as follows: Nonsegmented polymorphonuclears 21 percent, segmented polymorphonuclears 23 percent. (Note, 2 segments 16 percent, 3 segments, or more, 7 percent.) Blood picture: The lymphocytes noted above were large and small lymphocytes with a clear blue cytoplasm. Those counted as large mononuclears contained a large centrally placed nucleus occupying almost the entire cell; a small clear blue ring of cytoplasm surrounded this nucleus and contained no granular material; this cell was large and in some instances larger than a polymorphonuclear leucocyte. Those classed as metamyelocytes were very large cells with indistinct nucleus occupying from a half to almost the entire cell, often indented and contained a large number of coarse granules. Those classed as juvenile polymorphonuclears show definite indentation or lobulation of the nucleus. Goodpastures stain: Granulocytes 72 percent, nongranulocytes 28 percent.

February 11, 1931. Three hundred cubic centimeters blood transfusion today; temperature has been normal all day; heart rapid; throat and mouth condition very much improved. Anal fissures dressed; appetite shows slight improvement.

R. B. C. 5,420,000; Hb 81 percent. There is a slight variation in size and shape, a rare poorly staining red cell containing basophilic granules present. There is a marked increase of the red cells in number over yesterday, i. e., more than 1,000,000. No nucleated red cells seen.

W. B. C. 2,725. Differential: (Wright's stain), lymphocytes 22 percent, mononuclears 9 percent, metamyelocytes 13 percent, polymorphonuclears 56 percent, divided as follows: Juveniles 2 percent, with 2 segments 19 percent, with three or more segments 35 percent. Goodpastures stain: Granulocytes 74 percent, nongranulocytes 26 percent.

Urine.—Amber, strongly acid, specific gravity 1.012, albumin, faintest trace, sugar negative; negative for arsenic; Bence Jones negative; cerofilm negative. Little mucus, occasional leucocyte.

February 12, 1931. Long bones radiated and small doses ultralight administered; temperature remained normal all day, but pulse remains elevated. Mouth and throat condition subsiding; anal fissures dressed.

Blood count.—R. B. C. 4,960,000; Hb 85 percent, W. B. C. 3,350. Goodpastures stain: Nongranular 32, granular 68 percent. Differential: (Wright's stain), lymphocytes 30 percent, mononuclears 11 percent (these cells show a clear, pale blue zone of cytoplasm, no azure granules seen), myelocytes 2 percent, metamyelocytes 8 percent, juvenile polymorphonuclears 8 percent, segmented (2) 15 percent, segmented (3 or more) 26 percent. In a count of 200 white cells, an occasional red cell with basophilic stippling was noticed, also 10 megaloblasts were counted. No attempt at percentage was made; reticulocyte count averaged 7 percent.

February 13, 1931. Patient had a very comfortable day; appetite is poor; rectal fistula shows some improvement.

Urine.—Amber, acid, specific gravity 1.020, albumin and sugar negative.

R. B. C. 4,720,000; Hb 85 percent. There is slight if any variation in size. The red blood cells look normal. W. B. C. at 8 a. m., 2,150, at 3 p. m., 2,650. Peroxidase stain: Granular 60 percent, nongranular 40 percent. Differential: (Wright's stain) polymorphonuclear leucocytes with two or more segments, 48 percent, juvenile polymorphonuclears 9 percent, lymphocytes 29 percent, metamyelocytes 4 percent, myelocytes 1 percent, large mononuclears 9 percent. There is a definite shift to the more mature polymorphonuclear leucocyte.

February 14, 1931. Temperature 98.6° F., pulse 84, respiration 18. Condition very satisfactory; rectal condition inspected in operating room and found to be healing satisfactorily; appetite is improving.

R. B. C. 4,360,000; Hb 85 percent. There is a dimunition of red blood cells but no marked variation in size and shape or haemoglobin. W. B. C. 2,500. Differential: (Wright's stain), juvenile polymorphonuclears 8 percent, with 2 segments 17 percent, with 3 or more segments 32 percent, large mononuclears 7 percent, lymphocytes 36 percent. Peroxidase stain: Granulocytes 54 percent, nongranulocytes 46 percent. Compared with Peroxidase stain of February 13, 1931, there is a diminution of granulocytes.

Blood culture on dextrose-brain broth, glucose broth, blood agar and plain agar since February 9, 1931, shows no growth.

February 15, 1931. Temperature normal all day; pulse is beginning to drop; patient out in the sun for 25 minutes today. Feels invigorated; appetite better.

R. B. C. 4,740,000; Hb 86 percent, W. B. C. 2,920. Differential: (Wright's stain), polymorphonuclears 51 percent, large mononuclears 4 percent. The polymorphonuclear leucocytes are more mature over yesterday's. The mononuclear leucocytes show a reticulum.

February 16, 1931. Condition very satisfactory; out on deck all the morning.

February 18, 1931. Blood picture shows a gradual improvement; patient's morale is better and he is beginning to show some improvement in his appetite.

February 25, 1931. Patient's general appearance is much better; his strength is slowly returning. A high protein is being administered together with 40 grains of iron daily.

March 1, 1931. A diagnosis of agranulocytosis is proposed and permission has been requested of the Bureau of Medicine and Surgery to establish same.

March 6, 1931. Anal ulcers and carbuncle on buttock have healed; bowels regular; patient eats fairly well.

March 31, 1931. Patient has shown a steady improvement; the blood count is now normal; to duty this date, very much improved.

April 7, 1931. Patient transferred to the San Diego Naval Hospital with the established diagnosis of agranulocytosis. On admission the chief complaints were general malaise and occasional attacks of neuritis in the upper extremities. White blood counts averaged 4,000 with polymorphonuclear percentages 42 to 32 percent. Red cell count 4,000,000 with Hb 80 percent.

Patient remained under supportive treatment and liver extract, with daily blood counts until October 18, 1931, when a return to duty was tried. Readmission was necessary on February 1, 1932, with an aggravation of previous symptoms. Respiratory passages were congested and intestinal tract upset. Courses of leucocytic extract were given but the leucopenia increased to 1,400 with chills and great anxiety of the patient. Blood transfusion, 500 cc was given followed by small doses (one-twentieth)) erythema, over long bones with gradual improvement as indicated in the hemogram, and patient became able to take moderate exercise but did not reach a duty status and was retired on October 1, 1932. Since retirement patient has continued as an outpatient with frequent blood counts and occasional courses of leucocytic extract. K-96 was tried but unfavorable constitutional reactions occurred. The subsequent progress is shown in the hemogram and gradual improvement has occurred as the condition has become more chronic. Yellow bone marrow, tablespoonful t. i. d., is now being tried and a general well being is present at this time, April 1934.

Case 2.—Secondary neutropenia following neoarsephenamine administration. "F. M." fireman third class, United States Navy.

February 1, 1934. Admitted on U. S. S. *Portland* with diagnosis: Syphilis. #1207; due to own misconduct. Entry July 28, 1933, of gonococcus infection. urethra, and entry January 15, 1934, lymphadenitis, right inguinal region. Patient reported to the sick bay with marked enlargement of the right inguinal lymphatic gland and slight serous urethral discharge occurring 6 weeks after exposure. Gram stain negative except for scattered pus cells. Blood Kahn at this time four plus. Congo Red and dark field examinations of urethral discharge are negative for treponema. There is no scar suggestive of a primary lesion about the external genitalia. Repeated blood Kahn four plus. Following second blood test patient developed a macular rash of the trunk, thighs, and arms, typical of secondary luetic infection. Physical examination at this time reveals adenitis of the left cervical glands. In view of the above findings a diagnosis of syphilis is made and antiluetic treatment instituted. No definite foci of infection is determined. It is probable that

the original lesion either existed in conjunction with the recent gonococcus infection or that the urethral discharge mentioned was from an intraurethral chancre of longer standing than admitted by the patient's history.

February 1, 1934. To duty: admitted and discharged for record only.

February 22, 1934. Admitted, contributory disability; U. S. S. *Portland;* diagnosis: Poisoning, acute (arsenic), antisyphilitic. No. 2605. Not due to own misconduct. Classification A. 1. Within command. 2. Not work. 3. Negligence not apparent. 4. Patient sustained poisoning due to injection of neoarsphenamine (Merck & Co., lot no. 846), intravenously in 12 cc distilled water at 8:30 a. m., February 21, 1934. Rate of injection approximately 2 minutes. Patient felt well after injection until about 11:30 p. m., when he awoke feeling chilly, but went back to sleep again and did not report to sick bay until this morning. He then complained of a sore throat, temperature 103° F., pulse 104, respiration 20. Blood pressure 104/60; tonsils and pharynx swollen and injected. Heart, lungs, and abdomen; no abnormal findings; reflexes normal. Differential: Polymorphonuclears none, large lymphocytes 31 percent, small lymphocytes 61 percent, mononuclears 4 percent, eosinophiles none. Hb 70 percent. R. B. C. 5,390,000. Urine: Appearance clear, amber, specific gravity 1.022, sugar negative, albumin slight trace, reaction acid. Microscopical: Few epithelial cells and occasional pus cell. Dicken's test: Slight trace of arsenic. Dicken's test after first injection showed normal elimination of arsenic. From the blood picture it is probable that the patient is developing an aplastic anemia.

February 22, 1934. Transferred to Naval Dispensary, San Pedro, Calif., for treatment.

February 22, 1934. Re-admitted United States Naval Dispensary, San Pedro, Calif. Diagnosis: Poisoning, acute (arsenical), antisyphilitic. No. 2605. Not misconduct. Examination shows slight membrane on left tonsil. General congestion of throat. V. A. (?) Slight bulging of left tonsil (early peri-tonsillar abscess).

February 23, 1934. Condition same; sodium thiosulphate gm 1. intravenously. Dicken's test: Positive.

February 24, 1934. Temperature 103° F. Ulceration left tonsil spreading (mixed infection). Hot MgSo4 gargle q. 2. h. Neosalvarsan and glycerine applications to tonsils discontinued due to positive Dicken's test.

February 25, 1934. Diagnosis changed and admitted. Diagnosis changed to septic sore throat, no. 542, by reason of concurrent disease. Not misconduct. Culture negative for K. L. B. Patient toxic; ulceration on left tonsil continues to spread. Tonsil congested and edematous. Temperature 103.2° F., pulse 120. Sodium thiosulphate gm 1. intravenously.

February 26, 1934. Patient toxic, temperature 104° F. No improvement in condition of throat, ulceration extending. Transferred to the United States Naval Hospital, San Diego, Calif., for further treatment.

February 26, 1934. Readmitted. United States Naval Hospital, San Diego, Calif. Diagnosis: Septic sore throat. No. 542. Not misconduct.

Acute sore throat, chills and fever. On February 22, 1934, patient received an injection of neoarsphenamine. About 4 hours later he had chills, but no other evidence of a reaction. The next morning he complained of a severe sore throat, chills and fever. Differential count showed polymorphonuclears none, large lymphocytes 31 percent, small lymphocytes 61 percent, mononuclears 4 percent, eosinophiles none. R. B. C. 5,390,000. Patient transferred to Naval Dispensary, San Pedro, on February 25, 1934; diagnosis changed to septic sore throat; transferred to this hospital.

Eyes, ears, nose normal; throat-tonsils acutely inflamed; left tonsil has a large ulcer on surface, covered with a white exudate; gums bleeding and show marked infection with V. A. Throat smear loaded with V. A. Chest expansion good, few coarse moist rales heard over both bases; no impairment noted on percussion. Heart: B. P. 115/70, no murmurs noted. Abdomen, G. U., extremities normal. No rash noted. Temperature 104° F. Treatment, 10 percent $CuSo_4$, applied to tonsils twice daily. Sodium perborate, saturated solution, used as a mouth wash every hour. Force fluids.

February 26, 1934. Dicken's test: Negative. R. B. C. 4,410,000. W. B. C. 2,400. Hb 70 percent plus. Differential: Myelocytes 12 percent, juveniles 33 percent, band 7 percent, lymphocytes 47 percent, Turk cell 1. Moderate anisocytosis, slight achromia. Smear for V. A. and K. L. B. loaded with V. A.; no K. L. B. found. Blood type: International type "O".

February 27, 1934. Urine: Albumin positive, 3 plus; sugar negative; numerous coarse granular casts, few leucocytes, few renal and squamous epithelium. W. B. C. 3,350.

February 28, 1934. Treatment continued; patient given intravenous saline with 5 percent glucose. Temperature 103° F. Urine: Albumin positive, 3 plus; sugar negative; some amorphous material, many coarse granular casts, some mucus, few leucocytes. W. B. C. 2,750.

March 1, 1934. Treatment continued; blood picture shows a marked shift to the left on differential count. Patient complains of dull pain on left side of chest. Moderate impairment on percussion over left base. X-ray shows a broncho-pneumonia left side of chest. W. B. C. 3,375. Differential: Myelocytes 21 percent, juvenile 5 percent, band 4 percent, lymphocytes 25 percent, basophiles 1 percent, premyelocytes 21 percent, blast 14 percent, immature lymphocytes 16 percent.

March 1, 1934. Diagnosis changed and admitted, contributory disability; diagnosis pneumonia, broncho. No. 811. Not misconduct; complication. Did not exist prior to enlistment; transferred to medical service for further treatment.

March 1, 1934. Received from eye, ear, nose, and throat service; condition very poor. W. B. C. 1,000. Placed on serious list, 400 cc 5 percent glucose in saline given.

March 2, 1934. Evidence of broncho-pneumonic infiltration both bases; heart action fair; general condition somewhat worse; leucocytic extract, intramuscularly, 5 cc every 4 hours. X-ray chest, bedside: "The heart shadow is central, upper limits of size. There is a broncho-pneumonic exudate throughout the left lower lobe. There is also a beginning exudate right mid lobe. Impression: Bilateral broncho pneumonia." 3 p. m., W. B. C., 1,000. Differential: Myelocytes 21 percent, band 8 percent, small lymphocytes 51 percent, large lymphocytes 6 percent, metas 1 percent, blast 6 percent. 10 p. m., W. B. C. 1,500. Differential: Myelocytes 22 percent, juveniles 9 percent, band 8 percent, lymphocytes 58 percent, basophiles 1 percent, Turk 2 percent. R. B. C. 3,750,000. Hb 80 percent.

March 3, 1934. About the same; blood picture shows slight improvement; W. B. C. 2,800. Differential: Myelocytes 28 percent, juveniles 18 percent, band 16 percent, lymphocytes 28 percent, basophiles 2 percent, Turk 8 percent, R. B. C. 4,180,000; 2 p. m., W. B. C. 1,460. Differential: Myelocytes 31 percent, juveniles 4 percent, band 11 percent, lymphocytes 47 percent, Turk 1 percent, blast 2 percent. Hb 80 percent.

March 4, 1934. Apparently somewhat improved; W. B. C. 6,050. Differential: Myelocytes 18 percent, juveniles 20 percent, band 14 percent, lympho-

cytes 42 percent, basophiles 2 percent, Turk 2 percent, normoblast 1 percent. 8 p. m., W. B. C. 8,500. Differential: Myeloblast 3 percent, myelocytes 5 percent, juveniles 17 percent, band 29 percent, eosinophiles 2 percent, mononuclears 6 percent, Turk 2 percent.

March 5, 1934. Blood picture much improved; ulcer on left tonsil healing rapidly; right side of chest apparently clearing. Three p. m., W. B. C. 12,200. Differential: Myelocytes 8 percent, juveniles 10 percent, band 51 percent, segmented 14 percent, lymphocytes 13 percent, mononuclears 1 percent, premyelocytes 3 percent.

March 6, 1934. Eight a. m., R. B. C. 4,500,000. W. B. C. 10,600. Differential: Premyelocytes 10 percent, myelocytes 16 percent, juveniles 16 percent, band 32 percent, segmented 9 percent, lymphocytes 13 percent, mononuclears 1 percent 8 p. m., W. B. C. 10,800. Differential: Premyelocytes 1 percent, myelocytes 4 percent, juveniles 7 percent, band 23 percent, segmented 28 percent, lymphocytes 35 percent, eosinophiles 1 percent, unclassified 2 percent. Smear of sputum for typing: Smear—predominate organisms, streptococci; mouse inoculated, no ill effects. No pneumococci found in peritoneum. Ten p. m., W. B. C. 15,150. Differential: Juveniles 5 percent, band 15 percent, segmented 61 percent, lymphocytes 16 percent, Turk 3 percent. X-ray chest, bedside: "Check bedside plate taken March 1, 1934, shows an advancing process. The right mid and right lower lobes now show extensive pneumonia. The left chest—The broncho pneumonia is still apparently confined to the left lower lobe."

March 7, 1934. General condition much improved. Apparently patient had pseudo-crisis this morning. Highest white blood count 20,000. Eight a. m., R. B. C. 4,020,000. W. B. C. 14,800. Hb 70 percent (Newcomer). Differential: Juveniles 2 percent, band 22 percent, segmented 54 percent, lymphocytes 18 percent, eosinophiles 1 percent, mononuclears 3 percent. Eight p. m., W. B. C. 20,000. Differential: Myelocytes 2 percent, juveniles 14 percent, band 16 percent, segmented 52 percent, lymphocytes 16 percent.

March 8, 1934. Throat ulcer practically healed; intravenous glucose and saline discontinued. Leucocytic extract b. i. d. Eight a. m., R. B. C. 3,410,000. W. B. C. 12,750. Hb 70 percent (Newcomer). Differential: Myelocytes 1 percent, juveniles 5 percent, band 26 percent, segmented 49 percent, lymphocytes 11 percent, basophiles 1 percent, mononuclears 5 percent, Turk 1 percent, blast 1 percent. Eight p. m. W. B. C. 15,900. Differential: Myelocytes 1 percent, juveniles 9 percent, band 21 percent, segmented 57 percent, lymphocytes 11 percent, mononuclears 2 percent.

March 9, 1934. General condition much improved; appetite returning; cough improving. Fair amount of sputum; W. B. C. holding around 15,000 plus; leucocytic extract discontinued. Eight a. m., R. B. C. 4,500,000. W. B. C. 15,950. Hb 81 percent (Newcomer). Differential: Band 19 percent, segmented 61 percent, lymphocytes 13 percent, eosinophiles 2 percent, basophiles 1 percent, mononuclears 4 percent. Eight p. m., W. B. C. 13,700. Differential: Myelocytes 1 percent, juveniles 7 percent, band 27 percent, segmented 43 percent, lymphocytes 20 percent, mononuclears 2 percent.

March 10, 1934. Chest examination—Few scattered rales, right base, slight impairment of percussion, breath sounds come through. Apparently slight enlargement of cardiac dullness; aortic and mitral areas O. K. Slight accentuation of first sound at apex. Few scattered rales left base. B. P. 122/66. Eight a. m., R. B. C. 4,120,000. W. B. C. 19,500 Hb 80 percent. Differential: Myelocytes 2 percent, juveniles 4 percent, band 14 percent, segmented 60 percent, lymphocytes 14 percent, eosinophiles 1 percent, basophiles 2 percent. Eight p. m.,

W. B. C. 15,250. Differential: Myeloblasts 2 percent, juveniles 2 percent, band 8 percent, segmented 50 percent, lymphocytes 37 percent, eosinophiles 1 percent. X-ray chest: "Check plate as compared with the plate taken March 5, 1934, shows there is a noticeable resolution of the broncho pneumonic exudate left lung and there is a partial resolution of the pneumonic exudate, right lung."

March 11, 1934. Eight a. m., W. B. C. 17,400. Differential: Myeloblast 1 percent, juveniles 7 percent, band 5 percent, segmented 73 percent, lymphocytes 13 percent, eosinophiles 1 percent.

March 12, 1934. Kahn test 4 plus. Eight a. m., R. B. C. 4,160,000. W. B. C. 15,800. Hb 80 percent. Differential: Juveniles 4 percent, band 11 percent, segmented 61 percent, lymphocytes 18 percent, eosinophiles 1 percent, basophiles 1 percent, mononuclears 3 percent. Cardiac examination: "There is both a pericardical and bronchial friction rub, loudest at the apex, a soft blowing double mitral murmur, to and fro, heard over base of heart, but not transmitted, both A_2 and P_2 accentuated. The same double murmur is heard at right sternal margin, probably transmitted from mitral area. Recommend complete rest for at least another month, probably small doses of digitalis, M X of the tincture. Would like to get electro-cardiagram if he can be safely transported to medical one on wheel stretcher."

March 13, 1934. Pulse 84; patient is doing very well. He has a good appetite, white count remains about the same.

March 14, 1934. R. B. C. 4,290,000. W. B. C. 12,250. Differential: Polymorphonuclears 76 percent, lymphocytes 18 percent, mononuclears 5 percent, transitionals 1 percent.

March 15, 1934. Patient sent to medical one for electrocardiogram. He was tired after being moved and sweat a great deal; 8 a. m., R. B. C. 3,960,000; W. B. C. 10,700; Hb 85 percent. Differential: Band 16 percent, segmented 55 percent, lymphocytes 25 percent, eosinophiles 1 percent, basophiles 1 percent, mononuclears 2 percent. Electro-cardiogram—1. Sinus tachycardia with a rate of 116. 2. T-1 segments 1 and 2 one mm elevation above base line. Otherwise tracing within normal limits.

March 16, 1934. Pleural rub still heard over lower left lung, pericardial rub still present; R. B. C. 4,380,000; W. B. C. 10,750; Hb 75 percent. Differential: Juveniles 4 percent, band 27 percent, segmented 48 percent, lymphocytes 16 percent, eosinophiles 1 percent, mononuclears 5 percent.

March 17, 1934. R. B. C. 4,260,000; W. B. C. 7,800; Hb 75 percent. Differential: Juveniles 26 percent, segmented 45 percent, lymphocytes 24 percent, eosinophiles 2 percent.

March 18, 1934. Patient complains of precordial pain on coughing; W. B. C. 11,600. Differential: Juveniles 1 percent, band 14 percent, segmented 57 percent, lymphocytes 25 percent, eosinophiles 1 percent, mononuclears 2 percent.

March 19, 1934. R. B. C. 4,350,000; W. B. C. 11,600; Hb 80 percent. Differential: Juveniles 1 percent, band 22 percent, segmented 53 percent, lymphocites 21 percent, eosinophiles 1 percent, basophiles 1 percent, mononuclears 1 percent.

March 20, 1934. Precordial pain still present; left base is dull on percussion; pericardial rub and many rales heard; X-ray chest bedside: "Check plate as compared with the plate taken March 9, 1934 shows a change in the appearance of the heart shadow. It is now nearer "jug" shaped, with broadening of the base. The left border is convex, apex well to the left. There is still some unresolved pneumonia exudate, right mid lobe and probably left base. The heart shadow is now suggestive of a possible pericardial effusion."

March 21, 1934. Condition still the same.

March 22, 1934. Patient states he had the best night he has had since he has been sick. W. B. C. 8,450.

March 23, 1934. Patient has had another good night. W. B. C. 6,400. Differential: Juveniles 1 percent, band 19 percent, segmented 54 percent, lymphocytes 21 percent, eosinophiles 3 percent, mononuclears 2 percent.

April 1, 1934. Diagnosis changed and readmitted with original diagnosis of syphilis. Transferred to urology for further treatment.

COMMENT

Two cases of granulocytopenia are presented—one of primary origin and the other a secondary neutropenia following an infection.

As typical of many primary cases reported, the onset of case 1' dates from the extraction of teeth. No definite conclusions can be drawn from the early drug therapy as an etiological factor. It will be noted that amytal and amidopyrine were occasionally taken in small doses as dental and neuritic sedatives, but the patient was unable to state the total amounts. Following the various reports against amidopyrine and the barbiturates, Renznikoff, of Cornell University, made a special study of the compiled cases for the council on pharmacy and chemistry of the American Medical Association. He found no evidence of a barbiturate alone producing a granulopenia, but amidopyrine is an offender in special cases, probably due to a sensitivity of the individual. It is not a specific, as few cases of white cell disturbance are reported in comparison to the enormous amount of the drug consumed. All patients who take or are given amidopyrine are already suffering from some complaint or illness. One case of granulopenia is cited who had never taken drugs of any kind prior to recurrent attacks and yet recovered from the last attack in spite of the administration of amidopyrine throughout the acute phase of illness. Reznikoff recommends the drug should be stopped if any rash, dizziness, or chills appear; in the absence of symptoms, periodic white blood cell and differential counts should be made. Selt administration is emphasized as particularly dangerous.

The graph of the blood studies covers a period of 3 years following the acute onset. The recurrent attack is shown in March 1932, 13 months after the original one, responding to transfusion, X-rays, and leucocytic extract. Periods of fatigue and low spirits were present when the polymorphonuclear cells and lymphocytes approached each other or became reversed, but fair health has been experienced during the past 2 years with a mean average leucocyte count of 4,000 and may be considered a chronic neutropenic state. The shifting total white count has been purely a shift of granulocytes.

Case 2 is one of secondary neutropenia brought on by one of the bone marrow depressants, neoarsphenamine, which contains the benzine ring in its basic structure. While leucocytic extract administration was followed by a prompt leucocytosis, viz, 1,000 to 20,000, in

4 days, it is more probably the result of the pneumonia infection. Kracke is a strong advocate of infection being the best treatment of granulopenia, and while dangerous we may logically conclude this patient's life was saved by his pneumonia.

With the early recognition of a relative leucopenia and a full appreciation of the serious aspects of any disturbance of the neutrophils, it is believed the prompt application of accepted therapy will markedly lower the mortality figures of the future.

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REPORT OF SO-CALLED EPIDEMIC OF GLANDULAR FEVER (INFECTIOUS MONONUCLEOSIS)[1]

By ROGER A. NOLAN, Lieutenant Commander, Medical Corps, United States Navy

The diagnosis of glandular fever may be made by the clinical symptoms and blood picture—but now we have a confirmatory test which is most illuminating. This test was developed by Paul and Bunnell in 1932 while working on antigens and their antibodies and

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the agglutination of sheep cells—using serum from a near-by hospital as controls. They noted that one of the human serums agglutinated sheep cells in abnormally high dilutions. Upon investigating the source of this one serum specimen it was found to be from a case of glandular fever. This finding was checked and rechecked—always with the same results. The following year (1933) they used the serum from some 1,700 control cases, namely: Tuberculosis, syphilis, cervical adenitis, the leukemias, Hodgkins disease, and others, only to find them negative. But the blood from uncomplicated glandular fever cases in 98 percent read positive.

As this article is being written and submitted, here in Coronado, Calif., an epidemic of such proportions (to date, 220 cases) prevails that it afforded the opportunity of using the above test. Twenty convalescent children reported with their parents to the clinic and in each case 1 cc of blood was drawn from the arm. Blood from 20 others who were bed patients was secured at their respective homes. A series of control cases, namely: Whooping cough, mumps, syphilis, measles, catarrhal fever, chronic cervical adenitis, and normal infant blood was also collected. Dilutions of 1 cc, of 1:20, 1:40, 1:80, 1:160, etc., using 0.1 cc serum in the first tube of 2 cc of saline and carrying over 1 cc of the mixture into the succeeding tubes, each one of which contains 1 cc of saline. Then to each tube is added 0.1 cc of a 1 percent sheep cell suspension; incubation 1 hour at 37° and ice box over night. Read the following morning; the agglutination is very definite and characteristic. In all our cases of uncomplicated glandular fever agglutination was obtained in dilutions from 1–80, to 1–320; in the control cases agglutinations were noted at 1–40 or below. This has proven to us that clinical glandular fever is a real entity and its presence in a community may be detected by this simple laboratory test.

(1) The early cases were first noted in Coronado 10 days after the arrival of the fleet from the east coast on November 9, 1934, in the homes of officers and men who were given leave. Many were suffering from head colds, etc., and it is said that some 500 cases of mild influenza were noted in the fleet on their trip up from Panama to Coronado, San Diego area.

(2) The number of cases from 6 months to 5 years of age, 55; the number of cases from 5 years to 13 years, 160; and 5 adults (2 males and 3 females); total, 220.

(3) The symptom complex noted in the present epidemic is distinctive. In the cases from 6 months to 5 years the onset was noted by the parent; in those between the ages of 5 to 13 years the onset was noted by the parent or school teacher, and was aptly termed "a change in the personal characteristics." Among the younger

children, some were cranky, others fretful—not wanting to play, preferring to lie about; this sign in most cases was constant. The changed mannerisms lasted from 3 to 5 days and then a series of symptoms developed of such moment that a doctor was called to the home.

(4) The symptomatology varied but little in the 220 cases.

(*a*) Fever was constant symptom 100 percent, ranging from 100° to 104°.

(*b*) Pain in the back of neck, 80 percent.

(*c*) Vomiting, 10 percent.

(*d*) Abdominal discomfort, 20 percent.

(*e*) Blood counts, total, red, white, and differential were made on over 50 cases. The persistent picture was this:

(1) Red counts normal or nearly so, 100 percent.

(2) Hyperleucocytosis, 40 percent as high as 28,000.

(3) Leucopenia, 60 percent as low as 4,000.

(4) Startling increase of abnormal lymphocytes in the blood in all cases.

(5) Presence of abnormal monocytes seen in 80 percent.

(6) Lymphoblasts in abnormal amounts in 35 percent.

SYMPTOMS

(*a*) *Fever.*—The temperature was taken in many cases by the parents who became alarmed at the sudden rise. In 95 percent of all cases this initial rise of temperature always occurred at about 4 p. m. No chill preceded the rise in temperature. The patients became flushed, were lackadaisical for 3 days prior to the onset; initial rise of temperature was always over 100°, and in many cases reached 104°. The patients were dopey at this time; sallowness and a pinched expression of the face were noted in those over 4 years of age.

(*b*) *Pain in back of neck.*—The symptom of pain in the back of the neck was investigated and in all of the cases it was due to cervical myositis shown by the exaggeration of pain when patients voluntarily or involuntarily rotated the head on the neck. The reason for this myositis is easily traceable to the infallible diagnostic symptom, namely, a particular type of adenopathy of the posterior cervical and glands of the neck. In differentiating this adenopathy from other types, canary bird, feathery, palpation is essential, so that the small shoe-botton glands may be made out bilaterally numbering from 15 to 40, by running the finger over the mastoid process down to the acromio-clavicular joint. These glands are identical in feel with those of the glands felt in lues with the exception that they are mildly painful. This finding is now further checked by extending

the lower limbs of the children and finding the same glandular condition in both groins and axilla. This glandular condition is persistent for a period of 1 month or more after the onset of convalescence.

(*c*) *Vomiting.*—The symptom of vomiting occurred in about 10 percent of all the cases and followed the rise in temperature. It was not severe and the withdrawal of cold drinks and solid foods, giving no cathartics, appeared to control this symptom and differentiated it from the vomiting seen in enteritis. There was no pain, tenderness or rigidity of the abdominal musculature. The abdominal discomfort may be due to the involvement of the mesenteric glands and spleen and is apparently reflex in character. Have looked constantly for enlarged spleens but have failed to find any.

(5) *Interesting complications with notations.*—In five cases a rash was noted over the body, extremities, and face. In each of these cases, a prior history of attacks of urticaria was elicited; the rash was fleeting but did not appear to be true urticaria. In 60 percent of all cases, for 2 nights after fever appeared, sleep was restless. Dreaming was constant with mild delirium. In four cases a mild otitis media of 24-hours duration was noted. In two cases a mild conjunctivitis and photophobia was noted. In 28 cases a brassy cough existed for a few days. This was thought to be due to peribronchial glandular involvement. The urine from 40 cases was examined and found to be negative for acetone.

One case developed a mild stomatitis of 8 days' duration. Recovery in all cases to a normal status within 3 weeks has been noted. The blood picture, agglutination test, and noticeable specific adenopathy, all modified, remain however for an undetermined time.

It is highly infectious for all children under 13 years of age. It must be differentiated from The Exanthemata, catarrhal fever acute, meningitis, influenza, tonsilitis, and diphtheria.

Glandular fever in a baby, child, or adult seen for the first time is difficult to diagnose. When seen in epidemic form it is easily noted and is a pleasant condition to treat, for the aftermaths are few if any. In severe adult cases it must be differentiated from typhoid, diphtheria, lymphatic leukemia, common cold, tuberculosis, syphilis, influenza and acute catarrhal fever.

(6) *Treatment.*—(*a*) No drug that I know of influences the duration or course of this disease.

(*b*) No heat or hot applications to glands.

(*c*) No hot baths in the acute stages.

(*d*) Temperature of room should be between 65° and 70°.

(*e*) No rubbing, massaging or applications in or about the cervical or inguinal glands.

(*f*) When convalescent, no violent exercise involving strenuous stress about the groin or neck.

(*g*) No catharsis.

(*h*) Cod liver oil and sunshine advised for all convalescent patients.

(7) *Examples.*—(*a*) A child, 5 years of age developed a toothache. The mother put a hot water bag to the child's jaw. The cervical glands on that side were enlarged and became extremely painful. The blood count jumped and the temperature rose.

(*b*) A child while on fifth day of convalescence was compelled by the mother to attend her acrobatic dancing school. Hand stands, etc., were the order of the day. That night all the glands in her neck were enlarged and painful. Temperature and blood count rose and the patient suffered a definite setback.

(*c*) Because some 18 cases complained of pain in the back of the neck, mothers rubbed that part with salves, ointments, etc., and in all of these cases rise in temperature, enlargement of glands and rise in white count was noted.

(*d*) High temperatures in rooms—75° to 82° is considered harmful. In these cases convalescence was protracted, also in some few cases where solicitous mothers had given children hot tub baths.

(7) *Questions.*—(*a*) If this simple agglutination test backs up the clinical picture of epidemic glandular fever and proves it to be a definite entity then hemotological investigation in the so-called " mild epidemics of catarrhal fever " acute and influenza is to be considered and encouraged, for it is a quick step in differentiation.

(*b*) Is epidemic glandular fever in children a blessing in disguise? Does it mean a percentage of immunity against the dreaded future pandemic of influenza vera?

(9) *General statements.*—(*a*) Etiology to date unknown.

(*b*) Pathology indefinite.

(*c*) Apparent sequellae, none.

(*d*) Death rate nil if uncomplicated.

(*e*) Blood picture in epidemics definite.

(*f*) Agglutination test is a remarkable check on clinical picture.

(*g*) Introduction of this disease into a juvenile community is preceded by a mild epidemic of influenza in contact adult population.

(*h*) Influenza may demonstrate itself in the adult household following a case of glandular fever in the child.

HEMATEMESIS

By JAMES D. RIVES, Lieutenant Commander, Medical Corps, United States Navy

The practicing physician will be faced, sooner or later, with the problem of the diagnosis and the treatment of hematemesis. It is, of course, not a disease of itself, but since it is so spectacular, and brings consternation and fear of death to the patient, it will not be wasted time to spend a moment in its contemplation.

What, then, are the causes of this condition? One classification of the etiology (1) considers the following:

(*a*) *Trauma.*—By trauma is meant injury to the stomach by violence or by foreign substances introduced into it. Injury to the stomach sufficient to cause hemorrhage has been noted following a severe blow upon the abdominal wall over the stomach, even though the abdominal skin showed no sign of injury. Naturally the already ulcerated stomach would be much more likely to start bleeding following such a blow. Usually the history of the case will point out such an accident and clarify the diagnosis. Injuries due to gunshot or stab wounds are so obvious as to cause no difficulty in their recognition. At times foreign substances, such as glass, nails, pins, and so forth, are swallowed, either accidentally or deliberately, and may cause lacerations of the esophageal or gastric mucosa, causing hemorrhage which may be vomited. Here, if the history of the case is obscured, the diagnosis may be possibly cleared by the X-ray.

(*b*) *Corrosive poisons.*—Certain corrosive poisons, e. g., phosphorus, mercury cresol, etc., may cause hematemesis. Since bichloride of mercury is so frequently used by those attempting suicide, it is of importance in the consideration of the causation of hematemesis.

(*c*) *Ulcerative lesions of the stomach.*—These are known to be the most frequent cause of gastric hemorrhage. Statistics compiled at the Mayo Clinic place gastric and duodenal ulcers as the cause in 80 percent of gastrointestinal bleeding. (2) In a series of 668 cases, Rivers and Wilbur (3) show that peptic ulcer, gastric carcinoma, inflammatory processes and benign tumors accounted for 90 percent of hematemesis. Included in the classification of gastric ulcerative lesions are neoplasms, which may be benign, as hemangioma, polypi; or malignant, as sarcoma, carcinoma. Tuberculosis and syphilitic lesions may cause gastric bleeding, and occasionally syphilis may cause severe hematemesis (1), although Rivers (4) questions that syphilitic lesions are, per se, responsible, rather placing the blame on liver lesions.

(*d*) *Hepatic diseases.*—In cases of cirrhosis of the liver there is very great congestion of the portal circulation, causing an attempt at the establishment of a collateral circulation by the anastomosing of the veins of the lower part of the esophagus and the coronary veins of the stomach, which form large varicose trunks. (5) As the pressure increases one or more of these varicositie may rupture. The hemorrhage may come from either the stomach or the esophagus, but, in either case, the first symptom often will be a startling hematemesis. Cecil (6) states that about 25 percent of patients with liver cirrhosis vomit blood and that, of these, about one-fifth die from the hemorrhage. At times an ulcer may be situated over one of these engorged veins, and, the vein becoming eroded, drains the patient's blood into his stomach, whence it is repeatedly vomited until he dies. At other times the blood in the stomach simply oozes from numerous points on the congested mucosa. Large and serious hemorrhages can occur in this manner, and when the surgeon opens such a stomach at operation, he is very much chagrined at finding no visible point of bleeding to ligate. The same may be said of a ruptured esophageal varix.

(*e*) *Infectious diseases.*—Yellow fever, pneumococcus infections, septicemia, measles, smallpox, and scarlet fever may cause hemorrhage into the stomach. Any disease causing a severe toxemia may do the same. The black vomit is one of the prominent features of yellow fever and at times this may occur in severe cases of measles and smallpox (the so-called black variety).

(*f*) *Blood dyscrasias.*—Hematemesis is, at times, encountered in such conditions as pernicious anemia, leukemia, purpura, and hemophilia. Rivers and Wilbur (4) found that these conditions occurred, in the series of cases studied by them, less frequently than is popularly supposed to be the case. In their cases of hematemesis they found only one case of hemorrhagic purpura, one of hemolytic icterus, and one of hemophilia, and in the case of hemophilia there was clinical evidence of ulcer sufficient to lead them to conclude that the hematemesis was probably instituted in an nitrinsic gastric or duodenal lesion and maintained because of the hemophilia.

(*g*) *Cardiopathies.*—Congestion of the gastric circulation, due to cardiac valvular insufficiencies with decompensation and portal congestion, could cause hematemesis in the same manner as that due to portal cirrhosis.

Diagnosis.—As in all except the most obvious cases a correct diagnosis can be arrived at only by means of an exhaustive and detailed history, followed by a careful physical examination of the patient, and this information correlated with that obtained from laboratory examinations of certain materials, e. g., body fluids, excreta, and so

forth. This information must be further applied to what we may call an expectancy table and the deductions drawn from these. By expectancy table is meant a tabulation of the findings reported by observers of the condition under consideration; this then becoming the list of possibilities. While there may be slight differences in the figures reported by different men, all seem to be agreed that the causes of hematemesis will fall into 3 groups. These, as classified by Rivers and Wilbur (4), are:

Group 1. Intrinsic gastric and duodenal lesions.

Group 2. Cirrhosis and splenic anemia.

Group 3. All other causes.

The percentage incidence of these groups, in their cases, was as follows: Group 1, 90.5 percent; group 2, 5.1 percent and group 3, 4.5 percent.

Of group 1 more than 75 percent were caused by duodenal, anastomotic or benign gastric ulcer. Duodenal ulcer alone caused more than 50 percent. They also note that carcinoma rarely causes gross hemorrhage, but that the bleeding is more in the nature of a slow ooze.

In view of the fact that gastric and duodenal ulcer are found to be the cause of hematemesis oftener than any other condition, we shall briefly point out the high lights of their symptomatology. First, attention is called to the fact that there are cases of gastric or duodenal ulcer who have never had a symptom. Cases have been reported time and again, where the first and only symptom was a sudden and ofter very serious hematemesis. Most cases, however, give a history of chronic indigestion, usually associated with varying degrees of gastric discomfort, this having, as a rule, the character of pain which is of a gnawing or aching quality, rather than sharp. This discomfort is periodic and, as a rule, bears a definite relation to the time of taking food. In the case of duodenal ulcer, the pain and discomfort come on from one to one and a quarter hours after eating, and last until the next meal. The pain is absent when the stomach contains food, and reappears when the stomach is empty. In gastric ulcer the pain comes on earlier following the taking of food, and then gradually fades away before the next meal. There may now ensue a period lasting from a few days to weeks or even months, during which the patient is practically symptom free, only to have the symptoms return as before or in aggravated form. Associated with this pain the examiner will find that there is generally an area or spot of epigastric tenderness. There may be referred pain felt in the back, at a level and on the side corresponding to the location of pain as occurring anteriorly. Some patients will, at times, be much tormented by nausea and

vomiting. This is usually due to spasm of the pylorus, caused by the ulcer. From time to time some cases will show a "tarry" stool, which, if bismuth and iron preparations have not been taken, will indicate that the patient has passed blood from the stomach or duodenum through the intestine. Hematemesis occurring in a patient presenting such a history should not keep one long in doubt as to the probable source of the bleeding. One should mention the importance of ruling out of the picture the possibility that swallowed blood could be the cause of the hematemesis. A close examination of the suspected or possible points of extra gastric bleeding will suffice to determine which, if any, of these is responsible for hematemesis due to swallowed blood, although it may be necessary to use the esophagoscope to determine the bleeding point in a lesion of the esophagus.

The next most frequent cause of hematemesis is portal cirrhosis. Here the diagnosis is more difficult, due to the insidious onset and the difficulty in getting an accurate history. A history of chronic indigestion, loss of appetite, attacks of nausea and vomiting—especially morning vomiting—occurring in a person who has been addicted to the intemperate use of alcoholics and who has pampered his appetite, might indicate an early portal cirrhosis. Of course the diagnosis is much easier after the obstruction has progressed to a point where the patient has ascites and an enlarged and tender liver. Unfortunately many cases of portal cirrhosis are not diagnosed ante mortem.

The diagnosis of hematemesis due to the results of trauma and the action of corrosive poisons offers no great obstacle, once a good history has been obtained. Hematemesis accompanying acute infectious diseases will be recognized as such from the symptoms of the underlying condition. Acute hemorrhage due to blood dyscrasias is of course recognizable by the blood pictures peculiar to each. In the majority of cases the correct diagnosis of the cause of the hematemesis is made after the acute condition has subsided and the physician has had time to go into nicer detail of history and physical examination, because in such cases the first aim must be to stop the hemorrhage.

This brings us to a consideration of what to do for these patients. There is a sharp division of professional opinion as to the best mode of treatment for gastric bleeding. One (2) states that it should be almost a surgical axiom that exploration be carried out in such cases, unless there is "definite and exact" contraindication to it. On the other hand, another (7) says, "Do little." He would give rest in bed with nothing by mouth, large doses of morphia, and supportive treatment in the form of glucose and saline, and, when indicated,

small transfusions. The thought expressed by one of our colleagues (8) seems to cover the situation as to whether to operate or not. He says, "Our belief is that either one of these rules, if rigidly followed, will show as many failures as the other * * *. Two safe rules can be given. Death from the first hemorrhage is rare. Repeated hemorrhages demand surgery."

The medical treatment used in cases of hematemesis seen at this hospital has been as follows:

1. Patient treated as a strictly bed patient. Foot of bed elevated for shock.
2. Nothing by mouth if there is still bleeding.
3. Morphine sulph. gr. ¼ every 4 hours. Discontinue if the respirations drop to 10.
4. Saline proctoclysis for supplying fluids and chlorides.
5. Glucose 5 percent in normal saline, intravenously, for nourishment and sustaining body fluids and chlorides.
6. Ice cap to epigastrium.
7. General warmth.

The surgical service has been called on for small blood transfusions from time to time, as indicated.

On admission the following laboratory procedures are carried out: Specimen of blood for (a) red cell count, (b) white cell count, (c) hemoglobin estimation, (d) coagulation time, (e) bleeding time. These are repeated as considered necessary. All patients with hematemesis are blood typed and cross matched for transfusion, should this become necessary. Urinalysis.

In addition to the above, the following have been administered to hasten the clotting time of the blood: (a) thromboplastin, (b) calcium chloride, and (c) 30 cc of a 30 percent solution of sodium citrate. Thromboplastin administered subcutaneously has shown apparently no beneficial effect. Calcium chloride given intravenously was no better. However, the sodium citrate solution given intramuscularly decreased the clotting time by one-half. It should be given to a conscious patient only after some anaesthetizing procedure has been accomplished at the site of the injection, as it is very painful.

The prognosis is, of necessity, the prognosis of the underlying disease or condition, as hematemesis of itself is rarely fatal. (5)

Case 1.—J. M. T., SM2c, U. S. N., emergency admission at 1,000, June 18, 1934, age 29, single.

C. C.: "Vomited blood."

P. I.: Patient states that he was on leave and went to bed last night feeling well, but was restless and tossed all night. He was awakened by a feeling of nausea at 0800, on June 18, 1934, and found that he was covered from head to

foot in blood, which he believes he vomited just before he awakened. Says he got up and tried to go to the toilet, but being unable to get there, called for help. A civilian doctor was called, and he administered a hypodermic injection and had the patient sent to the naval hospital.

F. H.: Negative.

P. H.: Usual childhood diseases, including scarlet fever; gonococcus infection, urethra in 1930; was treated for this at the naval hospital, Canacao, P. I. During the treatment he developed urethral strictures, for which sounds were passed; he also developed gonorrheal rheumatism; states that he has occasional nocturia; denies lues; states that he has had attacks of gastritis since 1928, and that at intervals he would get attacks of severe epigastric pains lasting several days; in these attacks he would lose his appetite; eating or smoking aggravated this condition; last attack was 1 year ago.

P. E.: Patient is a well nourished white male; pale skin; restless and apprehensive, apparently showing the effect of the morphia administered before admittance to the hospital; no glandular enlargements noted; lips somewhat pale but still quite pink; chest full and round; no rales heard; normal resonance; heart: Rate 70, regular rhythm, no murmurs heard; blood pressure 95/50; abdomen: soft, no tender spots (palpation very guarded); extremities and genitalia normal.

The patient was put to bed as a strictly bed patient, and saline proctoclysis started; morphine sulphate, gr. 1/4 q 4 h. was ordered, and the patient was put on the serious list.

R. B. C. on admission was 2,800,000, hemoglobin 65 percent. At noon, he vomited 400 cc of bloody material; he was given 20 cc of thromboplastin subcutaneously; at 1930 vomited 100 cc bloody material; this was repeated at 2045 and at 2110; thromboplastin was also repeated.

June 19, 1934. Vomited 100 cc blood at 0600; proctoclysis continued; morphine discontinued because of lowered respiratory rate; 0930 began to hiccough; 1045 morphia gr. 1/8; hiccoughs slightly diminished; P. 72; 1100 passed large amount blood by rectum, also vomited 100 cc blood; hiccough improved; 1110 vomited 410 cc bright blood; 1125 vomited 410 cc bright blood; 1200 P. 82, R. 14; 1225 vomited 150 cc clotted blood; calcium chloride intravenously; T. 97, P. 80, R. 14; 1500 transfusion 250 cc blood; much improved; 1530 placed on critical list; 2400 vomited 400 cc bright blood following a deep cough.

June 20, 1934. Improved; had a good night; 1030 hypodermoclysis 500 cc of normal saline; 1400 P. 92, R. 10; 1415 intravenous saline with 5 percent glucose given; 1435 hypodermoclysis normal saline; 1800 P. 100, R. 12; 1900 patient irrational; 1915 vomited 200 cc bright blood; 1945 vomited 300 cc blood; 2000 vomited 300 cc blood; 2030 transfusion 250 cc blood in emergency; R. B. C. 1,510,000, hemoglobin 45 percent; 2200 P. 108, R. 10; 2400 P. 110, R. 14.

June 21, 1934. Small hemorrhages at 1345, 1400, and 1410, P. 120, R. 16; 0400 P. 120, R. 12; 0600 P. 134, R. 12; 0900, 30 cc of a 30 percent solution of sodium citrate given in the gluteal muscles. The bleeding time was lowered from 4½ to 2½ minutes in 2 hours; 1400 P. 160, R. 22; patient delirious.

June 22, 1934: Expired at 0415.

Autopsy report.—The body is that of a white male of about 31 years of age, well developed, and nourished. There is a generalized slight icterus.

Lungs: Left weighs 570 and and right 720 g; patches of early bronchopneumonia in the dependent portions and slight edema are present in both.

Heart: Not important.

Liver: Weighs 1560 g; its capsule is slightly thicker than normal. Evidence of periportal fibrosis is present. Numerous small nodules varying in size from pin point to 6 mm in diameter are present.

Spleen: Weighs 490 g; no adhesions are present at its site; there is a large group of blood vessels leading to and from the organ; the veins especially are greatly dilated and tortuous; they invariably drain into the splenic vein which is largely dilated, but apparently without any anatomical changes; on section the organ has a beefy appearance; the splenic corpuscles are greatly reduced in number and fibrous tissue content is increased; the splenic juice is markedly decreased in volume; on excision there is no shrinkage of the organ.

Stomach and intestines: Many dilated and varicose veins travel along the walls of the cardiac end of the stomarch and its adjoining esophagus. These, when traced, are seen to empty into the vasa brevia veins and into the splenic vein which is widely dilated. Stomach and intestines are moderately dilated and when opened show presence of a large quantity of blood undergoing decomposition. On washing away the blood and gastric contents, a 0.5 cm ulceration is found 7.5 cm below the distal end of the esophagus on the ventral wall of the body of the stomach, close to its left lateral margin. This ulceration on inspection is seen to enter a large submucous cardiac vein. This is the site of hemorrhage. The remainder of the gastrointestinal tract is negative except for the autolysis that had taken place after death.

Pathological findings.—1. Cirrhosis, liver, portal. 2. Hemorrhage, gastric, due to ulceration and rupture of a varix. 3. Parynchymatous changes in solid viscera.

Case 2.—A retired lieutenant commander was admitted to the United States Naval Hospital at San Diego on May 11, 1925, for the treatment of appendicitis; chronic.

F. H.: Negative.

P. H.: Measles, scarlet fever, diphtheria, pneumonia, mumps, influenza, dengue, and malaria. Not strong as a child and young man, but after age 25 gained in weight and enjoyed good health.

P. I.: In 1918 he had an attack of quite severe pain in the abdomen, in the region of the umbilicus. This lasted several days. In 1921 and 1922, and again in 1924, he had similar attacks of abdominal pain. These attacks of pain lasted from 2 or 3 days to a week each. The last three attacks were colicy in nature, and localized in the right lower abdomen. Has complained of an epigastric "gnawing" as long as 10 years ago. In the beginning this was relieved by food, but more recently, food gives no relief. Belching gas relieves the discomfort somewhat. Frequently, vomiting is the only thing which will give relief.

P. E.: Age 41, weight 200, corpulent individual of medium height, tonsils imbedded, teeth in good condition, pupillary and deep reflexes normal, heart and lungs normal. Abdomen: Heavy panniculus adiposus. Some tenderness and muscle spasm on deep pressure over McBurney's point. Otherwise negative.

An exploratory laparotomy was done May 26, 1925. The stomach and duodenum were normal; round ligament free and unattached to the abdominal wall; this was sutured and the wound closed; a McBurney incision was made and a long and kinked appendix removed; patient developed a postoperative pneumonia, following which he was given sick leave; upon his return from sick leave, a ventral hernia, which had developed in the laparotomy incision, was repaired; he was discharged February 1, 1926.

On May 1, 1927, this patient was readmitted to the hospital complaining of weakness, drowsiness, and aching of the bones and muscles. He was placed

on an obesity regime and made a satisfactory progress until May 31, 1927, when he complained of a constant pain in the right upper abdominal quadrant.

On June 27, 1927, while at home over the week-end, he suddenly developed marked weakness and passed tarry stools. At this time the possibility of portal cirrhosis was considered, as it was believed that the bleeding was apparently not from an ulcer.

He had a gastric hemorrhage enroute to Mare Island, to appear before a retiring board; he was retired August 21, 1928, with a diagnosis of gastroduodenitis; following retirement he was admitted three times for gastric hemorrhage, as follows: March 2, 1929, July 14, 1929, and March 9, 1934; he remained in the hospital, during the last admission until March 20, 1934, when he was discharged symptom free. Finally, on August 17, 1934, while at his home, he had a severe gastric hemorrhage, and the following morning was admitted to the hospital; he continued to bleed, and in spite of all remedial measures, expired at 0410 on August 19, 1934.

Autopsy report.—The body is that of an adult male, well developed and nourished, somewhat obese. There is an old healed right upper right rectus operative scar and a healed McBurney incision.

Head: Negative.

Chest: The lungs are not adherent; no fluid in the pleural cavity.

Heart: Considerable fat surrounding the heart; aorta is smooth, not enlarged; coronaries are patent; heart muscle shows some fatty infiltration; valves are negative.

Abdomen: Omentum densely adherent to the peritoneum at the operative scar, and densely fat; there is an old posterior gastroenterostomy which admits two fingers readily.

Stomach: The stomach is large, not adherent; pylorus readily admits two fingers; no scars nor areas of infiltration; on opening the stomach it contained about 500 cc of old clotted blood; the mucosa is smooth and there is no evidence of any ulcerated area in the stomach or duodenum. There are, however, many scattered areas of injection which give the appearance of small punctate hemorrhages. The jejunal mucosa at the site of the gastroenterostomy is negative. There are no enlarged veins about the esophagus, and the mucosa of the esophagus is negative.

Liver: The liver is pale; it extends about 1 inch below the costal margin and upwards to the fourth rib; weight of the liver is 2,650 g; it shows the typical picture of atrophic (Laennec's) cirrhosis; the gall bladder is small but densely adherent to surrounding structures; otherwise negative.

Kidneys: Negative.

Spleen: Slightly enlarged.

Diagnosis: Cirrhosis of liver (atrophic—Laennec's) with multiple punctate hemorrhages from gastric mucosa.

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A SYSTEM OF ROUTINE DENTAL EXAMINATIONS AND TREATMENTS AS USED ON THE U. S. S. "ALTAIR"

By E. W. WILLETT, Lieutenant Commander, and E. H. DELANEY, Lieutenant, Dental Corps, United States Navy

In an effort to develop a definite method for "Routine dental examinations" and "Follow-up dental treatments" we have devised and inaugurated a system of procedure, as described below, for the personnel of the U. S. S. *Altair* and vessels of Destroyer Squadron 2, Battle Force.

The plan was first instituted on the U. S. S. *Altair* during the cruise to Hawaii in the spring of 1932. Every man on the ship was given a dental examination and the results were recorded on a 3- by 5-inch card, in accordance with the following notations:

Name, Doe, John R., Rate CM2/c.
Ship, *Altair;* Division, 6; Date, March 13, 1933.

Carious teeth	2, 17.
Useless teeth	32 impacted.
Missing teeth	1, 3, 18.
Restorations	2 o am, 14 mo am, 30 e am.
Prophylaxis needed	No.
Prosthesis needed	No.
Class of treatment needed	Routine.

Then at the bottom of the card the class of required treatment was indicated as *urgent*, *routine*, *prophylaxis only*, *prosthesis* or *no treatment needed.* Upon the completion of these examinations the cards were filed as follows:

(*a*) The cards of men needing work were filed alphabetically by divisions in two groups, *urgent* and *routine.*

(*b*) The cards of men needing no work were filed alphabetically after the dental abstract sheets in their health records had been corrected to date.

The results of our examinations were thus rendered readily available under the following categories:

(1) The number of men requiring (*a*) urgent (*b*) routine (*c*) prophylaxis only or (*d*) prosthetic dental treatment.

(2) The number of men requiring no dental treatment.

Preparations were then made to begin actual treatments. Division officers were furnished a list of the names of men needing work and were requested to send patients, selected by them from the list, at stated times, thus rendering it possible for the division officer or petty officers to detail the men whose services could most readily be spared at the stated time. This practice met with hearty approval of all concerned and all patients were available when the dental officers were ready for them. When the tender operated

independently, the entire day was devoted to the personnel of this vessel but when destroyers were alongside the entire morning was given to their personnel.

Since urgent cases were given priority as far as possible, results were soon noticeable, the most evident being the decrease of emergency cases among the personnel of the *Altair*. It so happened that the examinations of the tender personnel were completed en route to Hawaii so that nearly the entire time was devoted to actual treatments while she was at Lahaina Roads and on her return cruise. Upon arrival at San Diego, destroyers came alongside and the tender personnel was assigned the afternoon periods only. Through the enthusiastic cooperation of the responsible officers we were able to extend the same system to all of the destroyers of Squadron 2. At the completion of these examinations the commanding officer of each ship was furnished a report of the examinations, giving him a list of the names of the men under the headings "Needing urgent treatment" or "Needing routine treatment."

When a division of destroyers came alongside, the pharmacist's mates of the various destroyers reported on board and were informed of the number of new cases desired from their ship each day and of the time for them to report. The selection of the men rested entirely with the ship concerned but it was stressed that the urgent cases should report first insofar as practicable. Most hearty cooperation has been accorded us by all the officers of the various destroyers, the division commanders, and the division medical officers.

All new men who report to the various destroyers during the periods between overhauls are examined, cards prepared, and reports forwarded to commanding officers during each overhaul period. By this method the cards of the various ships are kept up to date. All pharmacist's mates inform the dental division of transfers of men from their ships so that their cards may be disposed of accordingly.

At no time have we allowed the application of this system to interfere with the treatment of any emergency cases that may arise. In other words, emergencies are not forced to wait for appointments but may report to the dental officer at any time.

The personnel of the U. S. S. *Altair* was reexamined during Fleet Problem XIV and it is with great pleasure and satisfaction that no *urgent* cases were found and, although the *routine* cases numbered to approximately 35 percent of the crew, the dental defects were of a simple nature and could be treated and completed promptly.

Due to the operating schedules of the destroyers and the large amount of work uncovered by the examinations, we have not been able to complete all the work required on all the ships. Most of the urgent cases, however, have been completed and a large percentage

of the routine cases are also finished. It is very unusual for an emergency case to report for treatment now, while prior to the inauguration of the present system as many as from 5 to 20 emergency cases could be expected each morning from the destroyers.

In our opinion the system has the following advantages:

1. It furnishes a readily available and definite record of the actual conditions existing in each individual case.
2. It provides a means for determing the most urgent cases.
3. It allows ships' officers to select the men they can best spare to send for treatment.
4. It apportions the work more evenly and allows the dental officer to arrange for patients at regular intervals throughout the day, thereby causing the least possible loss of time to everyone concerned.
5. It affords each and every man an opportunity to obtain dental treatment and advice with the least possible interference with his regular duties.
6. It permits the dental division to concentrate to a large extent on the personnel of the destroyers undergoing overhaul alongside, thereby reducing boating for dental cases to a minimum.

TUMORS AND ASSOCIATED PROBLEMS, PART 11[1]

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TREATMENT

All patients are required to report periodically for examination and any persistant disease or early recurrence is promptly treated.

(*a*) Skin cancer, unless extremely advanced or altered in its tumor bed by rèpeated recurrences, is by choice a radium problem. In all, except the most superficial lesions, heavily filtered radium at distances of 1 to 3 centimeters gives the best results. The wider zone of tissue radiated prevents recurrences at the border. The penetration at depth is greater and the scar is much better because of the lesser amount of fibrosis. It is more adaptable to localized application than X-rays and superior to thermic or electro-cauterization because of wider zone of tumor bed affected (6).

(*b*) In carcinoma involving mucous membranes of mouth, nose and throat the growths are predominantly epidermoid carcinomata—metastasizing tumors involving chiefly cervical lymph nodes. An intensity of 7 to 10 skin erythema doses of radiation at all points within the tumor-bearing area is necessary to produce complete regression of adult epidermoid carcinoma (6).

Only three skin erythema doses is the maximum dosage that can be delivered by external radiation within the oral cavity, during a

[1] Continued from July 1935 number of the U. S. Naval Medical Bulletin.

2- or 3-week interval, consistent with tolerance of patient. The balance of 4 to 7 skin erythema doses must be supplied by direct implantation of radon (or radium) into the tumor (6).

The implantation of gold radon seeds is used to control the primary growth. External radiation, radium or X-rays or both, is used over both the primary growth and cervical lymph nodes in all cases when the lymph nodes are not palpably enlarged (6).

The palpably involved node of adult type epidermoid carcinoma, when unilateral (that is when on one side of neck only) and with intact capsule is treated by a complete unilateral neck dissection plus radon implantation within the wound in such fashion as to irradiate heavily any suspicious areas.

Bilateral involvement of cervical lymph nodes or nodes with perforation of the capsule by the growth are treated by surgical exposure under local anesthesia and implanatation of gold radon seeds.

Cervical lymph nodes involved by metastatic growths of undifferentiated type are treated by radiation entirely, even though technically operable (6).

(*c*) Extrinsic laryngeal cancer is (practically) inoperable. The only resource is external and interstitial radiation, except for the few highly radiosensitive growths where maximal X-ray dosage alone may be sufficient to produce complete regression (6). When radiation is given to the neck, the patient should be in a hospital—immediate tracheotomy may become necessary due to radiation edema of larynx.

A certain percentage of the intrinsic laryngeal cancers are operable. Total extirpation of larynx is possible. Metastasis rarely occurs until late in the course of the disease. Following heavy external radiation, in most cases a direct radon implantation is done through exposure by laryngotomy (6).

(*d*) Few growths in the mucous-membrane-lined tract of the bronchial tree, esophagus and upper gastrointestinal tract, lend themselves to gold radon seed implantation anatomically. However, growth restraint may be induced in these areas by external radiation and result in palliative relief (6).

(*e*) *Inoperable carcinoma of rectum.*—External radiation is given first. It consists of both radium and deep X-ray treatments. The X-ray treatments are given daily or on alternate days and are followed by daily applications of 8,000-milligram hours of radium until the full dose has been delivered. Heavy preliminary external radiation is used in patients who present possibilities of a clinical cure or in whom a high degree of palliation is expected. By heavy radiation is meant a pelvic cycle of X-rays consisting of seven portals and in addition radium applications of from 60,000- to 75,000-milligram

hours at a distance of 15 centimeters from skin over each of two or more portals. (The X-rays are administered through six portals which include the greater part of the pelvis and a seventh—the perineal field, when tumors are situated low in rectum.)

The usual factors for the X-ray treatments are: Sixty-minute exposures at 185 kilovolts, 50 centimeter skin-target distance, 4 milliamperes of current filtered by ½ millimeter of copper and 1 millimeter of aluminum. The time of exposure may be increased to 80 minutes. If a water-cooled machine which operates at 30 milliamperes is used, the time of exposure is decreased. The short time required to administer thus an erythema dose of X-rays is greatly appreciated by the nervous and debilitated patients who find it trying to lie quiet in one position for an hour or more daily.

Of the total X-ray and radium radiation administered over the 7 portals only 2 to 2½ erythema doses reach the tumor (12). This is not sufficient to produce complete regression of the neoplasm. The effect of this radiation upon the tumor mass determines its degree of radiosensitivity and the advisability of providing more intense radiation by implantation of gold radon seeds into the tumor. Tumors that are not deeply infected respond well to this type of radiation. The gold radon seeds which are left in situ are distributed in high-lying tumors under direct vision through a proctoscope and in low-lying cancers usually by sense of touch alone. The upper limits of constricting tumors are reached by passing the proctoscope downward through the colostomy opening. A clear field of vision for the implantation is maintained by suction apparatus. Adequate dosage and the proper placing of seeds are essential (13).

In those patients in whom only a moderate degree of palliation can be anticipated, a pelvic cycle of X-rays with a 2- or 3-day interval between treatments is given. After about one week radium treatments are begun, the total dosage for each portal seldom exceeding 50,000 milligram-hours (12). Colostomy is done and radiation given but no surgery otherwise in the inoperable group. If there is infection it is difficult to eradicate carcinoma by radiation. Bladder symptomatology seldom occurs.

For operable cases: Perineal resection is done and radiation therapy given.

(*f.*) In carcinoma of the bladder external radiation is inadequate. The maximum amount of radiation that can reach a bladder tumor from a single pelvic cycle of four high voltage X-ray treatments is 1.2 skin erythema doses (15). It has been calculated that 10 skin erythema doses are necessary to control radioresistant tumors of the bladder (14). The dosage actually used is much larger, 20 to 25 skin erythema doses. Patients are examined cystoscopically at fre-

quent intervals to detect recurrence while small enough to be treated through the cystoscope. Such recurrence usually means the disease is beyond control. It is for this reason that such large doses are used, the aim being to cure by a single treatment when dealing with such an inaccessible organ as the bladder (15).

The gold radon seeds are implanted through the cystoscope when the base of a papillary carcinoma of the bladder is not larger than 1.5 centimeters in diameter and is entirely visible through the systoscope (15).

Larger papillary cancers and infiltrating tumors are treated under full vision with the bladder opened (15). Spinal anesthesia is used. The bladder is not mobilized; the abdominal wound is thoroughly screened with gauze prior to opening of bladder; great care is used not to spill bladder contents over the wound. Good exposure is necessary. Cameron light is used for retraction and to illuminate the bladder cavity. Papillary portions of tumor are removed by some form of cautery (14). Throughout the tumor and to a distance of at least one centimeter beyond any visible or palpable evidence of involvement, 1½ to 2 millicurie radon gold seeds are implanted. These seeds must be placed with greatest precision, one centimeter apart and one centimeter deep; each seed marks the vertex of an equilateral triangle of one centimeter sides. Over 50 such seeds, totalling 100 millicuries have been used without harm (15) (16). The minimum dose permissible is 2 millicuries to 1½ centimeters radius of tumor in all 3 dimensions (14). Any tumor tissue at a distance of 1½ centimeters or preferably 1 centimeter from a 2 millicurie radon seed is not likely to be completely destroyed, regardless of the direction. No special attention is paid to ureteral orifice when involved in tumor (16).

If the radon seeds be near the rectum, there will be considerable rectal irritation and in all cases bladder irritability may occur following large dosage of radon. The radium burn, however, is the lesser of the two evils and the malignancy should cause more concern (14). A suprapubic drainage tube, 18° to 22° F. is left in place for a week or longer if the bladder is dirty or the radon dose is large. The bladder is not sutured to the abdominal wall (14).

(*g*) *Carcinoma of prostate.*—Urinary retention must be promptly relieved. The obstruction is punched out with Young's instrument (or electric loop). The tissue removed serves as biopsy specimen and if punch operation is not done a specimen is obtained by aspiration or with the Hoffman punch through the perineum (16). To control carcinoma of prostate 10 to 15 skin erythema doses delivered to tumor are necessary (14). External radiation (X-ray and radium) cannot deliver over 1½ skin erythema doses (14). Gold radon seeds must be implanted or radon bearing needle or needles used.

Because some prostatic carcinomata are highly malignant and radiosensitive, before anything else is done a thorough cycle of deep X-radiation is given, using five portals of entry (14). This also promotes absorption of edema and congestion (16).

Under local infiltration anesthesia a needle carrying a 2-millicurie gold radon seed is passed through perineum and guided by left forefinger in the rectum, the radon seeds are deposited 1 centimeter apart as accurately as possible (16). Sometimes it may be desirable because of necessity of accurate placement of radon seeds, to do a cystotomy. Any obstructive portions of prostate are removed with cautery and entire tumor implanted with 2-millicurie gold radon seeds 1 centimeter apart in all planes (14). (Accuracy in implantation may mechanically be improved with Ferguson device.) When radon-bearing needles are used, the strength is about 50 millicuries. The needle is left in place until a dose of 300-millicurie hours has been delivered, then it is moved to another location or withdrawn completely. This treatment is repeated at weekly intervals until the entire growth has been radiated. Treatment may be hastened by using two needles instead of one (16). Temporary irritability of bladder or rectum sometimes occurs. Occasionally the gland swells so much as to require catheterization of patient until this subsides. In six to eight months, if growth activity can be demonstrated, further treatment is given (16).

(*h*) *Carcinoma of penis.*—This is always proved histologically and usually is squamous carcinoma. Biopsy is carefully performed with sharp razor (16). If the lesion is 2 centimeters or less in diameter 1,200 millicurie-hours of radon per square centimeter at 1 centimeter distance is applied in the form of a plaque (14). If the primary lesion is larger than 2 centimeters in diameter and no metastases are found, the radiation with the plaque is followed in 3 or 4 weeks by a conservative operation (14). The amputation is done 2 centimeters proximal to visible or palpable disease (14). The success depends on knowledge that metastasis is by embolism (14). If tumors are extensive, amputation is done without preoperative radiation (14).

Adenopathy with no metastases will subside (16). If inguinal nodes become larger or harder or aspiration biopsy reveals cancer, the case is treated palliatively with additional external radiation (16). It may be advisable to expose the lymph nodes and implant radon gold seeds. Block dissection is not advised (16). Radiation is powerless against metastatic squamous carcinoma in infected lymph nodes. No treatment is given the groins until infection subsides (16).

(*i*) *Carcinoid tumors of testis.*—A biopsy leads to a rapidly growing fungating tumor and should never be attempted in these cases

(16). Roentgenograms of lungs are taken routinely (16) and Aschheim-Zondek test (17) is done in all cases. In embryonal tumors metastases are early to retroperitoneal and other lymph nodes and to lungs. However, embryonal carcinoma is very radiosensitive (16).

In cases of testicular tumor in which no metastases can be found, maximal exposures of external radiation are given to the testis and to the abdomen of the same side (16). The normal testis is protected carefully with sheets of lead suitably molded. In spite of this, transitory aspermatogenesis occurs in almost all cases (16). Repeated cycles of radiation to insure complete devitalization of the tumor destroy permanently the spermatogenetic function of the sound testis (16). Instead of attempting such complete devitalization of the tumor, when the tumor has regressed to size of the normal testis or smaller, which occurs in about 4 weeks after radiation, orchidectomy is performed (16). Under local anesthesia the spermatic cord is doubly ligated and cut as it leaves the inguinal canal and the testis is gently separated from the tunica vaginalis. Great care is taken that the tunica albuginea is not punctured. Care is taken not to squeeze the testis and so prevent dissemination of tumor cells (14). Local recurrences have promptly followed when the coats of the tumor have been cut accidentally and even a few drops of the contents spilled in the wound (16). After the operation 2 or 3 radiation treatments are given to the scrotum, groin, and pelvic and lumbar lymphatics at suitable intervals. Metastases are treated with external radiation alone. No operation of any kind is attempted. Heavy doses of radiation frequently control the disease (16).

Results.—At Memorial Hospital, New York, 41 of 113 patients are living and free from disease (14).

(*j*) *Carcinoma of cervix.*—In carcinoma of the cervix X-ray cycle is given first, except in very early cases (18). The portals used are left anterior, left posterior, right anterior, and right posterior, and each field is 12 by 15 centimeters, receiving 700 to 800 roentgens at a single sitting. The treatments are given daily or 1 or 2 days apart and are spaced farther apart if there is much gastrointestinal disturbance or general malaise; and may be given in divided doses if the condition of the patient demands it. Under this treatment the bulk of the lesion is diminished as well as the inflammatory process (18).

Radium treatment is begun within 10 days after the X-ray treatments have been completed. Vaginal applicators, which vary in shape and size and each of which contains 400 to 1,000 millicuries of radon, are used. An applicator is applied against the cervix until a dose of 1,000 to 2,000 millicurie-hours has been given. When

feasible, this is followed by applications against the right lateral and left lateral vaginal fornices for 1,000 millicurie-hours to each, making a total dosage, by this means, 3,000 to 4,000 millicurie-hours to the cervix and the surrounding lymphatics. A day or two later a dose of 3,000 millicurie-hours is given at one sitting by means of the tandem (18).

All above treatments deliver together about 10 S. E. D. to the cervix (18).

This finishes the treatment in favorable cases and the entire cervical lesion disappears within 8 weeks. In some cases there will still be evidence of active disease in the cervix or vaginal fornix and within 4 to 6 weeks after the first treatment is given, 1.5 to 2 millicurie-gold radon seeds are implanted in remaining lesion or additional treatment is given by means of one of the vaginal applicators. The greater part of the original lesion having disappeared, the gold radon seeds can be placed more accurately and with less danger of overdosage, of damage to normal tissues, of formation of fistula, of spread of metastases, etc. For these reasons they are implanted at this time rather than at the time of the primary treatment (18).

In advanced cases, the high voltage X-ray cycle is repeated 6 or 8 weeks after the first cycle (18).

The time of treatment varies from 2 to 10 weeks. No further routine treatment is planned, but is determined by subsequent development of active disease (18).

Hysterectomy is done only for some persistent complication such as pyometra, or pus tubes, or rarely in an otherwise favorable case, in which the lesion has not responded to radiation therapy (18).

In dealing with a large cauliflower lesion of the cervix, the high voltage X-rays are used first, followed by implantation of gold radon seeds and then the lesion is cauterized. There is then less danger of scattering viable cancer cells in the surgical manipulations (18).

In very early cases, with the lesion apparently limited to vaginal fornix, radium is used first, followed by X-rays (18).

(*k*) *Carcinoma of Uterus.*—In carcinoma of uterus a pelvic cycle of deep X-ray therapy is given through four portals, one exposure every other day. About 10 days later a radium tandem is inserted into uterus. Then about 2 months later uterus is removed surgically.

(*l*) *Carcinoma of breast.*—When tumor in the breast is small or of moderate size and there is no invasion of axillary nodes or when but one or two nodes are palpable, a radical amputation is indicated (19). Roentgenograms of chest and osseous system are studied first for evidence of metastases.

Before surgical intervention, a cycle of deep X-rays is given over the breast and drainage area; this is especially necessary in young patients because of early and rapid metastases. In radiosensitive tumors regression in size will follow within 3 to 4 weeks. No benign tumor of mammary gland responds in this way to radiation (19). Later sufficient gold radon seeds to bring total dosage of radiation to 13 skin erythema doses at all points within tumor are implanted (20). (The method is unsuitable for tumors larger than 6 centimeters in diameter) (20). Four to five weeks, preferably six weeks after implantation of radon seeds a radical amputation of the breast is done (19). Radiation lowers vitality of tissues. Almost complete devitalization of tumor is found on histological examination of amputated breast in many instances (19). Time will show whether end results will justify this method.

The following cases are inoperable: (1) Breast largely occupied by tumor tissue, (2) axillary nodes extensively invaded, (3) presence of one or more cutaneous nodules overlying the tumor in the breast, (4) involved supraclavicular node, (5) presence of lung or peripheral metastases. The management of inoperable cases is similar to that of operable ones. If an ulcerating mass is present a palliative mastectomy is done 2 months after completion of adequate radiation. This operation is never attempted if tumor tissue extends continuously from breast up to and into the axilla (19).

In treatment of cancer patients certain factors should be kept in mind. They were embodied in an address delivered by Dr. Ewing at the New York Academy of Medicine in October 1932.

As long as cancer tissue is well nourished and there is no obstruction, the host feels no ill effects. It is surprising what an extesive amount of cancer tissue the well nourished individual can carry with apparently little or no discomfort. The anemia in cancer may be of any type; it is of pernicious type in gastrointestinal tract cancers. This may be due to disturbed secretion of HCl and consequent malnutrition. In other case the anemia may be due to hemorrhage. In buccal and esophageal cancers anemia is due to failure to take nourishment. The judging of anemia by laboratory reports is unsound. The anemia may be marantic, the blood may be concentrated due to dehydration and blood count and hemoglobin may be 100 percent. The blood volume as well as quality is important. The transfusion of blood is of value in many cases. A fatal factor in cancer is infection of tumor tissue and of tissues of the host. All types of bacteria have been found and the failure to find specific causative organisms have led to a neglect of the study of types of infecting organisms in cancerous tissue. Cases of cancer have been observed in which every manifestation of the streptococcus followed in sequence, thus exhausting the patient.

Infection and intoxication, which occur so easily in cancer, are readily explained; the cancer contains growth stimulating substances, which, if they can stimulate cellular growth will much easier stimulate bacterial growth leading to fever, toxin production and exhaustion of the patient.

If one were an optimist one could say that to prevent infection in cancer would lead to prolongation of life.

Another fatal factor cancer is degeneration of tissue resulting in aseptic fever, intoxication, hemorrhage and infarction. These are the three reason—anemia, infection and degeneration of tissues—why cancer kills. Another powerful factor in the mortality of cancer is the psychic state—the pain, loss of work, hemorrhage, deformity, recurrences—all these powerfully affect the psychic state. The more highly developed the individual, the more destructive is this factor.

It is sound medicine never to tell the patient. Let him believe it is a disease such as other diseases but do not mention the word cancer. The word conveys to the mind of the patient the hopeless, most malignant type of the disease. Thus you have not been honest with the patient and conveyed the wrong impression. Remember what the word connotes to the patient. "It might become cancer" but not "cancer" in the presence of the patient.

We must displace the methods of severe surgery and severe radiation. Twenty-five years hence we will be horrified by what we do for these patients now. The severities to which patients are subjected are due to the frantic efforts of the medical profession to cure the incurable.

If the cancer is early and there is reasonable hope of cure, go after it vigorously, otherwise restrain yourself. There is nothing toxic in cancerous tissue.

Applied Radiation in Treatment

Radiation is used so extensively in the treatment of malignancies that the proper understanding of the entire problem requires a consideration of this subject.

(*a*) Physics of radiation (26) (27) (28).—The radio elements are those which possess, in addition to properties of a normal element, the power of emitting alpha- or beta-particles. In so doing the element breaks up to form a new element, or as it is expressed technically, it disintegrates. The rate at which disintegration occurs proceeds always according to one fixed plan. The half value period of time represents the interval of time which must elapse for half of the initial amount of element to disintegrate.

Radium and radon are respectively the sixth and seventh of the disintegration series that begins with the rare element uranium and ends with the common element lead (26).

For radium the half value period is 1730 years, for radon it is 3.85 days.

Three types of radiation are emitted by radioactive bodies called alpha, beta, and gamma rays. The alpha radiation consists of positively charged particles traveling with velocities of about 10,000 miles a second. These particles become helium atoms as soon as they take up two negative electrons. The beta radiation consists of negative electrons traveling with high velocities, approaching in some cases that of light. Unlike the emission of alpha particles, a radioactive body emits beta particles over a considerable range of velocity.

Gamma radiation is of electromagnetic nature like the X-rays and ordinary light. It is probable that the gamma ray is due to rearrangement of the constitutents of the nucleus after expulsion of the beta particle. Unlike the alpha and beta particles, the gamma rays are undeflected by a magnetic or electric field. The gamma rays consist of groups of electromagnetic radiations of widely different frequencies.

One atom of radium, when it breaks up, expels an alpha particle and becomes one atom of radon. Since helium has an atomic weight of 4, the atomic weight of radon is equal to the atomic weight of radium 226 minus 4 or 222. Radium is a metal closely related to barium, whereas radon is an inert gas (in chemical sense of the word) like neon, krypton, etc. The quantity of radon liberated from 1 milligram of radium is called a millicurie.

One atom of radon breaks up, expels an alpha particle and becomes radium A, which is again a solid. Similarly, radium A changes into radium B. In the change of radium B to radium C there is no expulsion of alpha particles, but instead beta and gamma radiations are given off. In this case while there is a change of one element into another, there is no change in atomic weight. The valency, however, does change—expulsion of a beta particle increases the valency by one. The process continues until from radium F or polonium a substance is formed which is indistinguishable from lead (28).

The amount of radon in equilibrium with one gram of radium is 0.006 gram and occupies a volume of 0.06 cubic millimeter at normal pressure and temperature. The condition of equilibrium in a sealed tube is reached in about 30 days. For this reason the Bureau of Standards issues its certificate only after tubes have been kept under observation for a few weeks. The measurements are based on gamma-radiation from radium C (28).

Radiations from radioactive bodies affect photographic plates; produce luminescence; transform their energy into heat; produce chemical changes (decompose and synthesize); produce ionization, that is, break up some atoms of a substance into positively and negatively charged particles; and produce marked physiologic effects. Radiations are absorbed by matter:

The alpha-radiations are absorbed by small thickness of solids or liquids, less than one-tenth millimeter of aluminum. Their maximum range in atmospheric air is about 7 centimeters.

The swiftest particles of beta-radiation will penetrate two-tenths millimeter of lead or nearly 1 centimeter of aluminum; while gamma-radiations can be detected through 25 centimeters of lead. Since over 90 percent of energy emitted by radium is carried by alpha particles, radon as used in tubes or hollow gold seeds can supply at best only 10 percent of the energy of radium (28).

The greater the density or the higher the atomic weight of a substance through which radiations pass, the larger is the absorption of the radiations by that substance. When radiations meet a substance they are scattered by it. When the gamma-rays impinge on a substance, that substance emits a secondary radiation of both the gamma-ray type and of the beta-ray type (28).

Since the alpha and beta radiations are not very penetrating, they are absorbed by the superficial layers of the tissues and their absorption by those layers of tissues causes burns and destruction of the tissues. By placing a plate of metal between the source of radiation and the tissues the alpha and beta radiations and even those gamma rays of lesser frequencies can be absorbed by such a plate of metal—filtered out—and only the gamma rays of higher frequency will reach the tissues and penetrate to some depth. These do not burn since vary little of the radiation is absorbed by the tissues.

It follows that the subject of filtration is of much practical importance. A filter which removes all the beta rays (and of course also all the alpha) passes only 2.8 percent of the total radiation. Such a filter is

2.0 mm of brass, or
0.5 mm of pure gold or platinum, or
0.9 mm of lead, or
1.1 mm of silver (29).

The gamma radiation passing such a filter is not homogeneous. To screen out the softer gamma rays a thickness of 1.4 mm of gold or platinum is satisfactory. Nothing is gained by additional filtration beyond 1.5 mm of lead or 2.0 mm of brass, but additional gold filters will screen further rays (29).

The criterion of homogeneity for the selection of a filter yields a different result depending on the absorbing material used. From the point of view of tissue absorption, 2 mm of brass is certainly a suitable filter. If it is desired to use a more homogeneous radiation 0.9 mm of lead will be better and 1.4 mm of gold or platinum will be still better. Nothing is gained, from any point of view, by increasing the filtration beyond 1.4 mm of platinum (29).

(*b*) *Absorption of radium radiations by tissues.*—The fraction of the incident radiation which is absorbed or scattered per unit thickness of filter is known as the coefficient of absorption. For tissues, a close approximation is 0.075 for value of absorption of gamma radiation after filtration through 1.92 mm of brass. With this filter a thickness of 10 centimeters of tissues absorbs about one-half of the radiation (30).

Filtration is only one of the factors which determine the dose of radiation delivered to a deep-seated tumor. The effect of distance is

no less important. It is a consequence of the law of conservation of energy that the intensity of radiation emitted by a point source varies inversely as the square of the distance. In order that the greatest percentage of the radiation falling on the skin may reach a deep-seated tumor, the difference between the distance from the source to the skin and the distance from the source to the tumor must be small in comparison to those distances (31). Since no more radiation can be given than the skin can bear, it is very desirable to get the greatest percentage of the radiation falling on the skin into the tumor. When the distance of the radium from skin is 19 times the tumor depth the amount of radiation reaching the tumor is 90 percent of the amount falling on the skin. For a tumor 10 centimeters deep this would place the source at a distance of 190 centimeters from skin, which is impracticable (31). If we are satisfied with a ratio of 80 the source may be placed at 8.5 times the tumor depth. On account of the scarcity of radium, applicators are never placed at the most advantageous distance from the skin (31). The farther away the radium is the longer it takes to deliver a definite quantity of radiation to the skin, but the greater is the percentage of what reaches the skin that is delivered to the tumor at some depth below the skin.

(*c*) *Collection and use of emanation.*—Neoplasms below the skin can seldom be destroyed by radiations from a distance. If they are on the surface, radium or radon at a distance of a centimeter or more may be used in the form of some applicator. In case of deeper neoplasms hollow needles containing radium or radon may be inserted into the tumor and withdrawn after the proper dose has been given. Since radon disintegrates in a few days, it is possible to insert and leave in situ within the tumor small hollow tubes of gold called "seeds" containing from 1 to 2 millicuries of radon. Each "gold seed" is 3 millimeters long and 0.3 millimeters in diameter and is introduced by means of a hollow needle and stylet, which serves to push the "gold seed" out of the needle and so implant it in the tumor. Radon for these purposes is collected and used as explained below.

The radium, which is kept in a vault, is dissolved in acidulated water and radon pumped off every morning (32). The radon is purified by chemical means and led into capillary tubes about 40 centimeters long, containing about 200 millicuries of radon. A machine has been developed to divide the long tube into 100 small tubes. No radon is lost in the process, because the pressure of the gas is less than atmospheric pressure (32). Longer tubes are used for various applicators. Tubes may be arranged in a small box—oval, circular, square, or rectangular—to provide radiations for local ap-

plications. Molds can be made of dental modeling compound and radon-containing tubes imbedded in such molds. These molds are applied to lesions so situated that other applicators are unsuitable—for example in the mouth, on lip, etc. Plates of lead to protect healthy tissues are embedded in such dental-modelling-compound molds. The doses which have been found by experience to be effective range from 40 to 60 millicurie-hours per square centimeter (filer ½ millimeter of silver plus 2 or 3 millimeters paraffin and wax) (32). If the surface is curved or is large, crossfiring occurs and the smaller dose is given.

(*d*) *Dosage.*—The preliminary dosage of radiation was empiric. Martin and Quimby of Memorial Hospital, New York City (33), undertook the study of quantity of radiation necessary to produce complete regression of various types of epidermoid carcinoma. A series of cases in which a complete regression had been produced was selected and the intensity of radiation at all points within the tumor-bearing area was determined. The data thus obtained were applied to treatment of new cases of varied histological type, the dosage being calculated beforehand in terms of skin erythema doses. The method of delivery of this dosage was directed by a physicist on the most economic bases—X-rays and/or radium externally or by interstitial implantation. The results corresponded with the findings in the cases studied in retrospect (6).

For interstitial implantation of gold radon seeds the size and shape of tumor determines the dose. A table has been prepared showing the number of millicuries in gold radon seeds required to deliver specified skin erythema doses to masses of various diameters (33). For example, a tumor measured by a pair of dividers is found to be 4 centimeters in diameter. It is desired to deliver by means of gold radon seeds a dose equivalent to 10 skin erythema doses. Consulting the table it is found that 29 millicuries of radon must be implanted within the tumor in a definite manner to supply the radiation required (33).

(*e*) ***Measurement of radiation.***—The measurement of the strength of radon tubes is made by a physicist or under his immediate supervision and is based on passage of electricity through an ionization chamber, the degree of ionization depending on the quantity of radiation. The same principle is used in determining X-ray strength, that is, its power to ionize air. The air is a nonconductor of electricity but when X-rays or radiations from radium pass through air the air is ionized. The degree of this ionization depends on strength of radiation and determines the amount of electric current which passes through the ionized air. The current can be measured and so indirectly indicates the strength of radiation which is expressed in terms of a unit known as the "roentgen."

The " roentgen ", written with a small " r " to differentiate it from an older unit of same name, is the international unit of X-radiation. The official definition is:

> The international unit of X-radiation is the quantity of X-radiation which, when the secondary electrons are fully utilized and the wall effect of the chamber is avoided, produces in one cubic centimeter of atmospheric air at 0° centigrade and 76 centimeters mercury pressure, such a degree of conductivity that one electrostatic unit of charge is measured at saturation current (34).

It is possible to measure gamma-radiation in roentgens also and thus X-radiation and gamma-radiation become comparable quanti tatively.

(With proper corrections) we may say that one gram of radium filtered by 2 millimeters of brass emits gamma rays at such a rate that the intensity at a distance of 22.5 centimeters is 0.071 roentgen per minute. For a distance of 1 centimeter the value is 36 roentgens per minute. This is a preliminary estimate subject to later modification (34). It may also be stated at this point that an ionization current of one electrostatic unit corresponds to a gamma ray intensity of 1.85 roentgens per minute (34).

(*f*) *Quantitative effects of radiation on skin.*—Having a means of quantitatively measuring X-radiation and gamma radiation in terms of roentgens, the next question is: How many roentgens of either radiation will produce an erythema on the skin of a human individual? Difficulties are immediately encountered because different individuals will require varying doses of radiation to produce a standard erythema and the determination of a standard brings into the problem the personal equation of the observer. It is not possible even for the same observer at two different times to know whether the degree of erythema is the same in the two instances. It is quite simple to say that there is no erythema or that there is a faint but definite erythema. For this reason, the threshold erythema is used by preference at Memorial Hospital, New York City. The amount of radiation which, if delivered at one sitting, in 80 percent of the cases treated, will produce a faint bronzing or reddening of the skin in from 10 to 20 days and in the other 20 percent will produce no visible effect, is known as the erythema dose or skin erythema dose (S. E. D.) (33).

To produce 1 erythema dose with the radium element pack containing 4 grams of radium, a fair estimate is, at 6 centimeters distance from skin, 10,500 milligram-hours; at 10 centimeters distance from skin, 22,000 milligram-hours. Using these figures, the erythema dose for gamma rays with the pack in terms of roentgens is 500 (roentgens). This includes back scattering and taking the intensity at the center of the beam as a basis of calculation. If average

intensity is taken instead, the dose becomes 480 roentgens (34). For X-rays, with 200 kilovolts and 0.5 millimeter copper filter the erythema dose is 600 roentgens.

NOTE.—The number of roentgens of X-radiation required to produce an erythema of skin varies within wide limits depending on many factors, one of the most important being filtration. Each X-ray machine must be calibrated individually for each set of factors.

The erythema produced is comparable to that produced by 500 roentgens of gamma radiation. This difference means that while X-radiation may be equivalent quantitatively to gamma radiation in producing exactly the same degree of ionization of air, it is not equivalent quantitatively to gamma radiation in producing skin erythema (or any other biological effect). In both the above instances the erythema is probably more marked than the threshold erythema (of Memorial Hospital) for which the gamma-ray dose is known to be 425 roentgens. Because of back scattering of radiation, the larger the mass of tissue, for example the thigh, as compared with the arm, the fewer the number of roentgens required to produce an erythema in the same individual. For the 4 by 6 centimeter tray, for which 1,000 millicuries of radon is usually available at Memorial Hospital, a fairly full erythema dose appears with 3,000 millicurie-hours at 3 centimeters distance.

G. Combined use of X-radiation and gamma radiation.

When both X-radiation and gamma radiation are employed together in equal ratio, the skin tolerance is increased 20 percent or more (35).

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INFLUENCE OF THYROID IN THE HEALING OF WOUNDS

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During the year 1932 over 158,000 days were lost in the United States Navy from injuries, the majority of which were open wounds. This represents the loss of work of one man to the total extent of more than 432 years, and if the loss of time from injuries throughout the United States were considered, the results would be of much greater magnitude. However, such figures represent only the economic side of the picture, the untold suffering cannot be estimated.

The delayed healing of wounds is a problem with which the medical profession is confronted daily. It is not a new problem—it has been before the physician from the earliest history of man. From the ancient period of magic through the period of "laudable pus" little progress was made in its solution, until bacteria were discovered, this led to the rapid adoption of aseptic surgery and the sterilization of wounds, a procedure carried to a high degree of perfection during the World War (1). However, efforts to hasten the healing of sterile wounds has met with little success, and evidence accumulated in that field has been more or less negative in character.

It has been shown that wound healing is delayed by roentgen irradiation, starvation, hyperglycemia, and thyroid deficiency. Radiation retards fibroblastic growth, produces giant fibroblasts, and forms a persistant fibrin network (2). The healing rate is definitely slowed with partial starvation in young rats, while adult rats on the same type of diet are not appreciably affected (3). The lack of dextrose tolerance in persons who are not ordinarily classed as diabetics hampers the healing of wounds (4). With regard to thyroid deficiency, it has been known for some time that preparations from this gland aid in the healing of fractures in areas in which endemic goiter is present. Callus formation is much more rapid in these cases than in those which do not receive thyroid, pseudarthrosis is avoided and complications from the drug are not apparent. Three Italian investigators reported in 1931 that the thyroid gland had a marked influence on the cicatrization of superficial wounds. Thy-

roidectomy was performed on a series of guinea pigs of about the same age and weight; after a period of 5 days a rectangular incision was made over the scapular region, involving the skin and subcutaneous tissue. Thirty days after the latter operation the thyroidectomized animals were found to have wounds about 30 percent larger than those of the controls (5).

The internal secretion of the thyroid gland should theoretically have some tendency in promoting the healing of wounds, since the hormone regulates the anabolic and catabolic processes of cellular protoplasm, promotes protein oxidation, and influences growth.

Since the thyroid governs the metabolism, and since the metabolism about a wound is increased, it is probable that this gland plays some part in the physiology of wound healing. An instance of the physiological process that occurs in healing is the local congestion of the blood vessels, aiding in the nutrition of the newly formed cells and the elimination of detritus. It is a well known fact that in the nutrition of these newly formed cells (6), as in all factors of growth, protein metabolism plays an important part, and there is definite evidence that the complete oxidation of the proteins is dependent on the internal secretion of the thyroid. Urine of patients with hypothyroidism contains a high percentage of the purine bases together with lysine, an amino acid. Following thyroxine therapy the urine of these patients is restored to normal (7). Further proof of the influence of the thyroid on the process of healing was pointed out recently by Cohen, who reported a case of a hypothyroid patient who had large leg ulcers, which had failed to respond to any usual treatment after a period of 6 years, but healed in a few weeks when thyroid extract was administered by mouth (8).

With the foregoing facts in mind, it was thought of value to determine the influence of thyroid preparations on the healing of superficial wounds. The problem at hand was to determine the dosage, advantages, and disadvantages of this drug, with the possibility of its practical use in the treatment of wounds.

PROCEDURE

Six white mice and six white rats were used as the experimental animals. The white mice were approximately the same age and weight. They were placed in separate clean cages, given a balanced diet, and four of them received $\frac{1}{64}$ to $\frac{1}{8}$ grain of desiccated thyroid [1] daily, an approximate equivalent of 47 to 375 grains when compared with the human dosage. The drug was mixed with cheese and glucose, made into pill from, and fed by mouth. The standard used to determine the dosage was 3 grains of thyroid daily for a 60 kilogram

[1] Parke, Davis & Co. products were used throughout the entire experiment.

man. After a period of 3 weeks all of the mice, except one, were autopsied, and histological studies were made of the thyroid, heart, aorta, liver, and skin.

The six white rates were all of about the same age and weight, except one, an adult rat. The others were young. Their diet and cages were of the same character as those for the mice. The same standard was used in calculating the thyroid dosage, which was varied from an approximate equivalent of $\frac{1}{36}$ of a grain of thyroid daily to an equivalent of 10 grains daily during a series of three experiments. The animals were numbered from 1 to 6—no. 1 was the adult rat, and nos. 3 and 4 were used as controls. The same number for each rat and the same controls were used throughout each of the three series.

In the first series, rats 3, 4, 5, and 6 were used. The hair was shaved from the scapular region of each of the animals. The area was then painted with iodine and a wound of definite size was clipped from the skin and subcutaneous tissue. The surface of the wounds was determined by placing a piece of cellophane over each one and marking around the borders. The tracing thus obtained was transferred to a graph paper with squares of about 0.002 of a square inch. The area of the wound was then calculated by counting the number of squares included in the boundaries of the tracing. Since the wounds were small, this method of determining the area of the wounds was found to be as accurate as the use of a planometer. The surface of each wound was usually measured every 5 days until healing occurred. The wound was considered healed when all evidence of scab formation had disappeared. No. 5 received 1/36000 grain of desiccated thyroid daily for 23 days, and and no. 6 received 1/2500 grain of the same substance for an identical period, which was the duration of the first series.

The second series was conducted in the same manner as the first, except that rats nos. 2, 5, and 6 were given 1/238 grain of dessicated thyroid daily, and rat no. 1 was given twice that amount. In the third series the same amount of thyroid was continued for nos. 5 and 6, while no. 1 was given $\frac{1}{25}$ grain daily and no. 2 $\frac{1}{100}$ grain daily. In order to determine whether the frequency of the measurements hampered the healing rate, intermediate measuring of wounds was discontinued. All the animals were autopsied at the end of the third series.

RESULTS

All of the mice receiving thyroid developed a voracious appetite and lost weight rapidly. The heart rate increased markedly, and one of the animals died a few days before the experiment was ended. Histological studies were not made on this mouse because of an advanced state of autolysis at the time the body was found.

The thyroid gland, in the experimental animals studied, showed a decrease in the colloid, an increase in the epithelial elements, small acini, and other characteristic changes of hyperthyroidism; the aorta was dilated; polymorphonuclear leucocytes and plasma cells were found in great numbers in the liver; and the heart did not reveal anything remarkable. The sections of the skin had a myxomatous-like deposit in the subcutaneous tissue, which was infiltrated with young fibroblasts. New blood vessels also appeared here and there in this layer in the subcutaneous tissue.

In the first series with the rats, the healing rate was found to be approximately the same for both the experimental and control animals.

In the second series, it was also found that the healing rates of rats numbered 1 and 3 were not remarkably different. However, in the same series, rats nos. 2, 5, and 6 revealed an average daily decrease in their wound area from 26 to 32 percent more rapid than their control. (This percentage was calculated as follows: Average rate of decrease in area per day equals $\frac{\text{initial area of wound}}{\text{number of days to heal}}$. A equals average decrease in area per day of experimental animal, and B equals average rate of decrease per day of control. Percent of decrease in size of wound of experimental animal as compared with the control equals $\frac{A}{B'}$ or $\frac{A-B}{B}$ equals percent difference of the two rates.) Table 1 gives the data and results for the second series.

TABLE 1.—*Series 2*

Rat no.	Size of wound	Time	Remarks
	Square inches	*Days*	
1	0.405	0	Received an equivalent of 3 grains of desiccated thyroid daily for 34 days. An adult rat. Weight 195 g. Control inadequate due to age.
	.289	5	
	.213	10	
	.062	15	
	.000	34	
3	.389	0	Control for number 1. Weight 120 g. A young rat.
	.262	5	
	.176	10	
	.069	15	
	.000	33	
2	.262	0	Received an equivalent of 3 grains of desiccated thyroid daily for 20 days.[1] Weight 100 g. A young rat. Average daily decrease in wound area 26 percent more rapid than no. 4.
	.153	5	
	.129	10	
	.063	15	
	.000	20	
5	.276	0	Received an equivalent of 3 grains of desiccated thyroid daily for 20 days.[1] Weight 80 g. A young rat. Average daily decrease in wound area 32 percent more rapid than no. 4.
	.149	5	
	.127	10	
	.114	15	
	.000	20	
6	.202	0	Received an equivalent of 3 grains of desiccated thyroid daily for 15 days.[1] Weight 90 g. A young rat. Average daily decrease in wound area 29 percent more rapid than no. 4.
	.102	5	
	.061	10	
	.000	15	
4	.270	0	Control for 2, 5, and 6. Weight 110 g. A young rat.
	.172	10	
	.151	15	
	.000	26	

[1] The same dosage of thyroid was then continued until the end of this series, 34 days.

In the third series, all of the animals showed a decrease in their healing rates as compared with each of their healing rates in the two former series. Rat no. 1 failed to manifest any gain in its healing rate over its control. The gain that rats nos. 2, 5, and 6 revealed in their healing was not as marked as it was in the second series. The daily decrease in the wound areas averaged only from 13 to 17 percent over their controls. The data and results of this series are shown in table 2.

TABLE 2.—*Series 3*

Rat no.	Size of wound	Time	Remarks
	Square inch	*Days*	
1	0.266	0	Received an equivalent of 3 grains of desiccated thyroid daily for 34 days, and then 10 grains for 28 days. Weight, 240 g.
	.000	28	
2	.266	0	Received an equivalent of 3 grains of desiccated thyroid daily for 34 days and then 6 grains daily for 24 days. Weight, 105 g. Average daily decrease in wound area 17 percent more rapid than no. 3.
	.000	24	
5	.266	0	Received an equivalent of 3 grains of desiccated thyroid daily for 34 days and then the same dose continued for 25 days. Weight, 105 g. Average daily decrease in wound area 13 percent more rapid than no. 3.
	.000	25	
6	.249	0	Received an equivalent of 3 grains of desiccated thyroid daily for 34 days and then the same dose continued for 23 days. Weight, 135 g. Average daily decrease in wound area 13 percent more rapid than no. 4.
	.000	23	
3	.255	0	Control for rats nos. 1, 2, and 5. Weight, 150 g.
	.000	27	
4	.249	0	Control for no. 6. Weight, 110 g.
	.000	26	

All of the animals were autopsied at the end of the third series. There was no clinical or gross pathological evidence of disease in either the experimental or control animals. Histological studies of the thyroid, heart, aorta, liver, and skin were made through the courtesy of the United States Naval Medical School, Washington, D. C., which reported that nothing unusual could be seen from the microscopical examination, and that all of the rats showed a similar picture.

COMMENT

After tremendous doses of thyroid by mouth, the white mice in the first experiment developed clinical and pathological hyperthyroidism. There was evidence of fibroblastic proliferation in a peculiar deposit in the subcutaneous tissue of the skin, and a plasma cell infiltration in the liver substance. The significance of the plasma cells is that, according to some histologists, they are the precursors of fibroblasts.

The white rats, when fed an equivalent of 3 to 6 grains of thyroid by mouth daily, showed an increase in their healing rate of superficial wounds, with one exception, which occurred in an adult rat without an adequate control. It has been shown that the healing rate in young rats is more rapid than in adults, because fibroplasia begins earlier and is less retarded (9). The rates of cicatrization in these rats were greater at the beginning than at the end of the period of repair. This fact was pointed out several years ago by Carrel and Hartmann, who also found that the rate of cicatrization depends on the area, rather than on the age of the wound: i. e., the larger the wound the greater the rate of healing (10). Rats nos. 1 and 3 revealed a greater rate of healing than no. 4 in the second series, which can be explained on the basis of the larger wounds in the case of the first two animals. Therefore, it can be seen from the foregoing that in order to compare the rate of cicatrization in two animals, it is necessary that they be not only of the same age, but also that they have wounds of approximately the same size.

CONCLUSION

Desiccated thyroid based on the human therapeutic dosage seems to increase the healing rate of superficial wounds in young rats, and produces no harmful effects after daily administration for a period of approximately 3½ months. When this drug is used over a long period of time its effect on healing becomes less pronounced, and repeated wounds on the same animal apparently decrease the rate of cicatrization. This paper is offered only as a preliminary observation and no statement should be considered conclusive until further evidence in this study is submitted.

ACKNOWLEDGMENTS

Much credit is due the Fairview Hospital, Great Barrington, Mass., and Miss Marguarite Knight for the assistance rendered in carrying out this experiment. Valuable advice and suggestions were given by Capt. G. F. Cottle (M. C.) United States Navy, and Dr. William Dameshek, of the Beth Israel Hospital, Boston Dispensary, Boston State Hospital, and Tufts College Medical School. The paper was prepared for publication and the mathematical calculations were carefully checked by Lt. L. C. Cook, Coast Artillery Reserves, and my wife.

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CLINICAL NOTES

ANNULAR PAPULAR SYPHILIDE

By W. S. Rizk, Lieutenant, junior grade, Medical Corps, United States Navy

Annular papular syphilides are observed in less than 1 percent of the total early cutaneous secondary manifestations of syphilis (1), but average over 16 percent of the mucocutaneous relapses (2) seen in inadequately treated syphilis. The importance of relapse lesions from a public health point of view is paramount, since they are frequently highly infectious, and, therefore, seriously influence the potential propagation of the disease. Unfortunately, such lesions are often overlooked or unrecognized both by the patient and the physician following the disappearance of the early recognized lesions after an antileutic regime that is all too often inadequate. As to the incidence of mucocutaneous relapse, Stokes and his associates in a study of 5,952 cases, observed a 6.05 percent relapse rate, when the observations were extended regardless of the age of the infection, although the vast majority (93 percent) occurred before the end of the second year. Chesney (3) considers the immunological problems concerned in relapse and reinfection.

The annular papular syphilide is to be distinguished, at the outset, from the extremely rare annular macular syphilide or so-called "neuro-syphilide of Unna", that Dr. Howard Fox (4) called attention to in this country with the report of a case. Combes (5), in 1932, wrote an excellent monograph on this rarer type of skin manifestation in syphilis (only about 50 cases have hitherto been reported in the literature), which he considers to be not a syphilide at all in the histopathological sense of the word, but a vascular phenomenon of the disease. In this article, I refer to the much more frequent true annular papular syphilide that one sees most frequently in the negro, especially upon the face as a recurrent syphilide.

REPORT OF A CASE

J. R., a Negro mess attendant, aged 22, single, nativity Georgia, was admitted to hospital on January 2, 1935, diagnosis, syphilis. Has been in the naval service since February 6, 1933. No serious illnesses

in past history, aside from a gonorrheal urethritis developing November 13, 1933.

Present history.—Syphilitic infection dates from December 11, 1933. History of sexual intercourse in Tampa, Fla., with Negress, November 8, 1933. Diagnosis established aboard ship from the finding of the specific treponema from sore in coronal sulcus dorsally. Blood Kahn taken aboard ship was negative at the time. It was negative likewise on June 14, 1934, 2 months after the completion of a course of treatment, the test being made at the United States Naval Hospital, League Island, Pa. No history of a secondary eruption elicited.

On December 3, 1934, approximately 1 year following original infection, patient reported to the sick bay aboard ship with three small ulcers on glans penis. History of sexual intercourse 4 days previously. A diagnosis of chancroid was made upon failure to find the treponema pallidum. This diagnosis was probably in error as a Kahn blood test made December 27, 1934, at this hospital was 4 plus. Patient was then transferred to this hospital with a notation in health record that "ulcers are practically healed. One shows some induration at the base, which is apparently a second primary." Record of treatment as noted in health record, until time of admission to hospital was as follows:

From December 11, 1933, to April 13, 1934: 12 injections arsenicals and 12 injections bismosol.

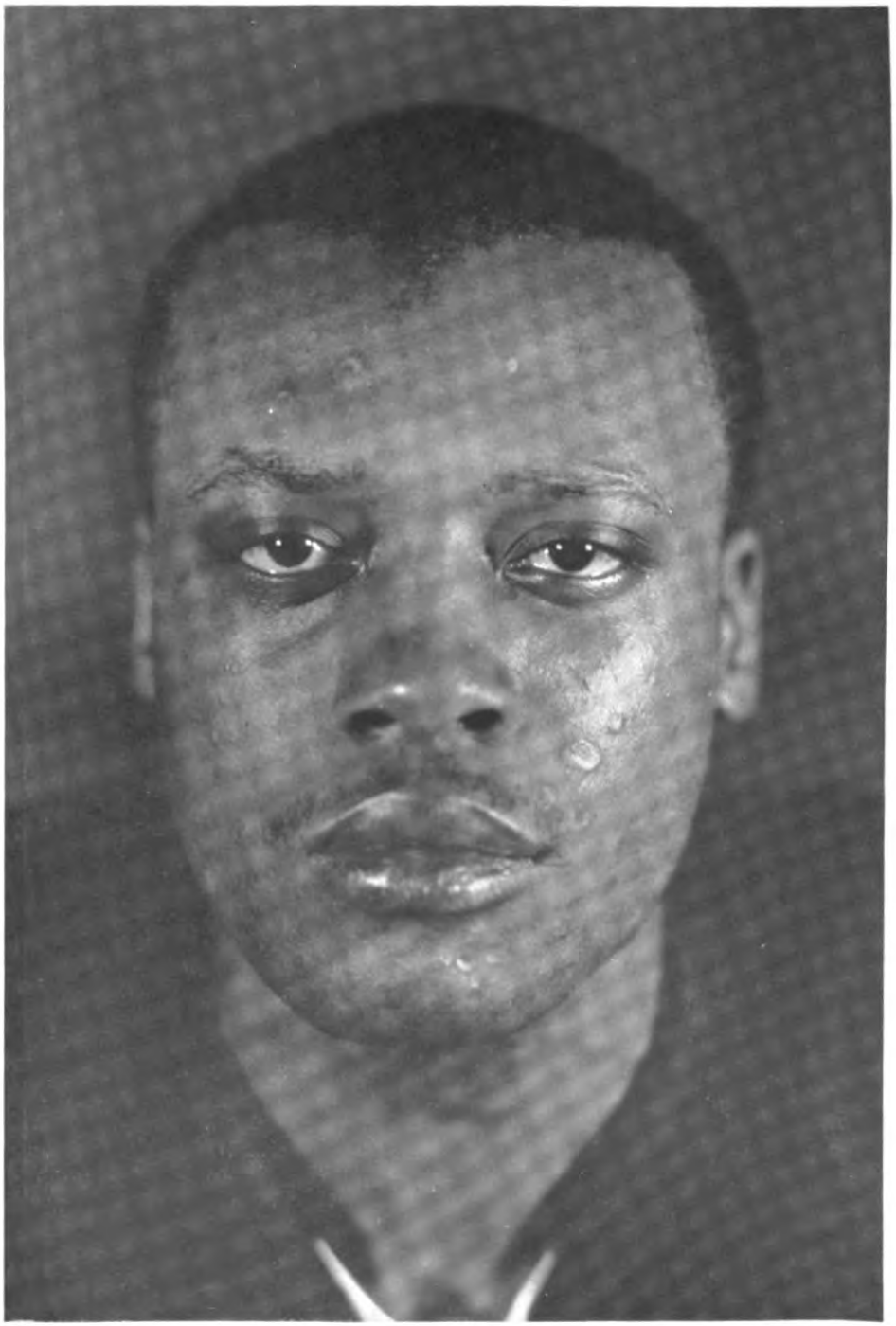
From July 15, 1934, to August 16, 1934: 6 injections neoarsphenamine and 12 injections bismosol.

From September 27, 1934, to October 18, 1934: 6 injections neoarsphenamine and 8 injections bismosol.

NOTE.—No mention is made of the specific amounts of the drugs given or the kind of arsenicals used in the first course.

Physical examination at the time of admission.—Robust, well-developed negro, station and gait normal, mental reactions normal. Weight 158 pounds. Blood pressure 118/80. Temperature 98.6, pulse 76, respiration 18. Healed ulcers were present on dorsum prepuce and small sear in left coronal sulcus dorsally. Lymph glands were definitely enlarged and indurated in inguinal, epitrochlear and cervical regions. Oral mucous membrane was devoid of luetic stigmata. Examination in other respects was also essentially negative.

Progress notes.—Patient was placed on neoarsphenamine intravenously once weekly (first dose 0.3 gram, second dose 0.45 gram, and subsequent doses of 0.6 gram) and bismosol intramuscularly twice weekly 1 cc each injection. Patient also received KI by mouth in graduated dosages. On January 22, 1935, following the third injection of neoarsphenamine (total 1.35 g) and six injections of bismosol



ANNULAR PAPULAR SYPHILIDE.

(1 cc each) patient developed an annular papular syphilide on face, about the cheeks and chin, characterized by about 40 complete and incomplete rings, with definitely raised and indurated circumferences and arcs, and varying in size from 1 to 15 mm in diameter (fig. 1). Darkfield examination of serum collected from one of the abraded circinate papules was positive for Treponema pallidum. On January 30, 1935, papular rings had disappeared leaving only darker colored macular areas where rings were present. On February 18, 1935, the residual areas of pigmentation were in turn, fading away.

Kahn blood tests taken on January 3, 7, 14, and 31, and February 18, 1935, have remained consistently 4 plus.

DISCUSSION

A chronological consideration of the dates on which patient developed the gonorrheal and leutic infections renders it probable that patient was infected with both diseases simultaneously. The recurrence of the penile lesions is significant. While no treponemata were found in the serum expressed from these lesions, it is highly probable that they were leutic in etiology and are to be considered as monorecidive lesions, which are usually the earliest form of mucocutaneous relapse. The subsequent development of the annular syphilide is a later type of cutaneous relapse, although in reality it is a delayed secondary rash, since the first group of secondaries did not appear or was overlooked.

It is highly important to differentiate the annular papular syphilide from other skin conditions that it may simulate, such as epidermophytosis, erythema multiforme, annular lichen planus, annular seborrheic dermatitis, annular pityriasis rosea, impetigo contagiosa, and annular granuloma. Any work on modern syphilology may be consulted for the distinguishing criteria. The pathognomonic sign of differentiation is the finding of Treponema pallidum in serum from one of the annular papules, although treponemata are not always found on such an examination.

Even with the finding of the specific treponema, it may be very difficult, in some cases, to say whether it is a case of syphilis or yaws. I am indebted to Capt. C. S. Butler (Medical Corps, U. S. Navy) for pointing out to me the similarity that is often observed between annular syphilides and some cases of framboesial dermatoses. Captain Butler in his writings on the treponematoses, has expressed himself on that very phase of the problem (6). Anyone who has searched the literature on this point cannot fail to come upon reported cases of syphilis that are framboeisiform in description. Such a case, for instance, was the one reported by Kingsbury (7), as a circinate syphilide: "The patient was a full-blooded negro

employed as a railroad porter. He was a native of the West Indies, but had been in this country for many years * * * Impossible to obtain history of any early manifestations of syphilis. Only cutaneous evidence of the disease at the time he was before the society was a circinate squamous lesion in the center of the forehead, breaking over the bridge of the nose. It was said by the patient to have been present over a year. The ring was about 2 inches in diameter, the border was moderately elevated and was an eighth of an inch wide. The lesion rapidly disappeared when the man was placed on specific treatment." Conversely, Nichols (8) reported a case of recurrent circinate dermatoses in a colored soldier, which was presented as a case of yaws, but which was consistent, withal, with the morphology and behavior of a relapsing circinate syphilide due to inadequate therapy. Nichols, in fact, later (9), assumed on the finding of a positive spinal serology that it might be a case of syphilis rather than yaws, since it is not believed that a cerebospinal involvement is a manifestation of framboesial infection.

As regards treatment, it is apparent that my case, herein presented, was inadequately treated from the beginning. We have progressed far from the conception of Fournier (10) who attributed recurring syphilitic roseolas to a modification of the syphilitic process by mercury. It is now quite generally agreed that the vast majority of recurrent or relapsing syphilides are due to inadequate treatment, and to rest intervals between treatments of too long a duration.

Syphilologists today are predominantly in favor of the continuous treatment of syphilis during the first year of infection, rather than the intermittent treatment with rest periods. An adherence to this principle with a competent follow-up system of adequate treatment to insure the eradication of the infection, will make relapsing syphilides rare indeed.

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RUPTURE OF THE DIAPHRAGM—REPORT OF TWO CASES

By J. J. A. McMULLIN, Captain, Medical Corps, G. B. McARTHUR, Commander, Medical Corps, and H. M. WEBER, Lieutenant, Medical Corps, United States Navy

Herewith are presented the case histories and hospital records of, first: An acute herniation through the left posterior portion of the diaphragm, with no history of trauma other than that incidental to the increased intra-abdominal pressure occurring during sneezing and, second; a herniation following a stab wound which involved the left diaphragm.

Case 1.—A young man of 21 years who had never had any serious illness. At about 10 a. m., August 9, 1934, had a moderately severe pain in the upper abdomen after a spell of sneezing. His pain was severe enough to cause him to sit down; was colicky in character, radiated into the left costo-lumbar area and was not associated with nausea. He continued to try to do the painting which he was doing, and at noon had a small amount of food though he was not particularly hungry. At 1 p. m. he reported to the sick bay and was given a hypodermic injection because the pain was becoming more severe. At that time he felt weak, was "sweaty" and was put to bed with hot water bottles and blankets. Several times during the afternoon and night be attempted to drink water but, to use his own words, "it came right back", indicating perhaps that it was a rather quick regurgitation rather than vomiting because this water that returned was only slightly colored with brownish green stain. Several times had hiccoughs for short periods.

Pain kept him awake all night, becoming unbearable unless he turned on left side. It centered about the left hypogastric area and into the back especially the left side; did not feel nauseated.

Bowels usually regular; no urinary symptoms; no previous gastro-intestinal difficulty nor pain.

Physical examination revealed skin pale with beads of perspiration; face drawn, lips cyanotic, respiration shallow, nose cold, patient listless and apathetic. Over left chest the percussion note was normal, voice transmitted with usual characteristics but there were no breath sounds; apex beat to the right of the sternum; heart sounds loud and booming though the pulse was thready and rapid; only comfortable position on left side with knees drawn up. The abdomen was felt to be flat with some rigidity of the upper abdominal muscles and marked tenderness in left hypochondrium; liver dullness present. There was some movement of the abdomen during respiration though shallow, as was also thoracic respiration. Deep breath caused severe aggravation of pain in hypochondrium and back. Marked tenderness in left costo-lumbar area with lumbar muscle spasm. Anus and genitalia, negative. Extremities, cold and clammy.

Treatment.—Put to bed with hot water bottles and blankets; 1,000 cc normal saline and 5 percent glucose by vein started on admission. Nothing by

mouth. Patient laid quietly on left side with knees drawn up in much pain, being resistant to examination and distressed by answering questions. It is to be noted that he was not nauseated, was thirsty but not hungry. Pulse thready and 180, respiration 25, temperature, 100. By 1 p. m. it was felt that his reaction from shock was sufficient to warrant exploratory operation. Received morph. sulph. gr. ¼ with atrop. sulph. gr. 1/150 and taken to operating room.

Operation record by Dr. J. J. A. McMullin.—Upper right rectus incision. Abdomen filled with blood and blood clots. Blood aspirated and baled out; an exploration of abdominal cavity made. A rent in the left posterior diaphragm through which the stomach and several feet of small intestines had herniated, was found. The opening in the diaghpram was enlarged by a short incision at right angles to the tear and the stomach and intestines were returned to the abdomen. The diaphragm was repaired with six interrupted sutures, and the abdomen closed in layers without drainage.

Intravenous infusion of 5 percent glucose in normal saline started during operation and blood donor summoned for transfusion. Received 750 cc 5 percent glucose in normal saline; post-operative condition poor. Pulse was fairly firm, rate 150; foot of bed on blocks; blood transfusion 500 cc, citrated blood at 3:30 p. m. followed by 500 cc, hot 5 percent glucose in normal saline intravenously; reaction fair; reacted fairly from anesthetic; levin nasal duo denal tube passed for siphon drainage. Some air evacuated through tube and 100 cc brownish-red liquid. Patient rational at times; respirations 25 to 30 and rather shallow; left chest tympanitic; P. M. 1 to right of sternum; heart sounds loud and booming. During late afternoon respirations became progressively more shallow; pulse weaker; patient lapsed into a stuporous state and at 9:55 p. m. was pronounced dead.

Autopsy revealed the repaired tear in the diaphragm about 3 inches long, securely closed. There was found in the pleural cavity about 1,500 cc of bloody fluid and perhaps 2,000 cc in the peritoneal cavity. This tear had occurred in what is described as the trigonum lumbo-costale of the diaphragm. It is the interval between the medial margin of the sterno-costal portion and the lateral portion of the lumbar portion of the diaphragm. A muscular hiatus is present on one or both sides of the diaphragm in 80 percent of bodies; disclosing the lower border of the pleura, it varies in size from a slight separation of fibers to a large gap. This is the site of the usual congenital type hernia.

It is probable that in this case the entering wedge for this herniation was through this space, subsequently tearing the thin muscular sheet upwards.

This case is of interest for the following reasons: (1) There was no trauma preceding the herniation, the symptoms coming on after several attacks of sneezing, while the patient had his arms raised, painting overhead. There no doubt was a deficiency of muscle at the trigonum lumbo-costale, through which the hernia made its entrance into the pleural cavity. (2) As to preoperative diagnosis, it is felt that this could have been successfully made. The patient, however, was desperately ill and suffered from any examination or movement. A portable X-ray plate was not satisfactory. Some most unusual condition was suspected, though a ruptured abdominal viscus was considered as a probable cause. (3) It is of interest to

note that the usual symptom of vomiting was not present, no doubt because the herniation of the stomach was such that it was not mechanically possible.

Case 2.—Man, age 21, in excellent general health. On November 4 was attacked and stabbed. Patient ran 100 feet, lost consciousness and was taken to a civilian hospital, where after administration of "first aid" he was brought to this hospital.

Physical examination on admission revealed only a clean stab wound below left scapula at the level of the tenth interspace. Lung resonance was normal as were breath sounds. No abdominal tenderness or rigidity.

Progress of case.—Treatment was expectant. Patient kept in bed and was comfortable with normal temperature, pulse and respiration until November 8. On this day he noticed that while on the bed pan he had a sharp pain in the left lower chest. This did not particularly disturb him but on the morning of November 9 the pain became more distressing and continued during the day though not severe enough to interfere with his eating. Temperature rose in afternoon to 100, pulse was 100, and respirations were 24. He slept fairly well during the night. On the morning of November 10 any movement of the patient was resisted because of the production of greater pain. He was moderately dyspnoeic, his pulse was 100, temperature 99, and respirations 24. During the late part of the day he refused to take anything by mouth, but there was no nausea nor vomiting. He became quite cyanotic, and at 6 p. m. the temperature had risen to 100.6 with a pulse of 110 and respirations were 28. Physical examination during the day at repeated intervals revealed absence of breath sounds in the left lower chest with an occasional gurgling sound. The remarkable finding however, was a tendency for the degree of tympany over the left lower chest to change from time to time. His pain was apparently quite severe and he appeared to be much worried about it. A modified Fowler's position was seemingly the most comfortable and, as said before, any movement, active or passive, was sufficient to aggravate this pain, and, of course, definitely decreased to a marked degree the depth of the respiratory excursions, each breath being taken very carefully and with a certain amount of hesitation. There was some left upper quadrant tenderness and rigidity. There was a leucocytosis to close to 12,000, 75 percent of which were mature polymorphonuclears. X-ray examination with portable flat chest plate was not satisfactory, so a small amount of barium was administered. A film at this time revealed a "contant collection of barium in the cardiac region."

On the morning of November 11, under Pantocain spinal anesthesia an upper left rectus incision was made from ensiform to 2.5 cm below the umbilicus. A hernia in the posterior portion of the left diaphragm 5 cm from the midline, had allowed the fundus of the stomach and a portion of the spleen to enter the pleural cavity. The hernial opening was enlarged and the contents reduced. A quantity of blood was evacuated from the pleural cavity and the tear sutured with interrupted sutures of no. 2 chromic catgut. Abdomen was closed without drainage. His convalescence was quite stormy. He developed some broncho-pneumonic areas in the right lung, and until the left lung began to again expand, his resulting dyspnoea and cyanosis was effectively treated with the oxygen tent. Two weeks after operation he was free of symptoms, and the wound had cleanly healed. X-ray examination revealed a clear right lung field with probably 50 percent expansion of the left. Three weeks after operation he was up and about. Fluoroscopic examination with

a barium meal on January 15, 2 months and a few days after operation, reveals a normally functioning left diaphragm and he was returned to active duty.

REMARKS

1. Neither case exhibited the usually described symptoms of nausea and vomiting.
2. While the herniation was in both cases in almost the same relative area of the diaphragm, one patient found his most comfortable position to be on the left side with the knees drawn up while the other was most comfortable in a modified Fowler's position. It is to be particularly noted that any slight movement, active or passive, in either case aggravated the pain exceedingly much.
3. The physical findings were essentially similar in the two cases, and diagnosis by an opaque substance preceding X-ray examination clinched the diagnosis in one case but unfortunately was not satisfactory in the other.
4. Both cases were false types of herniation, in that neither were covered by a sac. Traumatic hernia of the diaphragm are always without a sac, while congenital or acquired types may be true or false.
5. While relatively a rare condition, it should be borne in mind as a possible cause of an unusual sequence of symptoms involving the upper abdomen and diaphragmatic area.

MUMPS WITH PRODROMATA OF MENINGO-ENCEPHALITIS

By ROLLAND R. GASSER, Commander, Medical Corps, United States Navy

A few case of mumps have been constantly present in this hospital for several months. The following case is of interest because of the apparent great rarity of the occurence of symptoms and signs of a meningo-encephalitis as a prodromata of mumps. Meningeal symptoms and signs have supervened in more than a few instances, but the writer only finds such symptoms and signs as prodromata recorded in Nelson's Loose Leaf Living Medicine, where mention is made of three cases. Even during an epidemic of mumps the occurrence of such signs and symptoms presents a diagnostic problem.

Midshipman G. H. Age 23. Admitted United States Naval Hospital, Annapolis, Md., on January 9, 1935. Diagnosis: Dislocation, articular cartilage, right knee. Operation performed January 11, 1935. Uneventful convalescence from operation. Was to be discharged to duty January 30, 1935.

Past illness.—History of chronic sinusitis of left antrum several years ago. Acute otitis, media, left ear, in December 1934.

Current complaint.—Patient retired Monday night, January 28, 1935, in apparently good health. About 4 a. m., January 29, 1935, was awakened by severe frontal headache, vomiting and vertigo if attempting to sit up.

Physical examination.—Patient examined first thing in morning of January 29, 1935. Temperature normal, pulse 80, respiration 18. Moderate rigidity of neck; complained of pain on forced motion of neck; slight lateral nystagmus to right; reflexes all normal; no kernig; no paralysis; sensation normal; ears normal; no evidence of sinusitis; heart negative; lungs clear; blood pressure 144/66; headache severe; vertigo on movement of head; frequent vomiting. W. B. C., 10,650. Polys., 70 percent. Lymphs., 30 percent. Bands 3, segmented 67. Headache, vomiting, and vertigo continued. At 3 p. m. temperature went to 101.4, pulse 110, respiration 24. Marked hyperesthesia developed along the spine. Spinal puncture done. Spinal fluid under 12 mm, hg pressure. Clear; sugar 43 mgm per 100 cc; chlorides 693 mgm per 100 cc; cell count 50, 100 percent lymphocytes. Globulin not increased. Headache not influenced by drainage; vomiting and vertigo unchanged.

January 30, 1935: Patient had a fair night under morphine. Headache, vertigo, and vomiting persist; stiffness of neck; no nystagmus; no paralysis; no anaesthesia; no rash; reflexes normal; hyperesthesia along spine less marked; systolic murmur present over precordia; temperature at 7 a. m., 100.8; at 11 a. m., normal; at 3 p. m., 103.4 by axilla; at 7 p. m., 104.6 by axilla; pulse stayed around 110; patient conscious.

Necessary to give fluids by hypodermoclysis. W. B. C. 13,150. Polys, 88 percent; lymphs, 12 percent; bands 1, segmented 87. Consultation with Dr. Leake of the United States Public Health Service. Acute poliomyelitis ruled out. Memingo-encephalitis of undetermined origin.

January 31, 1935: Again had a fair night under morphine. This morning headache continues; vomiting is less frequent and vertigo is practically negligible. Stiffness of neck less marked. Systolic murmur less marked. Small palpable gland in left submaxillary region. Gland hard, discrete, and not tender, but slightly painful. During the day temperature (axillary) gradually dropped to 99.4, pulse dropped from 110 to 56. Gland in submaxillary region rapidly increased in size and by night was moderately enlarged and stood out.

February 1, 1935: Had a good night under mild sedation; left submaxillary gland greatly swollen and painful; no other glands involved; headache continues but less severe; systolic murmur only irregularly and faintly heard; very little stiffness of neck; vomiting infrequent; now can take gland fluids. W. B. C. 6,000. Polys, 74 percent; lymphs, 17 percent; baso, 1 percent; mono, 8 percent. Bands 1, segmented 73. Temperature during the day ranged from 99.6 to 101 by mouth; pulse 52 to 70.

February 2, 1935: Had a good night. Vomiting has stopped. Headache continues but is decreasing in severity. Systolic murmur absent. Left submaxillary gland enormously enlarged. General condition very good. Taking nourishment freely. Temperature normal to 100; pulse 68 to 82.

February 3, 1935: A good night. Left submaxillary gland is beginning to subside; no evidence of rigidity of neck; headache intermittent; head feels tight; temperature normal to 99.2; pulse 68 to 78.

February 4, 1935: Left submaxillary gland rapidly subsiding; temperature normal; pulse 50 to 56; headache intermittent, convalescence uneventful. All evidence of headache or tight feeling of head subsided on February 9, 1935. Pulse returned to normal on February 7, 1935.

February 15, 1935: Patient allowed up and about and was discharged to duty February 21, 1935, well.

PROSTATITIS DUE TO CHRONIC, CATARRHAL, FOLLICULAR TONSILLITIS

By EDGAR RICEN, Lieutenant, Junior Grade, Medical Corps, United States Navy

Inflammations of the prostate gland are all too often associated with gonorrhea by many clinicians. To many, prostatitis and gonorrhea mean one and the same thing. Nothing could be further from the truth. Gonorrhea and prostatitis are by no means synonymous. It is, of course, much easier to attribute each case of prostatitis of unknown origin to the gonococcus than it is to make a careful and painstaking search in an effort to discover the true etiological agent. The prostate gland may become infected in any number of ways:

(1) From the bacteria normally occuring in the anterior urethra.

(2) As an extension from a gonorrheal infection of the urethra.

(3) From the retention of urine in the urethra from obstruction, i. e., stricture.

(4) As a descending infection from the kidney or bladder.

(5) From undue sexual strain, which creates a chronic passive congestion of the prostate, thus rendering it more susceptible to the normal inhabitants of the urinary tract.

(6) As a complication of foci of infection, in the nose, throat, and teeth. Also as a complication or sequelae of any acute infection.

Before making a diagnosis of any specific or nonspecific prostatitis a thorough and exhaustive search should be made for the offending organism. It is only after the etiological agent has been positively identified and the cause removed that an intelligent diagnosis can be made. Many young men have been needlessly treated and unduly alarmed because a hasty diagnosis was made, based solely on the presence of a purulent urethral discharge. It is in this category that the case reported below belongs:

Case report.—E. S., aged 18, Civilian Conservation Corps patient, was admitted to the station hospital, Fort Wayne, Detroit, Mich., August 24, 1934.

(1) Sore throat. (2) Urethral discharge. (3) Pain in the lower rectum and sacral region of the spine.

Essentially negative.

Has had measles, mumps, chicken pox, and frequent attacks of tonsillitis. Tonsillitis has been more frequent and severe during the past 6 months. Denies ever having any venereal infection.

In February 1933 patient had a severe attack of tonsillitis. Within a few days after the onset, he first noticed a urethral discharge. At this time, he consulted a civilian physician who told the patient that he had gonorrhea. He was treated at this time with urethral irrigations. As the attack of tonsillitis subsided, the discharge gradually diminished. In April 1933 a similar attack of tonsillitis was followed by a urethral discharge, which again cleared up after the tonsillitis subsided. After the last illness, the patient began to notice pain in the lower rectum and sacral region of the spine. A week before his admission to the station hospital, the urethral discharge renewed itself. He was admitted, with the complaints listed above.

Physical examination.—Ears negative; pupils equal, regular, and react normally; nose negative; gums and teeth in good shape; hypertrophic cryptic tonsils; posterior pharyngeal wall injected; thyroid not palpably enlarged.

Heart rhythm regular. Rate: 72. No enlargement; no murmurs.

Lungs are negative.

Abdomen shows no rigidity. No tenderness; no palpable masses.

Rectal examination: Both lobes of prostate gland are enlarged, tense, and tender to touch.

Reflexes.—All elicited and equal.

Temperature, 99.4; pulse, 68; respiration, 18.

Urinalysis.—Color, straw. Appearance, cloudy. Reaction, alkaline. Specific gravity, 1.012. Albumin, a trace. Sugar, negative. Microscopic, numerous pus cells and staphylococci.

Blood.—White blood count, 9,200. Red blood count, 4,200,000. Hemoglobin, 80 percent.

Urethral smear.—Negative for intracellular diplococci. Numerous pus cells and staphylococci present.

Course of disease.—On August 28, 1934, a tonsillectomy was performed under local anesthesia. The pathological diagnosis was chronic, catarrhal, follicular tonsillitis. Three days later, the urethral discharge had entirely subsided and did not return. Daily rectal examinations revealed the prostate gland diminishing in size and tenderness. At the time of the patient's discharge, the prostate gland was of normal size and consistency. It was also no longer tender to the touch. Repeated prostatic smears revealed the absence of intracellular diplococci and also of pus cells. Subsequent urinalyses were also normal. The complement fixation test for gonorrhea was reported negative by the Army Medical School, Washington, D. C.

CONCLUSIONS

(1) Infections of the prostate gland are too often associated with gonorrhea. Prostatitis and gonorrhea are by no means synonymous.

(2) In all cases of nonspecific prostatitis a careful and painstaking search should be made for the etiological agent before treatment is instituted or the patient is told that he has gonorrhea.

(3) Foci of infection, especially chronically infected tonsils, may and often are the source of the infected prostate gland.

ACUTE MYELOGENOUS ALEUKEMIC LEUKEMIA, WHICH SIMULATED AGRANULOCYTOSIS—A REPORT OF A CASE

By JOHN J. WELLS, Lieutenant, junior grade, Medical Corps, United States Navy

This case is presented because it offers a differential diagnosis between primary agranulocytosis and an aleukemic leukemia (alekemic myelosis).

A 39-year-old white, single male entered the station hospital, Fort Ethan Allen, Vt., on October 9, 1934, complaining of a sore throat, sore mouth, and general weakness of 2 weeks' duration.

The present illness revealed that on September 21, 1934, the patient developed a sore throat. He reported to the camp surgeon and received mouth wash and pills. The sore throat continued and on September 25, 1934, he was admitted to the camp infirmary. The sore throat cleared up but his temperature which varied between 100 and 101 persisted.

On September 29, 1934, he was seen by the district dentist and the lower left fifth tooth was extracted in an attempt to find a cause for his temperature. For 24 hours following the tooth extraction the patient's temperature was 102.8, following which it fell and fluctuated between 100 and 101. On September 30, 1934, the lower gums became painful, red, and bled easily.

On October 8, 1934, a white blood count showed 3,300 cells, 8 percent of which were polymorphonuclear leukocytes.

On October 9, 1934, the patient was transferred to the station hospital.

Past history.—In the past history he stated that previous to his present illness he had not taken amido-pyrine or its allies, was not receiving arsenic, and was not exposed to chemicals.

Physical examination on admission to the hospital showed a well developed somewhat obese middle aged white male. There was paleness of the skin, lips, and mucous membranes. On moving about in bed there was dyspnea. The right nares was the site of a 3 by 4 cm scabbed over ulcer which had an inflammatory base. The lower and anterior left gums were red, spongy, and inflamed. The lower left fifth tooth socket was covered with a dirty grey necrotic membrane. The breath was foul. The tonsils were moderately enlarged and chronically inflamed. There was no membrane present. There was a tender marble sized deep lymph gland of the left anterior carotid triangle of the neck. The liver and spleen were not palpable. The temperature was 101; respiration 22; pulse 120. The white blood cell count was 3,200 cells with 2 percent polymorphonuclear-leukocytes and 94 percent small mononuclear cells. The platelets were diminished. The red blood cells were 2,632,000 Hg bn 60 percent. A smear was negative for Vincents spirochetes.

On October 12, 1934, the patient was placed on a diet high in vitamines B and pentose-nucleotides therapy consisting of 10 cc (0.7 g) intramuscularly B. I. D. The patient's white blood cell count responded very promptly, rising within 24 hours to 11,500 cells, with 4 percent polymorphonuclear leukocytes, 58 percent myelocytes, and 12 percent mononuclears.

On October 13, 1934, the patient developed hemorrhagic oozing from the naso-pharynx. This persisted despite packing with vaseline gauze. He vomited large clots of dark black blood mingled with bright red blood. His Hg bn fell to 40 percent. The red blood cells to 2,500,000 cells. He was typed for tranfusion but his serum agglutinated all other types including his own type (Moss 1). In an attempt to hasten coagulation he was given 250 cc of whole blood in the bottocks. This was noted as very unusual behavior for agranulocytosis, but because there was a possibility that we were dealing with agranulocytosis, pentose nucleotides was continued as excellent results were reported from the use of this drug in agranulocytosis whereas an acute leukemia was always fatal.

On October 16, 1934, the white blood cell count numbered 22,850 cells with only 7 percent polymorphonuclear-leukocytes and 71 percent myelocytic cells. A blood culture was positive for long chained streptococci.

On October 17, 1934, the patient died.

Discussion.—In this case we had a middle-aged white male who 19 days ago first developed a sore throat, persistent temperature, finally a tooth extraction. A rapid down-hill course characterized by air hunger, bleeding, severe anemia,

and prostration. He was given pentose-nucleotides for 5 days, twice daily. His white blood cell count rose from 3,200 cells, of which 2 percent were polymorphonuclear leukocytes to 11,500 cells within 24 hours, and on the fifth day of treatment numbered 22,850 cells, the majority (71 percent) of which were abnormal cells of the myelocytic series (promyelocytes myelocytes and metamyelocytes).

Jackson and his coworkers using pentose-nuclotides (1) in agranulocytosis stated that no clinical improvement could be expected before the fifth day of treatment, at which time there was an outpouring of matured polymorphonuclear leukocytes very similar to the outpouring of reticulocytes seen in pernicious anemia in response to liver extract therapy.

On the fifth day of treatment in this case the patient was extremely weak and prostrated. The red blood cell had fallen to 1,500,000 cells. The Hg bn was 30 percent. The white blood count was 22,850 cells. It was noted, however, that at no time did the mature polymorphonuclear leukocytes rise above 7 percent despite the high white blood cell count and the preponderance of myelocytes and metamyelocytes (71 percent). There was a constant ooze of blood from the naso-pharynx, and he showed multiple subcutaneous hemorrhagic areas. A culture was positive for blood stream infection of long-chained streptococci. The question then arose as to whether this was a case of primary granulocytosis of the malignant type (2) that is, that form of primary agranulocytosis, in which the maturation factor of the white blood cells was absent. The high white cell count being due to the chemotactic response of the bone marrow. The nucleic acid derivatives (pentose-nucleotides) supplied the stimulant but maturation of the white blood cells was absent or arrested. The severe and rapidly progressed anemia and thrombopenia being caused by an overwhelming infection which depressed both the platelets and the red blood cells. The high white blood cell count with its preponderance of myelocytic cells, "agonal' in type and a later stage of a septicemia. An aplastic anemia was ruled out by an occasional normoblast seen on a blood smear. When an acute leukemia first seen in an aleukemic stage was considered, we found that the blood picture fitted in very well. In the first place, an acute leukemia can pass through an aleukemic stage, and have "as its only evidence of aleukemia, a lack of differentiation or maturation of the white blood cells." Secondly, the blood picture in this case showed (*a*) a severe, achromic anemia with mild evidence of regeneration.

(*b*) A diminished number of platelets.

(*c*) A large number of immature white blood cells in a white blood count which varied from 3,000 to 22,000 cells.

Jackson (3) believes that at times it is extremely difficult to differentiate between acute leukemias and agranulocytosis because their signs and symptoms are closely alike. The diagnosis should depend more upon the clinical picture than upon biopsy, because pathological studies, frequently are inconstant and confusing. Given a clinical picture of a severe anemia, rapid in its development, accompanied by a diminished number of platelets, and hemorrhage; a variable white blood cell count, with a large number of immature cells favors a diagnosis of acute leukemia, because in primary agranulocytosis there is no anemia or a very mild one. The platelets are normal and there are only a few immature white blood cells. He very adequately sums up the situation by stating, "that until agranulocytosis is placed upon a more deliberate pathological basis, it will be difficult at times to differentiate the less fatal agranulocytosis from the always fatal acute leukemia."

Pathology.—At the autopsy, biopsy specimens were removed from the tibia and sternum, the liver and spleen. The spleen was one-fourth larger than

normal. These were forwarded to the Army Medical School, Washington, D. C. On microscopic sectioning and staining the following pathology was noted.

Spleen: Showed a diffuse hyperplasia or immature cells in the splenic pulp with atropy of the nodules due to overgrowth in the red pulp. The cells were immature, and resembled those of the granulocytic series with an occasional large giant cell resembling a megakoryocyte.

Liver: The liver grossly appeared normal. On section it showed indefinite cellular infiltration in the portal areas. The large sinusoids contained numerous large immature cells, of the granulocytic type.

Sections from the bone marrow were variable. The tibia was completely atrophic and contained no cellular exudate. That from the sternum was richly cellular, the majority of cells being immature granuloblasts and granulocytes.

Summary.—A report of a case is presented. Acute aleukemic leukemia oftentimes simulates primary agranulocytosis of the malignant type.

A severe anemia, diminished platelets, hemorrhages, and a large percentage of immature white blood cells, in a variable white blood cell count in acute leukemia. In primary agranulocytosis a normal red blood cell count, a normal number of platelets and a few immature white blood cells. In acute aleukemic leukemia of the lymphatic type, radiation at times will cause a marked lymphocytosis, thereby establishing the diagnosis. It is possible that the pentose nucleotides changed an acute myelogenous leukemia from an aleukemic stage to an acute leukemic stage? How many reported cases of primary agranulocytosis of the malignant type are aleukemic myelosis?

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ACUTE CATARRHAL PROSTATITIS RELIEVED BY TONSILLECTOMY IN THE PRESENCE OF ACUTE GONORRHOEA

By FREDERICK G. FOX, Lieutenant Commander, Medical Corps, United States Naval Reserve

Enrolled (W. P.) Civilian Conservation Corps, age 21, on duty at a camp in Michigan, contracted gonorrhea on August 18, 1934, and noticed his first symptom on the 24th, that consisted of a burning sensation in the urethra on urination. On August 26 he had his first discharge of serum or pus from the urethra, accompanied by pain on urination. He received no treatment at the camp for this disease, and on August 28 was transferred to the Station Hospital at Fort Wayne, Detroit, Mich. He was accepted as a patient, put to bed, and given the routine treatment. This consisted of 14 days' rest in bed a milk diet, and medication of urotropin and phenyl salicylate, in the regular dosage. The symptoms of burning, pain, and discharge from the urethra continued. On September 11 the patient was allowed to be out of bed, and the food was changed to a soft diet without spices. He also began using silver protean injections into the urethra three time a day.

The symptoms of burning and pain on urination, with a discharge from the urethra, continued until September 22, when he developed a urine retention due to a swelling of the prostate gland. This diagnosis was made by a finger palpation through the rectum, and there was found a generalized enlargement of the prostate gland. The patient was in distress due to the distended bladder, and he was catheterized. This treatment had to be repeated several times a day, and since there was danger of further infecting the urethra or bladder by catheterizing, and so producing a serious complication, an attempt was made to have the patient pass urine while taking a Sitz bath. This treatment was quite unsatisfactory, as it had to be repeated at least six times a day, and then he could only expell a small amount of urine, which but partially relieved the distended bladder.

On September 28, 1934, at 10 a. m., a tonsillectomy was performed under local anaesthesia, and on the same day at 8:30 p. m. before retiring for the night, the patient made an attempt to urinate, and was able to do so without difficulty, and he also stated that "he felt entirely different in the bladder." The gonorrhoeal discharge was not affected by the tonsillectomy.

An acute inflammation of the prostate gland is not an unusual complication of acute gonorrhoea. Its appearance causes an additional amount of suffering and distress to the patient, and calls for added treatment and care. It delays curing of the gonorrhoea, and tends toward extension of the inflammation and infection of the bladder. The need of relieving a distended bladder, calls for catheterization, which is painful to an already inflamed urethra, and produces a mechanical injury to the mucosa, that is liable to result in a stricture.

The presence of a gonorrhoeal infection in the urethra usually presages a similar infection in the prostate gland, when that gland enlarges and obstructs the free flow of urine. The case here presented proves that such is not necessarily the rule.

The prostatic retention developed 11 days after beginning the local injection of mild silver protein solution, and 18 days after noticing the burning on urination.

That the prostatic inflammation was due to a catarrhal condition, and not specific, is proved by the cure without massage of the gland.

The indication for the removal of the tonsils in this case followed an examination that revealed injected tonsil pillars, and the previous example of the tonsils as a point of focal infection in producing ulcers of the cornea, tinnitis aurium, goitre, rheumatism, and other diseases. This is the second case successfully operated by me resulting in a rapid cure of the enlarged prostate. The first case was one that was seen in consultation with Lt. (j. g.) Edgar Ricen, M. C., U. S. N., and reported by him. His case did not have gonorrhoea.

CONCLUSIONS

(1) Prostatic enlargements in the presence of gonorrhoea are not necessarily a complication of the urethritis.

(2) Focal infection should be removed in all cases of prostatitis of unknown etiology.

NAVAL RESERVE

MEDICAL CORPS

PROMOTIONS, SECOND QUARTER, 1935

Lt. John Clayton Norris, MC–V (G), U. S. N. R., from lieutenant (j. g.), MC–V (G), U. S. N. R., April 10, 1935.

Lt. James Bradford Vail, MC–V (G), U. S. N. R., from lieutenant (j g), MC–V (G), U. S. N. R.; May 24, 1935.

DENTAL CORPS

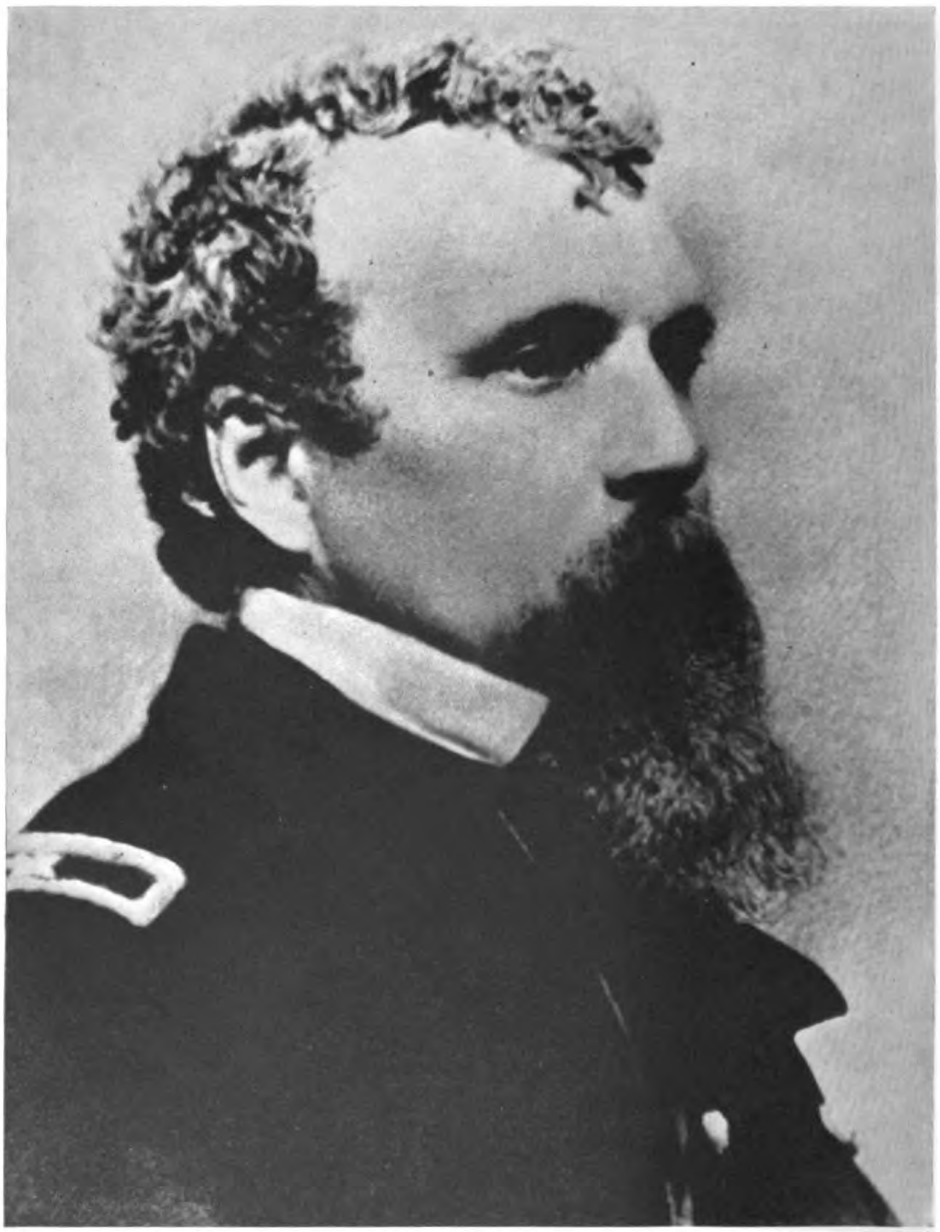
PROMOTIONS

Name	From—	To—
Clark, Edmond N	Lieutenant (junior grade), DC–V (G)	Lieutenant, DC–V(G).
Greenberg, Herbert H	do	Do.
Savage, George P	do	Do.
Ulen, Hall	do	Do.

MEETING OF MEDICAL RESERVE OFFICERS AT ST. LOUIS, MO.

A week of clinics and symposium on general and military medicine and surgery was held at St. Louis, Mo., for medical officers of the Army and Navy Reserve in that area. The meeting was very largely attended.

Capt. G. E. Thomas, Medical Corps, United States Navy, in charge of the Reserve Section, Bureau of Medicine and Surgery, Navy Department, and Lt. Comdr. R. H. Hunt, Medical Corps, United States Navy, from the recruiting station, Omaha, Nebr., were present as representatives of the Regular Navy.



JAMES C. PALMER

1872–1873

The seventh chief of the Bureau of Medicine and Surgery.

NOTES AND COMMENTS

JAMES C. PALMER, SURGEON GENERAL OF THE NAVY, 1872–73

James C. Palmer, the third to hold the office of Surgeon General of the Navy and the seventh to be Chief of the Bureau of Medicine and Surgery, was born in Maryland and appointed from that State as an assistant surgeon on March 20, 1834. His first service was on the frigate *Brandywine*, of the Pacific Squadron. From 1838 to 1842 he was with the celebrated Wilkes exploring expedition in the South Pacific, as medical officer of the U. S. S. *Relief*, one of the squadron with Admiral Wilkes which returned from Callao by way of the Sandwich Islands and Australia. He was commissioned surgeon October 27, 1841. He had service on the *Vandalia*, afterwards lost in the Samoan hurricane in 1889, and on the *Niagara* during the cable expedition of 1857. He was Admiral Farragut's fleet surgeon on the *Hartford* from 1863 to 1865, and participated in the battle of Mobile Bay. He was appointed Chief of the Bureau of Medicine and Surgery June 10, 1872, by President Grant, and held office until his retirement June 29, 1873. Thus the two Surgeons General of the Navy following the Civil War were the successive fleet surgeons with Farragut. The naval hospital in Yokohama, Japan, destroyed by the earthquake of 1923 and not rebuilt, was constructed during Palmer's term of office.

His picture suggests the typical Civil War officer in the uniform and cut of beard and hair.

Little can be ascertained regarding him after his retirement. His death occurred April 24, 1883.

SHIPS' WATER AS A SOURCE OF DISEASE

The purity of distilled water has been rather taken for granted by the Navy, a belief that has been rudely shattered in recent years by some very severe epidemics of water-borne diseases, notably the Guantanamo Bay outbreak of bacillary dysentery. Investigation shows that when ships are lying at anchor in the highly contaminated water of harbors and in close proximity to one another, either through priming or by leakage of contaminated cooling water into the dis-

tillate in the condenser, infection of the drinking water with disastrous results may occur.

The present fleet surgeon, Capt. K. C. Melhorn, recently instituted a bacteriological survey of the water of the San Pedro area and also of the distilled water in the tanks of various ships of the fleet anchored there. The survey was carried out by Lt. Comdr. W. W. Hall, chief of laboratories on the U. S. S. *Relief*. There were some significant results from this survey. It showed that the harbor water was very definitely contaminated and further that where the water from the ships' tanks showed high salinity it also was contaminated. The conclusion is clear that while the use of high salinity water for drinking might be defensible with the ship operating in the open ocean, it is a decidedly unsafe practice in harbors. Furthermore, a certain reservation must be made in regard to operation in the open sea, for as has been pointed out recently, our knowledge of the bacteriology of the sea is very limited.

When evaporators are working efficiently it appears that they produce a water so low in salinity that it is regularly checked as 0.5 grains or less per gallon, and by electrolytic detector methods used on the new type cruisers as low as 0.1 or 0.2 grains per gallon. Any rise in salinity appears to be evidence that pollution also may have occurred and it furnishes a kind of rough parallel test to bacterioligic methods.

The Bureau of Engineering early in the year published an exceedingly important circular letter (Bu. Eng. No. 34, Jan. 24, 1935) in regard to precautions to be taken to insure potable distilled water. The commander in chief, as a result in part of these recent studies in the San Pedro area, issued strongly worded instructions in regard to the subject.

THE MORTALITY FROM SYPHILLIS

" Men do not die of the diseases that afflict them " is an old medical maxim. It is peculiarly applicable to syphilis. Few other diseases show so low a mortality directly ascribed to them and few so great a mortality indirectly due to their effects. It is only when we examine into the remote effects of syphilis as a cause of mortality that we find it one of the important diseases as a cause of death. Recent research indicates that approximately 15 percent of all deaths from diseases of the circulatory system are due to lues. About 20 percent of deaths from diseases of the nervous system and an appreciable number from diseases of the kidney, liver, and stomach. An extremely important cause of deaths due to syphilis is that of deaths in infancy; as many as one-fifth are considered by some observers

to be due to syphilis. When these facts are considered, syphilis takes its proper place as one of the "Captains of the Army of Death", one of the great killers, and a genuine public enemy.

Recent careful studies in Germany including 8,500 autopsies from the Rudolf Virchow Hospital, indicate that the annual mortality rate of Germany is between 2.5 to 5 deaths per 10,000 inhabitants.

HEART DISEASE AMONG SEAMEN

In 1934, under the auspices of the United States Public Health Service, a careful study was made of heart disease among merchant seamen, both American and foreign. A small group of Coast Guardsmen were included. Though practically all the cases were merchant seamen this study made of cardiac disorders in seafaring men is of interest to naval medical officers. Particular attention was given to the etiology, and the work is of great value, therefore, as pointing the way toward prevention.

The investigator, Acting Assistant Surgeon Arenberg, United States Public Health Service, made a study of 189 cases of heart disease occurring among 2,500 admissions to the United States Marine Hospital, Ellis Island, N. Y. He found that heart disease from the standpoint of etiology was parallel to the findings in the general population except that there was a higher incidence of luetic cardiovascular disease and a lower incidence of rheumatic and arteriosclerotic types. This emphasizes again the importance of the prevention of syphilis and, if incurred, the importance of its early recognition and thorough treatment.

PROGRAM FOR THE 1935 MEETING OF THE ASSOCIATION OF MILITARY SURGEONS OF THE UNITED STATES

The forty-third annual meeting of the Association of Military Surgeons will be held in New York City, October 3, 4, and 5. The official headquarters will be at the Waldorf-Astoria Hotel. Following is the tentative program for this meeting which it is hoped will equal the meeting at Carlisle last year in attendance. The association has made great progress in membership during the past year. An enthusiastic meeting is anticipated of this association, which has done so much to advance the knowledge of military and emergency surgery and preventive medicine.

October 2: Registration at Waldorf-Astoria Hotel. Office open afternoon and evening of the 2d, as well as during the days and evenings of the convention.

October 3: A. M.—Opening of congress; speakers—Addresses of welcome;

President's address. Noon—Luncheon; demonstration. P. M.—Army medical supply depot—Inspection and demonstration. Naval medical supply depot—Inspection and demonstration. United States naval hospital—Inspection physiotherapy and other department. Dinner—Mess hall of naval hospital. Evening—Report on Eighth International Congress of Military Medicine and Pharmacy, Brussels, by delegates lately returned. Moving pictures of various military medical and surgical congresses, etc.

October 4: A. M.—Activities of Public Health Service; visit to quarantine station, New York Harbor. Noon—Luncheon. P. M.—Visit to Governors Island. Polo game; parade by the Sixteenth Infantry; reception and dance hour followed by dinner at the Governors Island Club.

October 5: A. M.—Business meeting. Noon—Luncheon. P. M.—Mitchell Field—Inspection of aviation activities; banquet—Waldorf-Astoria Hotel.

October 6 (Sunday): Trip to West Point; parade by the Corps of Cadets.

October 7: Visits to the Academy of Medicine, Rockefeller Institute, medical centers, and clinics for those who arrange to stay over; program for the ladies will be announced later by the ladies' committee.

BOOK NOTICES

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

The Editor,
UNITED STATES NAVAL MEDICAL BULLETIN,
Bureau of Medicine and Surgery, Navy Department,
Washington, D. C.
(For review.)

CLINICAL LABORATORY METHODS AND DIAGNOSIS, *by R. B. H. Gradwohl, M. D., Director of Laboratories, St. Louis County Hospital; lieutenant commander, Medical Corps, United States Naval Reserve.* 1,028 pages, 353 illustrations including 24 color plates. The C. V. Mosby Co., St. Louis, 1935. Price $8.50.

The growth of the importance of laboratory aids in diagnosis, prognosis, and treatment is one of the striking features of medicine in the twentieth century. Thirty-five years ago most of our commonly used laboratory tests were unknown. The examination of the urine was the only laboratory procedure usually used by the physician, and other tests were regarded with curiosity. Today clinical pathology has become one of the most essential branches of knowledge for the physician. A feature of Dr. Gradwohl's book is the space devoted to the interpretation of the meaning of laboratory findings. This is a most valuable thing, for often the physician is not quite sure of the full significance of laboratory data. The writer has necessarily encroached upon the fields of clinical medicine and surgery here but the trespass is more than justified in the results.

Another outstanding feature is the detail with which the technique is given. There is little opportunity for error left and thus the laboratory worker has an excellent guide for his work.

The illustrations of this books are another feature that will at once attract the attention of the student. They are remarkably fine. The black and white drawings, the photomicrographs, and the colored plates could not be surpassed for clearness and beauty.

PHYSIOLOGY IN MODERN MEDICINE, *by J. J. R. Macleod, M. B., L. L. D., D. Sc., F. R. C. P., F. R. S., regius professor of physiology at the University of Aberdeen, Scotland, assisted in the present edition by Philip Bard, Edward P. Carter, J. M. D. Olmsted, J. M. Peterson, and N. B. Taylor.* Seventh edition. The C. V. Mosby Co., St. Louis, Mo., 1935. Price $8.50.

It might be possible to practice the art of medicine without an understanding of physiology but to be a scientific physician it is essen-

tial that one have a thorough knowledge of the physiological principles which underly the science of medicine. It is difficult for one to acquire this knowledge from any one textbook but a careful reading of Macleod's seventh edition leads the reviewer to the opinion that it contains all the information about physiology that it is necessary for the practitioner of medicine to know. While this is strictly a scientific book it is written in language that may be readily understood by the average medical student or physician. Endocrines are playing an increasingly important part in medicine. If any one section of Macleod's book stands out above the rest it is the section devoted to this subject which, in this edition, was prepared by Prof. N. B. Taylor of the University of Toronto. It is greatly to be regretted that the untimely death of Professor Macleod will prevent him from supervising future editions, but it is greatly to be hoped that other editions will appear with Professor Taylor as a contributor. This is a book which should be available for reference and study to every naval medical officer.

DISEASES OF THE MOUTH AND THEIR TREATMENT, *by Herman Prinz, A. M., D. D. S., D. SC., M. D., Dr. Med. Dent. Professor of Materia Medica and Therapeutics, The Thomas W. Evans Museum and Dental Institute, School of Dentistry, University of Pennsylvania, Philadelphia, and Sigmund S. Greenbaum, B. S., M. D. Associate professor of dermatology and syphilology in the graduate school of medicine of the University of Pennsylvania;* Published by Lea & Febiger, Philadelphia. Price, $9.

This excellent book, by two well-known authors, contains 602 pages and 287 illustrations. The subject matter is well arranged and the book is rich in references. The complete index and well organized table of contents, should make it a valuable addition to medico-dental literature.

The authors have endeavored to combine the viewpoint of the physician with that of the dentist, and guided by their extensive classroom experience and clinical practice, have made an effort to treat the entire subject as a medico-dental problem.

THE DOCTOR'S BILL, *by Hugh Cabot, with an introduction by A. Lawrence Lowell.* Columbia University Press, 1935. Price $3.

This is a thoughtful study of the whole subject of medical care and its costs, made by one of the most eminent of American physicians. The conclusions arrived at are that without very radical changes it would be possible to give better medical service to all the population and at the same time increase the remuneration received by the individual physician. The means employed would be a sort of modified health insurance, a payment of the physician by the Government for care of the indigent, and placing of specialists in positions throughout the country where their services may be

needed who are paid by the State. Dr. Cabot does not believe that limitations on the output of physicians in this country is necessary, at least for a generation. All his views on the various problems of medical care are very conservative and of an extremely common sense character.

A TEXTBOOK OF BIOCHEMISTRY, *by Benjamin Harrow, Ph. D., Associate Professor of Chemistry, the College of the City of New York, and by Carl P. Sherwin, M. D.* W. B. Saunders Co., Philadelphia, 797 pages, illustrated. 1935. Price, $6.

The authors were really the editors rather than the authors as the work was prepared by a number of contributors, many of them specialists in particular fields of biochemistry. The book has exceptional value as a reference work and a feature is the remarkably large and complete index. A bibliographic index is also given considerable space.

BEE VENOM THERAPY, *by Bodog F. Beck, M. D.*, 238 pages, D. Appleton-Century Co., New York and London. Price, $5.

This is a study of bee venom, its chemistry, toxicology and finally its use in therapy, particularly in rheumatic ailments. The evidences that it has therapeutic value are not very convincing.

ELECTROTHERAPY AND LIGHT THERAPY, *by Richard Kovacs, M. D., Clinical Professor and Director of Physical Therapy, Polyclinic Medical School and Hospital, New York.* Second edition, enlarged and revised. 696 pages, 263 engravings, and 1 color photograph. Lea and Febiger, Philadelphia, 1935. Price, $7.50.

This is an enlarged and revised edition of a book well known to many physicians. It has several valuable features. First, it outlines not only the value but the limitations of each physical therapeutic measure. No other field of therapeutics stands so in need of "debunking." Furthermore, the value of light and electric therapy when properly used is so great that it stands in no danger but will be benefited by proper criticism. Second, the book is very practical. There is little or no speculative matter. Facts and clearly written directions as to technic make up the text. Third, there is an extensive glossary which constitutes an excellent dictionary of physical therapy.

WHAT YOU SHOULD KNOW ABOUT HEART DISEASE, *by Harold E. B. Pardee, M. D., Assistant Professor of Clinical Medicine, Cornell University.* 127 pages, illustrated. Lea & Febiger, Philadelphia, 1935. Price, $1.50.

This is the second edition of a little volume intended for the layman in order to assist the patient to more intelligently follow the physician's directions. It is a work similar in character to Joslin's well-known book on diabetes. Although concise it is very complete.

THE ROMANCE OF EXPLORATION AND EMERGENCY FIRST-AID FROM STANLEY TO BYRD. Published by Burroughs Wellcome & Co., London and New York.

This is a remarkably interesting little sketch of the medical and first-aid equipment used by such explorers as Stanley, Roosevelt, Hedin, Abruzzi, the Everest expedition, Byrd, and others. Although an advertisement of their own products, it is a real contribution to a certain phase of medical history. The little volume is well written and full of most interesting pictures.

THE KIDNEY IN HEALTH AND DISEASE *in contributions by eminent authorities. Edited by Hilding Berglund, M. D., Stockholm, Sweden, formerly professor of medicine, University of Minnesota, and Grace Medes, Ph. D., research biochemist in the Lankenau Hospital Research Institute, Philadelphia, with the collaboration of G. Carl Huber, M. D., dean of the graduate school, University of Michigan, Warfield T. Longcope, M. D., professor of medicine, the Johns Hopkins University, and Alfred N. Richards, Ph. D., M. D., professor of pharmacology, University of Pennsylvania.* 754 pages, 163 engravings, Lea & Febiger, Philadelphia, 1935. Price, $10.

In the introduction to one his four contributions to this splendid work Volhard states:

> There are few departments of internal medicine in which there has occurred such a change in our viewpoint during the last 20 years along with such a deepening of our knowledge, incomplete though it still is, as in the field of kidney diseases.

Every chapter of this extensive symposium reflects the heights and depths of these changes.

What symptom complexes accompany pale and red hypertension? What are the criteria of recovery from acute diffuse glomerulonephritis? What factors determine the choice of diuretics for clinical use? Why is the hope of being able to distinguish once and for all between normal pressure and an early hypertension a futile one? How do local anaesthesia and section of renal nerves influence glomerular blood flow? Why is renal reserve now seen to be two types—the native and acquired? What is the present-day status of dietary factors influencing the formation of urinary calculi? What is the significance and application of the theory of renal counterbalance? These are but a few of many factors investigated and ably discussed by 41 distinguished collaborators.

For the internist and urologist keen to enhance their powers of diagnosis and skill in therapy; for the general clinician desirous of keeping abreast of current developments applicable in daily practice; and for the physician about to stand examination for appointment or promotion, close association with this book will prove fruitful.

THE DIVISION OF PREVENTIVE MEDICINE

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

TOXIC EFFECTS OF ARSENICAL COMPOUNDS AS ADMINISTERED IN THE UNITED STATES NAVY IN 1934 WITH SPECIAL REFERENCE TO ARSENICAL DERMATITIS

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For the past 10 years medical officers of the Navy have been required to submit monthly reports to the Bureau of Medicine and Surgery of the number of doses of arsenicals administered and the reactions therefrom. This information, including that for 1933, has been compiled and published in the United States Naval Medical Bulletins of September 1925, January 1927, January 1929, July 1930, October 1931, October 1932, April 1933, October 1933, October 1934, and January 1935.

In table 1 are shown the number of doses of each arsenical administered in the year 1934, the reactions which occurred, and similar data for the 10-year period 1925–34. It is noted that in 1934 there was 1 reaction to 995 doses and 1 death to 42,129 doses. For the 10-year period 1925–34, there was 1 reaction to 1,305 doses and 1 death to 26,129 doses.

Table 1.—*Arsenicals, U. S. Navy, 1934 and 1925–34—Type of drug, reactions, and ratio of doses to reactions*

	Number of doses administered	Reactions				Ratio of reactions to doses, 1 to—	Ratio of deaths to doses, 1 to—
		Mild	Severe	Fatal	Total		
Year 1934:							
Acetarsone	26	0	0	0	0	0	0
Arsphenamine	2,865	0	0	0	0	0	0
Bismarsen	174	0	0	0	0	0	0
Neoarsphenamine	117,799	80	34	3	117	1,007	39,266
Silver arsphenamine	3	0	0	0	0	0	0
Sulpharsphenamine	2,541	8	2	0	10	254	0
Tryparsamide	2,980	0	0	0	0	0	0
Total	126,388	88	36	3	127	995	42,129
10-year period 1925–34:							
Acetarsone [1]	805	0	0	0	0	0	0
Arsphenamine	33,527	25	13	1	39	860	33,527
Bismarsen [2]	316	0	0	0	0	0	0
Neoarsphenamine	880,490	425	221	36	682	1,291	24,458
Silver arsphenamine [3]	227	0	1	0	1	227	0
Sulpharsphenamine	17,197	13	5	0	18	955	0
Tryparsamide	34,205	1	0	0	1	34,205	0
Total	966,767	464	240	37	741	1,305	26,129

[1] First administered during the year 1932.
[2] First administered during the year 1929.
[3] First administered during the year 1931.

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TABLE 2.—*Arsenical Reactions, U. S. Navy, 1934*

Classification	Cases	Deaths
Vasomotor phenomena	48	0
Arsenical dermatitis	43	1
Table reactions	15	0
Blood dyscrasias	8	1
Jarisch-Herxheimer	5	0
Liver damage (jaundice)	3	0
Gastrointestinal	3	0
Arsenical neuritis	1	0
Hemorrhagic encephalitis	1	1
Total	127	3

From table 2 it may be seen that the most frequent reactions are those that are classified as vasomotor phenomena and as arsenical dermatitis. Dermatitis in some form was observed in 33.07 percent of the cases. In 1933, dermatitis cases were observed in 34.74 percent of the cases.

TABLE 3.—*Proportion of reactions of various types, 1929–34*

Classification	Number of reactions	Percent of total reactions
Vasomotor phenomena	238	47.13
Arsenical dermatitis	161	31.88
Table reactions [1]	26	5.15
Blood dyscrasias	19	3.76
Reactions of minor importance	17	3.36
Liver damage	15	2.97
Jarisch-Herxheimer	12	2.38
Acute liver damage	6	1.19
Hemorrhagic encephalitis	4	.79
Gastrointestinal	3	.59
Border-line, hemorrhagic encephalitis	1	.20
Liver damage (doubtful reaction)	1	.20
Arsenical neuritis	2	.40
Total	505	100.00

[1] First classified during the year 1933.

In this article will appear a brief summary of the clinical history of each of the 42 cases of arsenical dermatitis which recovered and a detailed report of the fatal case.

With reference to the significance of certain skin manifestations and to treatment, both abortive and curative, attention is invited to the United States Naval Medical Bulletin of October 1934, pages 559 to 564.

ARSENICAL DERMATITIS

The 43 cases of arsenical dermatitis reported in 1934 were classified as 22 mild, 20 severe, and 1 fatal. The type of lesion was exfoliative in 15 instances, urticaria in 12, macular in 7, maculo-papular in 6, erythematous in 2, and 1 mild rash, type not stated.

MILD REACTIONS

The reactions occurred in 7 instances during the first course of treatment, 5 in the second course, 4 in the third course, 5 in the fourth course, and 1 in the seventh course. Of the 7 reactions during the first course, none followed the first injection.

The interval between the injection and appearance of symptoms varied from 5 minutes to 5 days.

The length of time required for recovery varied from 1 hour to 8 days.

A brief history of each case is cited.

(68—1934) This case is reported under severe reactions (68, 69—1934), as the individual experienced two reactions, the first of which was mild and the second severe.

(85–1934) A patient, who developed mucous patches on the tonsils and generalized adenopathy, was infected in December 1930. A Kahn blood test was 4 plus on March 6, 1931. From March 6 to April 3, 1931, he received 4 injections of sulpharsphenamine, total dosage 1.6 grams. From April 19 to July 6, 1931, he received 6 injections of neoarsphenamine, total dosage 3 grams. From September 17 to December 3, 1931 he received 9 injections of neoarsphenamine, total dosage 4.8 grams. From February 27 to April 31, 1933, he received 8 injections of bismuth salicylate, 0.13 gram each.

On February 17, 1934, he began his fourth course of arsenical treatment with a 0.3 gram intravenous injection of neoarsphenamine. On February 24, he received 0.6 gram, and on March 3, 0.3 gram.

On March 5, an urticarial rash developed on the chest, abdomen, back, face, and upper thighs, with swelling of the hands and face. He responded rapidly to treatment and was returned to duty in 2 days.

(86—1934) A patient developed a chancre on the prepuce, the exudate from which was positive for Treponema pallidum on February 1, 1933. He had generalized adenopathy and on February 6, a blood Kahn was 4 plus. From February 1, 1933 to November 15, 1933, he received 25 injections of neoarsphenamine for a total dosage of 13.8 grams. During the same period he also received 16 injections of bismuth and 20 mercury inunctions. From November 21, 1933 to February 7, 1934, he received 12 injections of bismosol.

On April 11, 1934, he was given a 0.3 gram intravenous injection of neoarsphenamine. Approximately 8 hours after the injection, while bathing, he noted a generalized macular rash which covered his chest, abdomen, and upper arms. No pruritus was noted. Within 24 hours the rash began to fade and had disappeared at the end of 5 days.

(87—1934) A patient had an ulcer on his penis on October 1, 1923. He developed general glandular enlargement and on December 12 and December 28, 1923, blood Wassermann tests were 4 plus.

During the year 1924 he received 6 injections of salvarsan (type not stated), 8 injections of mercury, and an unstated number of mercury inunctions. From December 4, 1927 to September 6, 1928, he received 8 injections of neoarsphenamine, and an unstated amount of protiodide of mercury. On December 13, 1933, he was given a 0.3 gram injection of neoarsphenamine. On January 10, 1934, the patient received 0.3 gram of neoarsphenamine and on January 17, 24, 31, February 7 and 21, doses of 0.6 gram each, a total dosage of 3.3 grams.

Approximately 8½ hours after the last injection the patient reported to the sick bay with a macular rash in the fold of his elbows, on his chest, abdomen, and inner sides of thighs. There was no pruritus and the patient offered no complaint. The next day there was marked fading of the rash, and within 60 hours it had completely disappeared.

(88—1934) On November 3, 1933, this patient presented himself for treatment because of a penile lesion. On darkfield examination Treponema pallidum were found. Neoarsphenamine was administered as follows: November 3, 0.3 gram; November 14, 0.3 gram; November 21, 0.45 gram; December 5, 0.3 gram; December 12, 0.45 gram; December 19, 0.45 gram; January 9, 1934, 0.3 gram; January 16, 0.45 gram; and January 23, 0.45 gram. A total of 3.45 grams was administered in a period of 82 days. As concurrent treatment he was given intramuscular injections of bismosol of 1 cubic centimeter each on November 4, 9, 16, 23, December 7, 14, 21, and January 11, 1934.

Within 5 minutes after the last injection of neoarsphenamine the patient developed urticaria of the face and neck; his eyelids were swollen and partially closed; he complained of a smothering sensation, moderate cramps in the epigastrium; and vomited twice.

The cutaneous and respiratory symptoms cleared up in about 4 hours and the patient recovered in 2 days.

(89—1934) A patient was infected in October 1932. A blood Kahn test was 4 plus and a darkfield was positive for Treponema pallidum.

From October 28 to December 8, 1932 he was given 7 injections of neoarsphenamine, a total dosage of 4.05 grams. From May 10 to June 20, 1933, he received seven injections of neoarsphenamine, a total dosage of 4.05 grams.

On January 16, 1934, the patient received 0.45 gram of neoarsphenamine and on January 23 and January 30, 0.6 gram, a total dosage of 1.65 grams, or an average of 110 milligrams per day.

Five hours after the injection of January 30 the patient felt badly and turned in. About 19 hours after the injection he had a bright red macular eruption over his entire body and conjunctival injection.

The rash slowly faded and in 8 days he was returned to duty.

(90—1934) A patient who was infected in September 1929 was given a diagnosis of syphilis on October 14, 1929, because of a genital sore, a luetic rash, and a 4 plus Kahn blood test.

From October 15 to December 5, 1929, he received 8 injections of neoarsphenamine and 9 injections of mercury. Following this treatment the patient developed a severe arsenical dermatitis which resulted in 73 sick days.

Treatment was resumed in 1933 and from February 5 to May 1, 1933, he received 600 cubic centimeters of mixed treatment. From September 4, 1933, to August 15, 1934, he received 38 injections of bismuth salicylate.

On August 15, 1934, the patient received 0.025 gram of neoarsphenamine. Two days later he developed a generalized punctate, itching, rash with edema of eyelids and genitalia. He had secondary anemia, malaise, and a temperature of 101° F. Duty in 7 days.

(91, 92—1934) Two reactions occurred in the same individual. This patient developed a small indurated penile ulcer on December 24, 1932, with inguinal adenopathy. A darkfield examination was positive for Treponema pallidum and a Kahn blood test was 3 plus on December 27.

From December 27, 1932 to August 10, 1933, the patient received 37 injections of neoarsphenamine in 3 courses, for a total dosage of 16.2 grams. From February 23 to December 11, 1933, he received 35 injections of bismosol, 0.1 gram each.

On December 21, 1933, he was given 0.3 gram of neoarsphenamine and on January 11, 1934, 0.45 gram. Within 5 minutes after the latter injection he became dyspnoeic and a short time later had pruritus and facial edema. He was given ephedrine and all symptoms disappeared within 1 hour.

On January 15 treatment was resumed and he was given neoarsphenamine as follows: 0.045 gram in 1 minute and 50 minutes later, 0.405 gram in 4 minutes. In 10 minutes after completion of the injection there was urticaria. He was given ephedrine sulphate but the urticarial lesions continued to appear on his arms and face. He was then given epinephrine hydrochloride and within a few minutes the general itching disappeared and the urticarial lesions became red and began to grow smaller. Within 24 hours all symptoms had disappeared.

(93—1934) A patient who was infected January 6, 1933, developed a hard chancre on his penis, a darkfield from which was positive for Treponema pallidum. On February 9 a Kahn blood test was 3 plus.

From January 24 to August 22, 1933, he received 8 injections of neoarsphenamine, total dosage 3.3 grams; 14 injections of bismosol, 1 cubic centimeter each; and 27 mercury inunctions.

Following an injection of neoarsphenamine on July 11, 1933, he experienced a mild vasomotor reaction of 1 day's duration.

On January 2, 1934, 0.3 gram of neoarsphenamine was given and on January 16 and February 13, 0.6-gram injections.

One and one-half hours after the last injection he developed a temperature of 101.5° F., nausea, chills, irregular raised red areas on flexor surfaces of arms and forearms, and a slight flushing of the neck, chest, and shoulders. Recovery in 24 hours.

(94—1934) A patient who was infected in April 1934, was admitted to the sick list with syphilis on July 20, because of general adenopathy and positive Kahn blood tests on July 9, on July 16, and on July 20.

On July 20 the patient was given 0.3 gram of neoarsphenamine, 0.3 gram on July 26, and 0.6 gram on August 1.

Three hours after the last injection the patient reported to the sick bay with a maculo-papular rash over his body which disappeared in 3 days.

(95, 96, 97—1934) Three reactions occurred in the same individual. This patient had a painless penile sore about November 15, 1929. He received no specific treatment and the lesion healed spontaneously without knowledge of any secondary manifestations. While in the hospital because of an automobile accident he was found, on January 30, 1934, to have a 4 plus Kahn blood test. On February 5, 1934, a recheck Kahn blood test was also 4 plus.

From February 7 to April 25, 1934, he received 10 injections of neoarsphenamine for a total dosage of 4.35 grams, and 16 injections of thio-bismol for a total dosage of 3.2 grams.

On September 26 he received 0.45 gram of neoarsphenamine and on October 3, 10, 17, 24, 31, and November 7, he received 0.6-gram injections of neoarsphenamine, an average of 94 milligrams per day. He also received 0.2 gram intramuscular injections of thio-bismol on September 28, October 5, 12, 19, 26, November 2, 9, and 16. One and one-half hours after the last injection of neoarsphenamine an urticarial rash developed on both arms and within an hour had disappeared.

On November 14 he was given a 0.6 gram intravenous injection of neoarsphenamine by Bezredka technic as recommended by Stokes. Following the injection a similar urticarial rash developed and as before the rash disappeared without treatment.

On November 21 he was given 0.6 gram of neoarsphenamine and 15 minutes later he had a sneezing attack. Thirty minutes later an urticarial rash developed over his neck, arms, and forearms. The

rash remained for 1 hour and then disappeared without treatment as in the previous instances. On November 23 a patch test revealed no skin reaction and on November 30 he was considered asymptomatic.

(98—1934) A patient was infected on September 15, 1934. Syphilis was diagnosed as the result of a penile chancre, a positive darkfield, and a positive Kahn blood test.

On October 31 he was given 0.3 gram of neoarsphenamine and on November 3, 0.6 gram. As concurrent treatment he was given 0.2 gram intramuscular injections of thio-bismol on October 30 and November 5.

Five days after the last injection of neoarsphenamine the patient developed a general macular rash with areas of confluence but without itching or vesiculation. He had no fever or subjective symptoms. The rash disappeared in 4 days.

(99—1934) A patient who was exposed October 15, 1932 developed a primary lesion on the ventral surface of the distal third of the shaft of his penis on November 1, 1932. A darkfield examination on November 17 was positive for Treponema pallidum.

From November 17, 1932 to April 30, 1934 he received 16 injections of neoarsphenamine for a total dosage of 7.54 grams, and 35 injections of bismuth.

The patient received 0.227 gram injections of neoarsphenamine on July 28 and August 25, 1934.

About 10 minutes after the latter injection the patient noticed a slight erythematous rash over his entire body, with generalized itching, but did not complain of other symptoms of discomfort. In about an hour the itching subsided and in 3 hours the rash disappeared entirely.

(100—1934) A patient developed a primary lesion on his penis and on his upper lip. A darkfield examination of the lesion on his penis was positive for Treponema pallidum on July 28, 1933, and a Kahn blood test was 4 plus.

From July 28 to September 20, 1933, he received 11 injections of neoarsphenamine for a total dosage of 6.6 grams. From September 26 to November 28, 1933, he received ten 1-cubic-centimeter intramuscular injections of bismosol.

On December 2, 1933, he received 0.3 gram of neoarsphenamine, and on December 27 and January 3, 1934, 0.6 gram.

Five minutes after the last injection the patient developed nausea, vomiting, and edema of the lips. In 5½ hours the edema subsided and the patient was returned to duty within 24 hours free from all symptoms.

(101—1934) A patient developed a penile lesion on June 24, 1931. Darkfield examinations and Kahn blood tests were negative at that

time. On August 24 and September 14, 1931, Kahn blood tests were 4 plus. On August 24, treatment was instituted and between that date and September 3, 1931, the patient received 3 injections of neoarsphenamine and 4 injections of bismuth salicylate. Four hours after the injection of September 3, the patient developed a mild vasomotor reaction which lasted 4 days.

On September 9, 1931, treatment was resumed and the patient received 5 injections of bismoid and daily mercury inunctions between that date and October 3, 1931.

From May 17, 1933 to March 7, 1934, he received 16 injections of neoarsphenamine and 26 injections of bismosol.

On March 13, 1934, he was given 0.3 gram of neoarsphenamine, on March 20, 0.45 gram, and on March 28, April 3, and April 12, 0.6 gram.

Twenty minutes after the last injection the patient developed marked edema of the face, ears, lips, and eyes and large urticarial wheals on the chest, back, abdomen, and upper extremities. Recovery in 20 hours.

(102—1934) A patient who was exposed October 4, 1934, developed a small superficial ulceration, one-fourth centimeter in diameter, in the coronal sulcus on November 6, 1934. On November 7, a darkfield examination was positive for Treponema pallidum, and on November 10 a Kahn blood test was negative.

On November 7 and 10 the patient was given 0.3-gram injections of neoarsphenamine and on November 14, a 0.45-gram injection. Following the injection of November 7 the patient had a slight headache. About 4 hours after the injection of November 14 he developed chills, fever, and a backache. The next day a faint maculo-papular rash appeared on his face and body. The dorsum of both hands were slightly raised and nearly covered with a blotchy erythema. The palms of both hands and the soles of both feet were covered with a bright red erythema without edema. Recovery in 6 days.

(103, 104—1934) Two reactions occurred in the same individual. A patient who was infected on September 8, 1934, was given a diagnosis of syphilis because of an intra-urethral lesion, positive darkfield examinations, general adenopathy, and positive Kahn and Wassermann blood tests.

Treatment.—October 2, 0.3 gram of neoarsphenamine; October 3, 0.13 gram bismuth salicylate; October 9, 0.45 gram neoarsphenamine; and October 10, 0.13 gram bismuth salicylate. Three days after the last injection of neoarsphenamine, the patient had a chill which was followed by fever of 101° F., sore throat, headache, general malaise, and a generalized rash. In 4 days the patient was considered fully recovered.

On October 24 the patient received a 0.13-gram intramuscular injection of bismuth salicylate and on October 30 he was given a 0.1-gram intravenous injection of neoarsphenamine as a test dose. About 4 hours later a marked erythema developed which was clinically similar to the rash noted in the previous reaction. The rash disappeared in 2 days.

The patient was given 0.13-gram intramuscular injections of bismuth salicylate on November 6, 9, 12, 16, 19, 23, and 27, without any apparent ill effects.

(105—1934) A patient was admitted to the sick list on September 25, 1934, with a diagnosis of chancroid of the frenum and glans penis. It became phagedenic with partial destruction of glans and complete destruction of the frenum, exposing the fibrous coat of the urethra. It failed to respond to Rosenwald's treatment, so neoarsphenamine was administered as follows: 0.45 gram on October 3, 8, and 9, an average of 193 milligrams per day. In the evening of October 11 the patient noticed a macular rash on the trunk and extremities. Recovery in 4 days.

SEVERE REACTIONS

The 20 severe reactions occurred in 10 instances during the first course of treatment, in 2 during the second, in 3 during the third, in 3 during the fourth, in 1 during the fifth, and in 1 during the sixth.

The interval between the injection and appearance of symptoms was quite variable. In 4 instances skin reactions were noted in about 2 hours after the injection. The longest interval noted was 26 days.

It appears difficult to predict the duration of an attack of severe dermatitis. Of the 20 cases, 5 recovered in less than a month, 6 recovered in less than 2 months, 3 recovered in less than 3 months, 4 recovered in less than 4 months, while in 2 cases the time of recovery was uncertain due either to flare up of symptoms or concurrent diseases.

(65—1934) A patient had a penile lesion in May 1932, and on August 4, 1932, a Kahn blood test was 4 plus. From August 2, 1932, to January 8, 1934, he received 24 injections of neoarsphenamine.

From June 24 to September 12, 1934, he was given 10 injections of neoarsphenamine, total dosage 4.35 grams and 10 injections of bismosol.

On October 8, 1934, 26 days after the last injection of neoarsphenamine, the patient reported to the sick bay complaining of coryza, photophobia, and lacrimation. There was a pruritic macular rash resembling measles on his face and chest. His face became

edematous and later the skin exfoliated. By December 1 the exfoliation had practically stopped, leaving a residual of faint redness of the skin. He was invalided from the service on January 31, 1935, after 115 days on the sick list.

This case is probably a delayed dermatitis although the onset did not point directly to the arsenic as the cause. To be considered is the free period of 26 days which existed between the injection and the onset of symptoms. Also to be considered is the fact that the reaction began in an acute form with coryza, lacrimation, photophobia and a maculo-papular rash. It was the opinion of a board of medical survey that alcoholism may have been a factor in the etiology of the skin reaction as the patient was known to be an excessive drinker.

(66—1934) A patient was given a diagnosis of syphilis because of a penile chancre, inguinal adenopathy, and a 4-plus Kahn blood test.

On October 26 he was given a 0.25 gram intravenous injection of neoarsphenamine and on October 30, a 0.6 gram injection. On November 1 he was given a 0.2 gram injection of thio-bismol.

Three days after the last injection of neoarsphenamine the patient complained of a "cold." His eyes and pharynx were red and there was pus in his nasopharynx. He had acute purulent balanitis, cellulitis of the foreskin, and his left inguinal lymph nodes were hot and tender. The next day he had a chill and his temperature rose to 105.2° F. The following day a few macules appeared over both patellae, about both ankles, and on the flexor surface of the left elbow. The macules persisted with slight itching of the skin over the patellae.

After 16 days the patient had about regained his weight and all symptoms had disappeared.

(67—1934) A patient was given a diagnosis of syphilis on February 15, 1922, as darkfield examinations of the lesions were positive for Treponema pallidum. From February 15 to June 18, 1922, he was given 8 injections of an arsenical, type and dosage not stated.

After an interval of 10 years, treatment was resumed and between September 6 and November 6, 1932, he received 8 injections of an arsenical, type and dosage not stated.

A third course of arsenical treatment, consisting of 7 injections, was given between June 22 and September 5, 1933.

On January 16, 1934, a 0.3 gram injection of neoarsphenamine was given. Six hours and forty-five minutes later the patient complained of pain in his epigastrium and a severe headache. Large and small macules of a bright red color developed on his knees and forearms, the macules tending to coalesce. The rash gradually faded and by

January 30 the large blotches had completely disappeared. Recovery in 24 days.

(68, 69—1934) Two reactions (one mild and one severe) occurred in the same individual. A patient developed a sore on the shaft of his penis after exposure in December 1930. A Kahn blood test was 2 plus on January 21, 1931.

From January 21, 1931 to May 22, 1934, the patient received 58 injections of neoarsphenamine for a total dosage of 28.95 grams, and 20 injections of mercury of one-sixth grain each.

On June 19, 1934, the patient received 0.3 gram of neoarsphenamine and on June 26, 0.45 gram. In the evening following the latter injection he had considerable itching of the skin and a mild rash which faded in about 48 hours. The patient did not report these symptoms at the time.

On July 3 he was given 0.6 gram of neoarsphenamine. Approximately 10 hours later there was itching of the skin and a dry, itching, macular, and scaling rash which eventually involved the whole area of the trunk and extremities. To duty under treatment in 30 days.

(70—1934) A patient was infected on November 29, 1933, and on December 3 a darkfield examination of the primary lesion was positive for Treponema pallidum.

The patient was given 0.45 gram of neoarsphenamine on December 15 and 0.6 gram on December 22, 29, January 6, 13, 19, 26, and February 2, an average of 93 milligrams per day. He was also given 0.1 gram intramuscular injections of bismuth salicylate on December 4, 12, 19, 26, January 2, 9, 16, 23, 30, February 6, and 13.

About 6 hours after the last injection of neoarsphenamine he developed dermatitis of both arms and ankles with edema of the ankles and pruritus. In 4 days the dermatitis had extended to the back and chest and in 14 days the skin of the hands and feet began to exfoliate. The dermatitis gradually improved and after 52 days on the sick list he was discharged to duty much improved.

On July 26 he was readmitted to the sick list with what was apparently a mild recurrence as he had received no arsenical treatment since the original admission. There was an itching, light red eruption over his entire body, without swelling of the feet or legs. The eruption entirely disappeared in 6 days.

(71—1934) A patient developed a chancre on his upper lip with a positive darkfield on April 1, 1932; a Kahn blood test was 4 plus on April 6; and secondaries appeared on April 17.

From April 12 to June 2, 1932, he received 8 injections of neoarsphenamine, total dosage 4.05 grams, and 8 injections of bismuth.

From July 20 to September 29, 1932, he received 10 injections of neoarsphenamine, total dosage 6.0 grams, and 11 injections of bis-

mosol, 0.1 gram each. About 3 weeks prior to the last injection of neoarsphenamine the patient noted a pruritic rash on his face and arms. For this he was given a mild ointment of salicylic acid, resorcin, thymol, and sulphur. The lesions spread rapidly and involved the face and arms. Both eyelids and both forearms were edematous and on the forearms there were confluent papules and vesicles. His condition gradually improved and on December 2 and 9, 1932, he was given 0.2 gram injections of neoarsphenamine. The dermatitis became worse on the arms and legs. Gradually the dermatitis again cleared and on April 5, 1933, the patient was discharged to duty well.

From July 5 to August 8, 1933, the patient was given six 0.1 gram intramuscular injections of bismosol.

On September 12, 1933, he was given 0.3 gram of neoarsphenamine; on September 19, 0.45 gram; and on September 26, 0.6 gram. On October 1 there was slight reddening and drying of a small area of the skin on the palmar surface of both forearms, which disappeared in a few days.

From October 10 to December 26, 1933, he received twelve 0.1 gram intramuscular injections of bismosol.

On January 9, 1934, he was given 0.3 gram of neoarsphenamine; on January 16 and 23, 0.45 gram injections; and on January 30, 0.6 gram. He was also given 0.1 gram intramuscular injections of bismosol on January 18, 25, and February 1. About 4 or 5 days after the last injection of neoarsphenamine the patient had a mild dermatitis on the flexor surface of the forearms. The condition cleared without treatment. Arsenicals were discontinued but bismuth therapy was continued until April 19, 1934, at which time he had completed 12 injections of 0.1 gram each.

On May 17, 24, and 30, 1934 he received 0.1 gram intramuscular injections of bismosol.

On June 4 the dermatitis again flared up with a slight itching and redness of the forearms. Following the application of resorcinol ointment by the patient his skin became angry, red, and weeping. Examination revealed erythema of the face and neck; edema of the eyes and right side of the face; discrete small vesicles over the entire face, neck, shoulders, and back of both forearms and hands. The vesicles exuded a thin, clear, slightly yellowish serum, which tended to crust. He gradually improved and on August 13, 1934, he was returned to duty under treatment.

(72–1934) A patient had a penile lesion on December 31, 1933. Diagnosis of syphilis was established by appearance of a secondary rash and generalized adenopathy.

On March 8, 1934, he was given a 0.3 gram intravenous injection of neoarsphenamine with 1 gram of sodium thiosulphate. The

course was continued, the patient receiving a 0.3 gram injection on March 13; 0.6 gram injections on March 20 and 27; and a 0.45 gram injection on April 10. An injection was not given on April 3 because the patient was not feeling well. This was attributed to the typhoid vaccine which he had received on the previous day. As concurrent treatment he was given 1 cubic centimeter injections of bismogenol on March 8 and 27. He did not receive injections of bismogenol on March 13, 20, and April 10 because his urine showed a faint trace of albumin.

On April 9 the patient received a third injection of a course of typhoid vaccine.

On April 11, 24 hours after the last injection of neoarsphenamine, the patient developed a morbilliform rash which lasted about 2 days.

On April 16 the patient received the last injection of a course of gonococcus vaccine.

On April 20 the patient again developed a morbilliform rash and 2 days later edema of the face and extremities appeared. On April 28 he had petechiae on the lower extremities. Exfoliation of the entire body began on April 30. He was considered recovered in 30 days but was retained on the sick list under observation for an additional 35 days.

(73—1934) A patient who had no evidence of a primary lesion had a 4-plus Kahn blood test on February 26, 1934. A recheck was made and on March 12 a Kahn blood test was again 4 plus.

On March 13 he was given a 0.3 gram injection of neoarsphenamine and on March 20, 23, 27, April 4, 10, and 17, 0.6 gram injections, an average of 108 milligrams per day. He was also given 1 cubic centimeter injections, 2 grains each, of bismuth salicylate on March 16, 20, 23, 27, April 4, 6, and 13.

Approximately 24 hours after the last injection of neoarsphenamine the patient developed a maculo-papular rash over the lower trunk and extensor surfaces of the thighs with edema of the feet and ankles. Recovery in 37 days.

(74—1934) A patient developed ulcers on his penis on August 15, 1931, and on September 10 a 4 plus Kahn blood test.

From November 20, 1931 to September 12, 1932, the patient received 17 injections of neoarsphenamine and 30 injections of bismosol.

On September 27, 1934, he was given 0.3 gram of neoarsphenamine; on October 4, 0.45 gram; and on October 11, 0.3 gram. In the afternoon of October 11 the patient developed an itching, scaling rash of the face, neck, and chest which partially subsided after 4 or 5 days, leaving in its place a fine rash which was confined

to the face and neck. Neoarsphenamine treatment was discontinued but bismuth therapy was started on September 27 and continued until November 9, by which time he had received 8 injections of bismosol of 0.1 gram each.

The patient developed a mild attack of pleurisy and upon removal of the adhesive strapping from his chest the fine rash spread from the face and neck to the chest. This rash developed into a maculo-papular dermatitis which was confluent in areas over the face, neck, upper thorax, shoulders, and flexures of the elbows. The rash was scaly and pruritic. Under treatment the rash and itching subsided. Recovery in 64 days.

(75—1934) A patient who was exposed January 5, 1934, developed patches in his mouth, body rash, general glandular enlargement and a 4 plus Kahn blood test.

At time of reaction the patient was on his first course of neoarsphenamine treatment which was administered as follows: April 7, 0.3 gram; April 9, 0.4 gram; April 11, 0.5 gram; April 13, 16, 19, 25, and May 2, 0.6 gram injections, an average of 162 milligrams per day. On April 27 and May 4 he was given 0.13 gram intramuscular injections of bismuth salicylate.

Five days after the last injection of neoarsphenamine the patient developed a burning and itching rash on his trunk which he did not report.

On May 9 he was given 0.6 gram of neoarsphenamine and following this injection he reported that he had the burning and itching rash over his body the day before the injection. He was placed on the sick list and in the following days there was a gradual increase in the intensity of the skin condition. He also developed an ulcerative stomatitis, neuritis of both feet, and later developed neuritis of arms and hands. The skin condition became generalized and began to desquamate. To duty in 106 days.

(76—1934) A patient was given a diagnosis of syphilis because of scars on his penis, right inguinal bubo, generalized glandular adenopathy, and a 4 plus Kahn blood test. He was infected on December 15, 1933.

He was given a 0.3 gram intravenous injection of neoarsphenamine on February 3, 1934, 0.45 gram injections on February 8, 13, 20, 27, March 6, and 13 and a 0.5 gram injection on March 19. On March 2, 9, and 16 he was given 2 grain intramuscular injections of bismuth salicylate.

Two hours after the last injection of neoarsphenamine the patient noticed a minute papular rash on his hands and arms. In about 4 days the rash had spread to all parts of his body surface. The rash itched and was worse when he was hot. Three days after

the onset the rash began to exfoliate. The patient developed purulent conjunctivitis. Gradually the conjunctivitis and exfoliation of the skin subsided.

On May 15, weekly intramuscular injections of bismuth salicylate 2 grains each, were started.

By June 29, 1934, 102 days after the onset of the first symptoms, the patient was considered recovered.

(77—1934) This patient developed a penile sore on December 7, 1932 and a secondary rash, generalized adenopathy, and a 4 plus Kahn blood test on March 17, 1933.

From March 18, 1933 to February 19, 1934 he was given an unstated number of injections of neoarsphenamine for a total dosage of 11.25 grams; 20 injections of bismuth salicylate, 0.13 gram each; and 10 injections of mercury succinimide, 0.026 gram each.

On March 2, 1934 he received 0.3 gram of neoarsphenamine, on March 9, 0.45 gram, and on March 23 and 30, 0.5 gram injections.

Three days later the patient developed a pruritic macular rash on his body, arms, and legs. The rash steadily increased in severity and desquamation developed. The condition gradually improved and on June 4, 1934 he was discharged to duty under treatment after 63 days on the sick list.

(78—1934) This patient was infected on December 26, 1932.

From January 24 to October 5, 1933 he received 16 injections of neoarsphenamine.

On August 9, 1934, he was given 0.3 gram of neoarsphenamine and on August 14 and 21, 0.45 gram. On August 6, 13, and 20 he received 0.1 gram intramuscular injections of bismosol.

Six days after the last injection of neoarsphenamine the patient noted a few sores developing on the forearms and legs. The condition became progressively worse with generalized distribution of lesions over the trunk, extremities, face, and neck. The lesions appeared as papules, became vesicular, and then ulcerated. In some cases they resembled impetiginous lesions. Recovery in 39 days.

(79—1934) A patient was infected in December 1932 and on January 11, 1933, had macular syphilis with mucous patches and a 3 plus Kahn blood test.

From January 11 to September 25, 1933 he received 7 injections of neoarsphenamine, total dosage 3.9 grams; 15 injections of bismosol, 1 cubic centimeter each; potassium iodide, 5 to 15 drops for 1 month; and mixed treatment, t. i. d., for 6 months. On February 28, 1933, 3 days after the last injection of neoarsphenamine, the patient developed a pruritic maculo-papular eruption which increased in intensity and finally exfoliated. The patient recovered in 86 days.

On March 5, 1934, he was given a 0.225 gram intravenous injection of neoarsphenamine as a test dose. Eight days later he had an attack of vertigo, nausea, vomiting, headache, temperature, 102° F., a high pulse rate, and erythema of skin with "goose flesh" appearance. The skin was dry with fine branny scaliness. After desquamation the skin cleared nicely with only a tendency to dryness after bathing. Recovery in 56 days.

(80—1934) A patient developed a chancre on his glans penis, a darkfield examination of which was positive for Treponema pallidum on November 17, 1932.

From November 17 to December 3, 1932, he received 6 injections of neoarsphenamine, total dosage 3.15 grams, and 7 injections of bismosol, 1 cubic centimeter each. Following this course of treatment the patient developed a severe exfoliative dermatitis. Recovery in 53 days.

From July 7, 1933, to June 26, 1934, he received 36 injections of bismuth and 32 days of specific mixture, 1 fluid dram t. i. d.

On June 27, 1934, he was given 0.3 gram of neoarsphenamine and in 12 hours developed pruritus of his hands. The next morning he had a scarlatiniform erythema of neck, axillae, forearms, and groins. On the fifth day the erythema subsided and was followed by a flaky desquamation of the involved areas. Recovery in 22 days.

(81—1934) A patient who was exposed November 1, 1933, developed an indurated lesion on the penis on November 27. On November 29 a darkfield examination was positive for Treponema pallidum and on December 4 a Kahn blood test was 4 plus.

From November 28, 1933, to June 5, 1934, he received 15 injections of neoarsphenamine and 14 injections of bismosol.

On July 2, 1934, he was given a 0.25 gram injection of neoarsphenamine and on July 9, 16, 23, 30, August 13, 20, and 27, 0.45 gram injections. On September 4 he reported for a ninth injection which was not administered because the patient stated that he had mild reactions after each of the 3 or 4 previous injections. He stated that about 2 hours after each injection dark spots appeared on his hands and legs which itched and burned. He also had frontal headaches, swollen eyelids, and bleeding gums for several days. The skin lesions were deep seated and consisted of brown pigmented areas of various sizes and shapes, distributed over the finger tips, wrists, buttocks, and legs. On September 6 there was a papular eruption on the palms of both hands and a discrete macular eruption on both forearms. He also had bluish colored macular spots on the lips, chin, buttocks, penis, and both legs with marked gingivitis and a fetid breath. During the past 6 months he had had eruptions on his body and legs. By September 14 he had no headaches and the

rash was fading slowly. On October 4 he was considered recovered from the reaction.

(82—1934) A patient was admitted to the sick list on May 18, 1934, because of a small scab on the glans penis, adenopathy, and a 4 plus Kahn blood test. The patient denied exposure since August 6, 1933. A Kahn blood test was again 4 plus on June 4.

On May 10, 1934, he received a 0.3 gram injection of neoarsphenamine, on May 17, 0.45 gram, and on May 24 and June 1, 0.6-gram injections.

On June 2 he was admitted to the sick list with an abscess, left sacro-iliac joint.

On June 12 he had a maculo-papular rash on the back of the hands, anterior surface of forearms, anticubital spaces, and back of the neck. The next day the rash extended to the chest and abdomen with the rash on the neck becoming vesicular. The skin then became indurated, reddened, and scaly. In 27 days after onset of symptoms he was considered recovered from the reaction.

His diagnosis was then changed to scabies and later to tuberculosis, left sacro-iliac joint and he was eventually invalided from the naval service after a total of 202 sick days.

(83—1934) A patient was infected January 5, 1934. A darkfield examination of the primary lesion was positive for Treponema pallidum. A Kahn blood test was negative.

On January 30, 1934, he was given 0.3 gram of neoarsphenamine, on February 3, 0.45 gram, and on February 8, 15, 22, March 1, 8, and 15, 0.6-gram injections, an average of 97 milligrams per day.

On the day of the last injection the patient noticed a few small papules on the left arm which burned. A week later the rash spread to both arms and forearms. He was treated with sulphur ointment locally and the rash spread over nearly the entire body. Over almost the entire body surface the skin was thickened, red, and showed miliary papules and vesicles which exuded a thin serum. There was intense pruritus and later desquamation. Recovery in 78 days.

(84—1934) A supernumerary patient (female) developed syphilis in January 1934.

On February 2, 1934, she was given 0.3 gram of neoarsphenamine and on February 8, 15, 22, and March 1, 0.45-gram injections, an average of 75 milligrams per day. She was also given 1 cubic centimeter injections of bismosol on February 6, 13, 20, and 27.

Eighteen hours after the last injection of neoarsphenamine she developed a dermatitis over the trunk and extremities which was accompanied by mild pruritus. The skin and sclerae showed mild icterus. There was slight exfoliation which disappeared in 14 days.

2605—35——10

(115—1934) Two reactions occurred in the same individual, the first of which resulted in liver damage (jaundice) and the second in arsenical dermatitis.

A patient who became infected with syphilis developed a positive darkfield on June 22, 1934, and a positive Kahn blood test on July 12.

On June 28 he was given 0.45 gram of neoarsphenamine and on July 12 and 19, 0.6 gram. He was also given 1 cubic centimeter injections of bismosol on June 29, July 3, 6, 10, 13, 17, 20, 24, August 10, 14, 17, 21, and 24.

Two days after the last injection of neoarsphenamine the patient developed jaundice. Recovery was complete in 17 days. (This reaction will be described in more detail in a subsequent article under case no. 114—1934.)

On August 9 he was given 0.3 gram of neoarsphenamine after which he felt feverish, had loss of appetite, itching of the skin, and a recurrence of jaundice. He was given sodium thiosulphate which appeared to relieve the condition. Later he noticed several small white blisters on the skin which lasted about 4 days. On August 28 he was admitted to the sick list complaining of swelling under the lower eyelids. A scaliness was noted over his right shoulder and the next day there was a fine desquamation over the entire body.

The patient then developed what was an apparent trichophyton infection of the gluteal regions.

The scaling of the skin gradually subsided and on November 5 the skin was normal except for fine, browny desquamation about the hips and soles of the feet. On November 10, 15, 19, and 22 he was given 1 cubic centimeter injections of bismosol. On November 27 he returned to duty after 110 days on the sick list.

FATAL REACTION

(64—1934) This patient experienced 3 reactions, 2 of which were of a mild vasomotor type and the third a fatal arsenical dermatitis. (The vasomotor reactions will be described in a subsequent article under cases nos. 62, 63—1934.)

The patient contracted syphilis in February 1934, and was on his first course of neoarsphenamine, which was administered as follows: March 12, 0.3 gram; March 20, 27, April 3, 10, and 17, 0.6 gram injections. He was also given nine intramuscular injections of bismuth salicylate, 0.13 gram each, between March 16 and May 1, 1934.

Following the first two injections of neoarsphenamine, the patient was nauseated and became so dizzy he had to lie down for several hours. A chill followed in the evening after each injection. About one day before the sixth injection the patient noticed a slight rash on

the inner surface of the thighs. After the sixth injection he was so dizzy he had to lie down for several hours. The rash then began to spread down the thighs and onto the scrotum. On April 26 he presented himself to the sick bay, where he was treated with sulphur and Whitfield's ointments. The rash continued to spread and appear in other locations. No arsphenamine was administered after the rash was observed by the medical officer. Three injections of bismuth salicylate were given after the rash appeared.

On May 3, 1934, a physical examination was made. Well developed, well nourished, white male, 21 years of age.

Head and neck.—The mucous membranes of the mouth are red, and in places, show minute ulcers. The gums around the teeth are dirty, acutely inflamed, and are exuding pus. The pharynx is acutely inflamed. The eyelids are edematous and small quantities of pus are seen to come from the nasal canthus.

Chest.—Symmetrical, expansion is good with slight dragging on the left side. A few coarse rales are heard over the base of the left chest, posteriorly. No change in fremitus, tactile or vocal. Heart sounds normal. Blood pressure 106/72.

Abdomen.—The liver descends two fingers' breadth below the costal margin. The spleen is palpable.

Skin.—The entire skin surface is red, varying in degree from bright red to a faint flush. On the inner surface of the thighs, on the scrotum, in the axillae, the anticubital spaces, and on the hands the skin is covered with pustules, a number of which have ruptured, giving off a very foul smelling exudate. In places, especially on the hands and fingers, the upper layers of the skin have shed off, leaving raw surfaces. Sudamina is present in all places where the skin surfaces come in contact. For the past 3 or 4 days the patient has had a burning pain in the epigastric region with gaseous eructations and constipation.

Treatment.—Sodium bicarbonate baths; sodium thiosulphate, 1 gram, b. i. d. Boric acid compresses to eyes, q. 3. h. Saline gargle and mouth wash, b. i. d. Boric acid ointment to all body surfaces, b. i. d.

Urinalysis.—Appearance, slightly cloudy, amber; reaction, acid; specific gravity, 1.023; albumin and sugar, negative; casts, rare granular; mucous, fair amount.

May 5: White blood count, 20,000; band forms 16, segmented 65, lymphocytes 9, eosinophiles 9. Secondary infection of skin is clearing. Temperature, septic type, 100° to 102.2° F.

May 7: White blood count, 21,000; myelocytes 1, juveniles 4, band forms 26, segmented 41; lymphocytes 16, eosinophiles 11, monocytes 1. Skin is peeling. Temperature 100° to 101.2° F. Urinalysis:

Appearance, clear, amber; reaction, acid; specific gravity, 1.021; albumin, 1 plus; sugar, negative; leukocytes, occasional; epithelium, large amount with renal cells. X-ray of chest: The lung fields are entirely clear.

May 8: Red blood count, 5,500,000; white blood count, 30,000; hemoglobin 90 percent; coagulation time, 2 minutes and 15 seconds; differential count, juveniles 6, band forms 27, segmented 34, lymphocytes 21, eosinophiles 12.

May 9: White blood count, 19,400; myelocytes 2, juveniles 12, band forms 28, segmented 36, lymphocytes 15, eosinophiles 7, monocytes 2. Urinalysis: Appearance, clear, amber; reaction, acid; specific gravity, 1.025; albumin, positive; sugar, negative; epithelium, scant. Began having hiccoughs about 3 a. m. Complains of acid eructations. Morphine sulphate grains one-eighth given. Intake of fluids 3,700 cubic centimeters; output, 1,500 cubic centimeters.

May 10: Slept very little during the night. Continues to hiccough. Skin continues to peel. Seidlitz powder given with good results. Sodium bicarbonate stops hiccoughs for about ½ to 1 hour. Temperature 100° to 100.2° F. Skin surface over buttocks and shoulders is covered with petechial hemorrhages. Tannic acid, 5 percent, applied. Baths discontinued. Morphine sulphate grains one-eighth.

May 11: Slept most of the night. Hiccough started as soon as he was awake. Skin much improved. Treatment during the day continued. Sodium amytal given at 11:30 p. m.

May 12: Slept very little during the night. Severe hiccough. Squibb's mixture, 2 cubic centimeters given. Hiccough relieved for 1 hour. Carbon dioxide gas 5 percent in oxygen, inhalations for 10 minutes every 2 hours. Luminal grains V. He hiccoughs at irregular periods for 15 to 20 minutes at a time. Sleeps between periods. Subsultus tendinum. Temperature 100° to 102.6° F. Blood cultures negative after 72 hours incubation. Blood chemistry: Urea nitrogen, 15 milligrams per 100 cubic centimeters; sugar, 97 milligrams per 100 cubic centimeters; chlorides 365 milligrams per 100 cubic centimeters.

May 13: No change during the night. Involuntary bowel movement at 12:30 a. m. Urinalysis: Appearance, clear, amber; reaction, acid; specific gravity, 1.021; albumin, 1 plus; sugar, negative; epithelium, large amount; crystals, large amount amorphous urates. Blood counts: Red blood count, 4,470,000; white blood count, 28,200; hemoglobin 83 percent; Differential: myelocytes 7, juveniles 15, band forms 33, segmented 38, lymphocytes 3, eosinophiles 4.

May 14: Temperature 102° F. Irrational. A to-and-fro murmur is heard over the base of the heart. Petechial hemorrhages are seen on both legs. Intake of fluids, 3,700 cubic centimeters; output, 2,200 cubic centimeters.

May 15: Blood counts: White blood count, 12,200; Differential: Myeloblasts 1, myelocytes 5, juveniles 14, band forms 21, segmented 39, lymphocytes 18, eosinophiles 1, monocytes 1. Temperature 101.6° to 102.2° F. Pulse 92 to 120. Respirations 24. Less restless. Hiccough decreasing. They appear every 2 or 3 hours and last only a few minutes.

May 16: Blood counts: White blood count, 13,100; Differential: Myelocytes 3, meta-myelocytes 7, band forms 17, bilobed polymorphonuclears 35, trilobed polymorphonuclears 14, lymphocytes 1, large lymphocytes 3, small lymphocytes 14, eosinophiles 5, monocytes 1. No change except skin surfaces are much improved.

May 17: Urinalysis: Appearance, clear, amber; reaction, acid; specific gravity, 1.009; albumin and sugar, negative; cylindroids, numerous; leukocytes, occasional; epithelium, large amount. No hiccough. Temperature 99.8° to 101.6° F. Appears to be much improved. Skin over lower portion of legs and feet still show petechiae. Heart murmurs cannot be heard now. Blood counts: White blood count, 21,500; Differential: Myeloblasts 1, myelocytes 4, juveniles 10, band forms 27, segmented—bilobed 33, trilobed 4, lymphocytes 14, eosinophiles 7.

May 18: Blood counts: White blood count, 17,000; Differential: myeloblasts 1, myelocytes 2, meta-myelocytes 9, band forms 12, bilobed polymorphonuclears 41, trilobed polymorphonuclears 25, lymphocytes 8, eosinophiles 1, monocytes 1. Numerous moist rales are heard over the entire chest. Has slight unproductive cough. Subsultus tendinum. Takes food well. Intake of fluids has always been between 3,000 and 4,000 cubic centimeters; output between 2,000 and 2,500 cubic centimeters.

May 19: Urinalysis: Appearance, clear, amber; reaction, acid; specific gravity, 1.003; albumin and sugar, negative; mucus, little; leukocytes, occasional; epithelium, scant. Temperature 100° to 100.8° F. Very restless and delirious. Rales are still present in chest. No areas of consolidation can be found. Heart sounds are now normal.

May 20: Blood counts: White blood count, 23,450; Differential: myeloblasts 1, pre-myeloblasts 1, myelocytes 10, juveniles 14, band forms 17, segmented 47, lymphocytes 10. Complains of difficulty in getting sufficient air. At 9 a. m. had an attack of stertorous breathing which lasted 3 minutes. Head of bed elevated. At 4:40 p. m. breathing again became stertorous. No evidence of tracheal or laryngeal obstruction could be found. Put in oxygen tank but became maniacal and had to be taken out. Unconscious. At 4:30 p. m. had a convulsion which lasted 15 minutes. Breathing was then a little easier. Pulse had been full and strong up to this time, when it

became weak. At 6 p. m. breathing became more difficult. At 6:25 p. m. he died.

Gross pathological findings on autopsy.—(1) Dermatitis, exfoliative; (2) congestion and edema of lungs; (3) dilatation, right heart; (4) congestion and cloudy swelling, liver and kidneys; (5) congestion of spleen; (6) petechial hemorrhages and abscesses of skin; and (7) congestion and edema of brain.

SUMMARY

In 1934, Naval Medical Officers administered 126,388 doses of arsenicals and reported the occurrence of 127 reactions therefrom. Of these reactions, one-third (43) were arsenical dermatitis; a ratio of 1 case of dermatitis to 2,939 doses. Of the 43 cases, 1 died and 42 recovered.

Of interest in connection with the etiology of arsenical dermatitis is the number of instances in which premonitory signs were noted. These signs are repeated below and serve to indicate the necessity for careful examination and questioning of each patient before administering an arsenical.

Case 90.—Exfoliative dermatitis in 1929. Over 4 years later a 0.025-gram injection caused urticaria and edema of eyelids.

Cases 91 and 92.—In this individual urticaria developed after the second and third injections of the fourth course of arsenicals.

Cases 95, 96, and 97.—In this individual urticaria followed the seventh, eighth, and ninth injections.

Case 102.—Slight headache followed second injection and maculopapular dermatitis developed after the third injection.

Cases 103 and 104.—Erythematous dermatitis developed after a second injection and 21 days later a test dose again caused erythematous dermatitis.

Cases 68 and 69.—Unreported itching and mild rash followed the second injection of the seventh course and exfoliative dermatitis followed the third injection administered 7 days later.

Case 71.—In September 1932, 3 weeks after the last injection, dermatitis developed. In December 1932, following 2 injections, the dermatitis was aggravated. In September 1933, after another injection, the skin became reddened with areas of dryness. In January 1934, after a fourth injection, skin on forearms itched and was red. Resorcinal ointment applied by the patient aggravated the condition and exfoliative dermatitis ensued.

Case 72.—The fourth injection of the first course was not administered because the patient did not feel "up to par." Exfoliative dermatitis developed after an injection administered 7 days later.

Case 73.—Marked sunburn on body at time of injection. Exfoliative dermatitis developed.

Case 75.—Unreported urticaria followed the eighth injection and 7 days later the ninth injection caused exfoliative dermatitis and peripheral neuritis of feet.

Case 79.—Maculo-papular dermatitis in 1933 and 1 year later exfoliative dermatitis followed a test dose of 0.225 gram.

Case 80.—Exfoliative dermatitis in 1932. Exfoliative dermatitis followed an injection given 1½ years later.

Case 81.—Unreported mild reactions with dark spots on hands and legs which itched and burned followed the previous 3 or 4 injections. Maculo-papular dermatitis developed after the next injection.

Case 115.—Cholangitis followed a third injection. Two days after recovery from the cholangitis and injection caused recurrence of jaundice and exfoliative dermatitis then developed.

Case 64.—Two mild vasomotor reactions with nausea and dizziness followed by chills occurred after the first and second injections of the first course. Exfoliative dermatitis developed after the sixth injection. The case terminated fatally.

TREATMENT OF GONORRHEA

In a recent lecture on gonorrhea at the United States Naval Medical School, Dr. P. S. Pelouze listed his anticomplication commandments. They are:

1. Use nothing larger than 6-cc syringe for anterior urethritis.
2. Consider posterior stage more serious, restrain patient more than in anterior, and give no local treatment until bladder comfort is regained.
3. Tell the patient that if he does heavy physical exertion with any great quantity of infected urine in his bladder he has at least a 25-percent chance of getting epididymitis. It will not happen if the bladder is empty.
4. Avoid all prostatic manipulation until the first urine is almost clear. Even then consider it as possibly explosive.
5. Pass no instruments whatever into the urethra until there is every reason to think the gonococcus is gone.
6. Give no intravesical irrigations at more than 3 feet of hydrostatic pressure and do not fill the bladder then.
7. Avoid prolonged discharge by slowing up on treatment as patient nears apparent cure. One or two treatments a day is often enough for gonorrhea at its worst.
8. Pay as much attention to hospital corpsman as to patient. A poorly instructed or rough one is a menace.
9. Institute charting of urine trends of every patient and make everyone clinical-course conscious. About the surest way to stimulate pride in results as poor ones are seen at a glance.
10. See that every patient realizes that he gets well because he builds up a temporary immunity, that getting well is his job, medical treatment is just an aid, that alcohol, sexual excitement or intercourse prevent cure and make treatment just so much time wasted. Most people do things better when they

cannot put the blame on others. Make no dietary restrictions but concentrate on those that really count—alcohol, sexual excitement or intercourse, excessive and prolonged physical exertion. Whenever possible put men on easy jobs during the posterior stage.

HEALTH OF THE NAVY

The admission rate, all causes, based on returns for January, February, and March 1935 was 490 per 1,000 per annum. The corresponding median rate for the first quarter, 5-year period 1930–34, is 461 per 1,000. For diseases the admission rate was 428 per 1,000 and the 5-year median, 405. The rates for all causes and diseases showed a decrease when compared with the rate for the previous quarter and an increase when compared with the 5-year median for the first quarter.

The rate for accidental injuries, 62, was 57 percent greater than the 5-year median rate. Destruction of the airship *Macon*, which crashed about 3 miles to seaward of Point Sur on February 12, 1935, resulted in the death of 2 enlisted men by drowning.

Poisonings decreased from 4.28 per 1,000 for the fourth quarter of 1934 to 0.65 per 1,000 for January, February, and March 1935. The 5-year median rate for the quarter is 0.44 per 1,000.

A total of 1,937 cases of the common infections of the respiratory type was reported from all shore stations, 1,421 of which were catarrhal fever, 231 tonsillitis, acute, 89 influenza, and 107 bronchitis.

The following table shows the numbers of admissions for various communicable diseases for the quarter recorded at certain shore stations:

Station	Catarrhal fever	Tonsillitis, acute	Influenza	Bronchitis	Angina (Vincent's infection)	Mumps	Measles and German measles	Chickenpox
Naval Training Station, San Diego, Calif.	160	28	0	66	2	51	238	1
Naval Training Station, Norfolk, Va.	458	77	0	4	1	13	64	1
Marine Corps Base, San Diego, Calif.	40	20	9	0	2	2	44	2
Marine Barracks, Quantico, Va.	120	8	11	1	2	1	0	0
Naval Academy, Annapolis, Md. (midshipmen)	165	8	0	1	1	12	9	1
Naval Academy, Annapolis, Md. (other than midshipmen)	51	4	5	11	0	0	5	0
Marine Detachment, American Legation, Peiping, China	29	0	51	0	25	0	1	0
Regimental Hospital, 4th Marines, Shanghai, China	48	7	3	0	0	4	9	0
Submarine Base, New London, Conn.	37	7	0	1	1	0	0	0
Navy Yard, Portsmouth, N. H.	27	6	0	1	0	0	0	0
Naval Torpedo Station, Newport, R. I.	29	1	0	3	2	0	0	0
Navy Yard, Washington, D. C.	11	6	0	1	0	0	0	1
Naval Station, Guam	2	1	0	1	0	0	0	1

In addition to the above, 1 case of scarlet fever was reported from the Marine Corps Base, San Diego, Calif.; 1 case of lobar pneumonia from the Navy Yard, Washington, D. C., and 2 from the Marine Detachment, American Legation, Peiping, China; 2 cases of broncho-pneumonia from the Marine Barracks, Quantico, Va., 2 from the Naval Training Station, San Diego, Calif., 1 from the Naval Academy, Annapolis, Md., among personnel other than midshipmen, and 1 from the Naval Station, Guam; and 1 case of whooping cough from the Naval Station, Sunnyvale, Mountain View, Calif.

The Naval Training Station, Norfolk, Va., reported that there have been a larger percentage of catarrhal fever cases with slightly aggravated symptoms than previously.

The following is quoted from the monthly sanitary report from the Naval Training Station, San Diego, Calif., for the month of February:

> By inspection of all recruits twice daily for skin eruption many mild cases of German measles without subjective symptoms or rise in temperature have been found.

This station reports 2 cases of scarlet fever, 1 case of lobar pneumonia, and 1 case of broncho-pneumonia hospitalized as undetermined, and 1 case of laryngitis, acute, which was later changed to cerebrospinal fever in hospital.

The medical officer of the Marine Detachment, American Legation, Peiping, China reports under date of March 1 regarding the influenza epidemic which developed at the end of January, reached its peak within 10 days, and rapidly subsided:

> A total of 110 cases was reported from the American colony and an estimated additional 250 foreigners were affected. Fifty-one cases occurred within the Marine Detachment, American Legation, with 3 deaths. All sections of the city reported cases but, as with the guard, the epidemic rapidly spent its force with only slight secondary waves. Peiping has been without rain or snow since December 1934 and it is thought the mild, dry, and dusty winter was an important factor. The cases were typical of influenza, about 90 percent manifesting short, high, febrile period with prostration but little or no severe pulmonary complications. The remaining 10 percent developed the old-fashioned post-influenzal pneumonia, showing mixed infection with pneumococcus, influenza bacillus, and the streptococcus hemolyticus.

The Naval Station, Tutuila, Samoa, reported one case of typhoid fever.

Deaths from cerebrospinal fever and meningitis, cerebrospinal, acute, during January, February and March 1935 occurred as follows:

	Ship or station	Date of death	Length of service
H. H. F.[1]	Recruiting Station, Kansas City, Mo.	Jan. 26, 1935	29 years.
D. L. K.	Naval Training Station, Norfolk, Va.	Jan. 28, 1935	1 month.
R. K.	U. S. S. *Minneapolis*	Feb. 12, 1935	1 year, 3 months.
H. B. G.	U. S. S. *Tennessee*	Feb. 13, 1935	9 months.
W. M. N.[2]	Naval Training Station, Norfolk, Va.	Mar. 6, 1935	3 months.
G. W. Y.[2]	Naval Training Station, San Diego, Calif.	Mar. 8, 1935	do.
J. C.	U. S. S. *Idaho*	Mar. 19, 1935	1 month.

[1] Primary cause of death, mastoiditis, acute; secondary, meningitis, cerebrospinal, acute.
[2] Meningitis, cerebrospinal, acute.
[3] Primary cause of death, otitis, media, acute; secondary, meningitis, cerebrospinal, acute.

In addition to the fatal cases listed above there were 3 nonfatal cases, as follows: 1 cerebrospinal fever on board the U. S. S. *Chester* and 1 on board the U. S. S. *Houston* in January, and 1 meningitis, cerebrospinal, acute on board the U. S. S. *Maryland* in March.

Reports from forces afloat indicate that morbidity rates for disease and injuries were a little more than expectancy. The admission rate for the quarter was 447 per 1,000 per annum as compared with 390, the corresponding rate for the preceding 5 years.

A total of 2,198 cases of acute respiratory diseases was reported by all ships of the Navy during January, February, and March 1935, indicating a 20-percent decrease from the number of cases notified for the preceding quarter.

Twelve ships of the Navy reported 40 or more cases of acute respiratory infections distributed over the quarter as follows:

Ship	January	February	March
U. S. S. Arizona	6	14	22
U. S. S. California	42	8	10
U. S. S. Idaho	24	12	24
U. S. S. Lexington	22	18	26
U. S. S. New Mexico	26	15	17
U. S. S. Northampton	16	29	10
U. S. S. Pennsylvania	7	21	19
U. S. S. Ranger	28	8	14
U. S. S. Tennessee	21	62	13
U. S. S. West Virginia	29	12	45
U. S. S. Wright	45	15	4
U. S. S. Wyoming	10	61	21

An unusually large number of cases of catarrhal fever appeared on board the U. S. S. *Tennessee* in February. As reported by the senior medical officer:

> These cases began to appear on the morning of the 5th and during the next 48 hours the number increased at an alarming rate to reach epidemic proportions. On the 7th of February the number of cases began to subside and gradually reached a normal level. These cases were diagnosed as catarrhal fever. However, it is believed that it is practically impossible to differentiate catarrhal fever from a mild type of injuries. The diagnosis of catarrhal fever was based upon (*a*) catarrhal manifestations; (*b*) headache, backache, and malaise; (*c*) mild febrile reaction (although in 5 cases the temperature reached 103° F.); (*d*) rapid response to rest and symptomatic treatment; and (*e*) absence of complications.

Cases of respiratory infection, usually diagnosed as influenza, were prevalent among the civil population of Long Beach and San Pedro at this time.

A moderately severe case of typhoid fever was readmitted to the U. S. S. *Relief* from the U. S. S. *Texas* and diagnosis established on January 23, 1935. A complete course of straight typhoid vaccine had been administered in December 1933. One case of paratyphoid fever was admitted to the sick list on board the U. S. S. *Guam* in February. No questionnaire has been received in the Bureau for this case, consequently no information is available regarding prophylaxis.

A typical case of dengue was admitted on board the U. S. S. *Augusta* on March 17, 1935, and died on March 26, 1935. Autopsy findings showed granular degeneration of the liver and kidneys, hypertrophy and hyperplasia of the splenic tissue, loss of cortical lipochrome substance of adrenal glands.

Fourteen cases of chickenpox were notified from forces afloat as follows:

Ship	January	February	March
U. S. S. Sirius	0	1	0
U. S. S. New York	0	3	1
U. S. S. Argonne	0	2	0
U. S. S. California	1	0	0
U. S. S. Canopus	1	0	0
U. S. S. Omaha	1	0	0
U. S. S. Ranger	1	0	0
U. S. S. Altair	0	1	0
U. S. S. Litchfield	0	1	0
U. S. S. Melville	0	0	1

Scarlet fever was reported in January and March by the U. S. S. *Saratoga*.

A case of typhus fever was admitted to the sick list in January on board the U. S. S. *Oahu*, Asiatic station.

TABLE 1.—*Summary of morbidity in the United States Navy for the quarter ended Mar. 31, 1935*

Average strength	Forces afloat, 72,730		Forces ashore, 38,723		Entire Navy, 111,453	
	Admissions	Rate per 1,000	Admissions	Rate per 1,000	Admissions	Rate per 1,000
All causes	8,124	446.80	5,538	572.06	13,662	490.32
Diseases only	7,025	386.36	4,892	505.33	11,917	427.70
Injuries	1,092	60.06	635	65.59	1,727	61.98
Poisonings	7	.38	11	1.14	18	.65
Communicable diseases transmissible by oral and nasal discharges (class VIII):						
(A)	190	10.45	398	41.11	588	21.10
(B)	2,253	123.91	1,850	191.10	4,103	147.25
Venereal diseases	2,017	110.93	488	50.41	2,505	89.90

TABLE 2.—*Deaths reported, entire Navy, during the quarter ended Mar. 31, 1935*

Cause—Disease		Navy			Marine Corps		Nurse Corps	Total
Primary	Secondary or contributory	Officers	Midshipmen	Men	Officers	Men		
Average strength		9,507	1,673	82,652	1,162	16,131	328	111,453
Abscess, brain	Hemorrhage, cerebrum					1		1
Abscess, lung	Abscess, brain			1				1
Appendicitis, acute	Peritonitis, general, acute			2				2
Arteriosclerosis, cerebral	Hemorrhage, cerebral					1		1
Arteriosclerosis, general	Thrombosis, coronary artery.	1						1
Carcinoma:								
Rectum	None			1				1
Stomach	do					1		1
General	Pneumonia, broncho-					1		1
Cerebrospinal fever	None			3		1		4
Colitis, acute	Hemorrhage, colon				1			1
Dengue	None			1				1
Glioma, brain	do			1				1
Influenza	Pneumonia, broncho-					4		4
Mastoiditis, acute	Meningitis, cerebrospinal, acute.	1						1
Do	Septicemia			1				1
Myocarditis, chronic	Arteriosclerosis, general	1			1			2
Meningitis, cerebrospinal, acute.	None			1				1
Nephritis, chronic	do			1				1
Do	Arterial hypertension			1				1
Otitis, media, acute	Meningitis, cerebrospinal, acute.			1				1
Pneumonia, broncho-	None	1		2				3
Pneumonia, lobar	do			3		1		4
Do	Abscess lung			1				1
Do	Myocarditis, chronic			1				1
Tonsillitis, acute	Septicemia			1				1
Tuberculosis, peritoneum, chronic.	Myocarditis, chronic			1				1
Tuberculosis, pulmonary, chronic.	Hemorrhage, pulmonary			1				1
Tuberculosis, pulmonary, chronic.	Tuberculosis, meninges			1				1
Ulcer, duodenum	Peritonitis, general, acute			1	1			2
Total for disease		4		26	3	10		43
Injuries and poisonings								
Burn, multiple	None			1				1
Crush, pelvis	do					1		1
Drowning	do	1		9				10
Do	Psychosis, unclassified			1				1
Fracture, compound, skull.	None			3		2		5
Fracture, simple, skull	do			1				1
Fracture, simple, vertebra, cervical.	do			1				1
Injuries, multiple, extreme.	do	4		6		2		12
Intracranial injury	do			1				1
Wound:								
Gunshot, head	None			4	1			5
Do	Psychosis, unclassified				1			1
Lacerated, neck	Hemorrhage, traumatic, jugular vein and carotid artery.			1				1
Poisoning, acute:								
Cyanide	None			2				2
Neoarsphenamine	Agranulocytosis pneumonia, broncho.			1				1
Strychnine	None			1				1
Total for injuries and poisonings		5		32	2	5		44
Grand total		9		58	5	15		87
Annual death rate per 1,000:								
All causes		3.79		2.81	17.21	3.72		3.12
Disease only		1.68		1.26	10.32	2.48		1.54
Drowning		.42		.44				.36
Poisonings				.19				.14
Other injuries		1.68		.92	6.88	1.24		1.08

ADMISSIONS FOR INJURIES AND POISONINGS, FIRST QUARTER, 1935

The following table, indicating the frequency of occurrence of accidental injuries and poisonings in the Navy during the first quarter, 1935, is based upon all form F cards covering admission in those months which have reached the Bureau:

	Admissions, January, February, and March 1935	Admission rate per 100,000, per annum	Admission rate per 100,000, year 1934
INJURIES			
Connected with work or drill	828	2,972	2,397
Occurring within command but not associated with work	439	1,576	2,064
Incurred on leave or liberty or while absent without leave	460	1,651	1,699
All injuries	1,727	6,198	6,160
POISONINGS			
Industrial poisoning	7	25	15
Occurring within command but not connected with work	8	29	244
Associated with leave, liberty, or absence without leave	3	11	15
Poisonings, all forms	18	65	273
Total injuries and poisonings	1,745	6,263	6,433

Percentage relationships

	Occurring within command				Occurring outside command—leave, liberty, or A. W. O. L.	
	Connected with the performance of work, drill, etc.		Not connected with work or prescribed duty			
	January, February, and March 1935	Year 1934	January, February, and March 1935	Year 1934	January, February, and March 1935	Year 1934
Percent of all injuries	47.9	38.9	25.4	33.5	26.6	27.6
Percent of all poisonings	38.9	5.3	44.4	89.3	16.7	5.3
Percent of total admissions, injury, and poisoning titles	47.9	37.5	25.6	35.9	26.5	26.6

Poisoning by a narcotic drug or by ethyl alcohol is recorded under the title "Drug addiction" or "Alcoholism", as the case may be. Such cases are not included in the above figures.

There were no cases during the first quarter of 1935 worthy of notice from the standpoint of accident prevention.

STATISTICS RELATIVE TO MENTAL AND PHYSICAL QUALIFICATION OF RECRUITS

The following statistics were taken from monthly sanitary reports submitted by naval training stations:

January, February, and March 1935	U. S. naval training station	
	Norfolk, Va.	San Diego, Calif.
Recruits received during the period	533	800
Recruits appearing before Board of Medical Survey	3	0
Recruits recommended for discharge from the service	3	0
Recruits discharged by reason of medical survey	4	0
Recruits held over pending further observation	0	0
Recruits transferred to the hospital for treatment, operation, or further observation for conditions existing prior to enlistment	11	27

The following table was prepared from reports of medical surveys in which disabilities or disease causing the surveys were noted as existing prior to enlistment. With certain diseases, survey followed enlistment so rapidly that it would seem that many might have been eliminated in the recruiting office.

Cause of survey	Number of surveys	Cause of survey	Number of surveys
Absence, acquired, teeth	2	Epilepsy	5
Anomaly of form (congenital short leg, right)	1	Flat foot	3
Arterial hypertension	3	Gonococcus infection, urethra	2
Arthritis, chronic, right knee	1	Hammertoe	2
Cardiac arrhythmia, extra systole	1	Hernia, inguinal, right	1
Constitutional psychopathic state, emotional instability	3	Hydronephrosis, both kidneys	1
Constitutional psychopathic state, inadequate personality	4	Malocclusion, teeth	2
Constitutional psychopathic inferiority, without psychosis	2	Migraine	1
Deformity, acquired, chest	1	Myopia	1
Deformity, acquired, fourth and fifth dorsal vertebrae	1	Osteoma, left tibia	1
Deformity, acquired, left forearm	1	Otitis, media, chronic	1
Deformity, acquired, right elbow	1	Periostitis, chronic, left heel	1
Deformity, acquired, right femur	1	Pes cavus	1
Dementia praecox	1	Ptosis, congenital, bilateral	1
Dermatitis, vesiculo-papular, both hands	1	Sinusitis, maxillary	1
Eczema	1	Stammering	1
Enuresis	5	Syphilis	4
		Ulcer, duodenum	3
		Ulcer, stomach	1
		Valvular heart disease, aortic insufficiency	1
		Valvular heart disease, mitral stenosis	3
		Varicocele	1

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